A Clinician's BPSD Guide 2023

Understanding and helping people experiencing changed behaviours and psychological symptoms associated with dementia

UNSW SYDNEY

CHeBA
Healthy Brains Positive Ageing

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ISBN 978-0-6459932-0-2

Disclaimer

This Guide was developed by the Centre for Healthy Brain Ageing (CHeBA) for the purpose of providing guidance for clinicians, residential care staff and community care staff supporting people living with dementia, who present with behaviours and psychological symptoms associated with dementia (BPSD). The information contained in this Guide was developed to be used as a resource by service providers. This Guide is provided for general information only and does not claim to reflect all considerations. As with all guidelines, recommendations may not be appropriate for use in all circumstances.

The Guide incorporates information from the most recent literature and other sources. Every effort has been made to ensure the accuracy and reliability of the information in this Guide at the time of publication. Links to Internet Sites and resources, which are identified, represent only a selection of those available. Links to other Internet Sites that are not under the control of the University of New South Wales are provided for information purposes only. Care has been taken in providing these links as suitable reference resources. However, due to the changing nature of Internet content, it is the responsibility of users to make their own investigations, decisions and enquiries about any information retrieved from other Internet Sites. The provision and inclusion of these links do not imply any endorsement, non-endorsement, support or commercial gain by the University of New South Wales.

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Suggested citation: Burns, K.*, Casey, A-N.* & Brodaty, H. (2023). *A Clinician's BPSD Guide 2023: Understanding and helping people experiencing changed behaviours and psychological symptoms associated with dementia* (2nd ed.). Centre for Healthy Brain Ageing (CHeBA), Sydney, University of NSW.
*K Burns and A-N Casey contributed equally to this work and are designated as co-first authors.

Aboriginal and Torres Strait Islander readers are advised that this book may contain the names of deceased persons.

Design and printing by *Vertifixprint*

November 2023

Contents

Acknowledgements

UNSW Sydney was funded by the Australian Government Department of Health and Aged Care to develop the Clinician's BPSD Guide as part of a suite of updated resources. These resources include two apps and two travel size summary Guides developed to support carers and clinicians caring for people living with dementia who present with changed behaviours and/or psychological symptoms. The authors would like to thank the following people and organisations for their advice, contributions and support for the project.

The Australian Government represented by the Department of Health and Aged Care

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The authors also acknowledge the knowledge, guidance and advice provided during consultation, in relation to Aboriginal and Torres Strait Islander peoples and those from culturally and linguistically diverse backgrounds, for the BPSD Guide (2012). The original sections outlining additional considerations for these groups have been retained and supplemented with updated resources and literature. Below are the original contributors and their affiliations at the time.

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Abbreviations used in this Guide

Term	Abbreviation	Comments
Alzheimer's disease	AD	
Activities of Daily Living	ADLs	
Aboriginal Medical Service	AMS	
Behaviours and psychological symptoms associated with dementia	BPSD	Also known as changed behaviours, behaviours of concern, responsive behaviours, neuropsychiatric symptoms (NPS) and others
Culturally and Linguistically Diverse	CALD	
Dementia Behaviour Management Advisory Service	DBMAS	Dementia Support Australia (DSA)
Dementia with Lewy bodies	DLB	Also referred to as Lewy body dementia and Lewy body disease (LBD)
Dementia Outcomes Measurement Suite	DOMS	Refer to Bentvelzen, et al. 2017[1]
Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (2022)	DSM-5-TR	https://www.psychiatry.org/psychiatrists/practice/dsm
Frontotemporal dementia	FTD	
General Practitioner	GP	
International Classification of Diseases 11th Revision (2022)	ICD-11	https://icd.who.int/en
International Psychogeriatric Association	IPA	
Mild Cognitive Impairment	MCI	
National Institute for Clinical Excellence	NICE	
Parkinson's disease dementia	PDD	
Pharmaceutical Benefits Scheme	PBS	Australian Government
Residential Aged Care Services	RACS	
Randomised Control Trial	RCT	
Severe Behaviour Response Team	SBRT	Dementia Support Australia (DSA)
Vascular dementia	VaD	
World Health Organisation	WHO	

Introduction

Aim

A Clinician's BPSD Guide: Understanding and helping people experiencing changed behaviours and psychological symptoms associated with dementia (Clinician's BPSD Guide, 2023) was developed by the Centre for Healthy Brain Ageing (CHeBA) at UNSW Sydney to replace and update the document Behaviour Management - A Guide to Good Practice: Managing Behavioural and Psychological Symptoms of Dementia (BPSD Guide, 2012). The Clinician's BPSD Guide aims to provide guidance for clinicians, residential care staff and community care staff supporting people living with dementia, who present with *behaviours and psychological symptoms associated with dementia (BPSD).

Untreated BPSD contribute to premature admission to residential care, decreased quality of life for the person living with dementia and carers, significant carer stress, increased care costs, stress to care staff and excess disability[2]. This document provides a comprehensive evidence and practice-based overview of care principles to support people who present with BPSD through practical strategies and interventions. The empirical evidence contained in this Guide, in combination with clinical expertise may assist in achieving improved outcomes for people with dementia.

Terminology

*The term and abbreviation behaviours and psychological symptoms associated with dementia (BPSD) are used respectfully throughout this Guide for communication between professionals supporting people living with dementia. Terms such as changed behaviours, responsive behaviours, behaviours of concern, neuropsychiatric symptoms (NPS), behavioural and psychological changes in dementia and others are also used to describe BPSD and may be terms preferred by people living with dementia. The term changed behaviours may be more appropriate when clinicians are communicating with a person living with dementia, carers/family and/or community members. For more information on terminology see article published in the International Journal of Geriatric Psychiatry, Language paradigms when behaviour changes with dementia: *#BanBPSD*[3]. For information about consumer preferences on terminology, please consult the Dementia Australia *Dementia Language Guidelines*[4].

The term carers can refer to care staff or family and friends who provide support and care, also known as care partners, informal carers or caregivers. Where necessary, the text differentiates between care partners and formal, paid or professional carers.

The terms Aboriginal and/or Torres Strait Islander peoples and culturally and linguistically diverse (CALD) are used throughout this document. The terms Aboriginal and Torres Strait Islander and Indigenous are respectfully used to refer to Australia's first people. This includes a wide range of nations, cultures and languages across mainland Australia and throughout the Torres Strait. The term CALD is used in this Guide to refer to communities with diverse languages, ethnic backgrounds, nationalities, traditions, societal structures, and religions. The Australian Bureau of Statistics[5] defines people from CALD backgrounds as those born overseas, in countries other than those classified 'main English-speaking countries'[6]. It is important to note that people born in English-speaking countries may identify with a different culture. We respectfully acknowledge that these are collective terms which may overlook the complex differences, diversity and nuances within communities. Additional considerations relevant to Aboriginal and Torres Strait Islander peoples and those from CALD backgrounds are included in this Guide.

How to use this Guide

This document has been designed as a set of modules to facilitate ease of use and provide specific information relevant to the most commonly presenting changed behaviours and psychological symptoms associated with dementia. *Module 1*: *Supporting a person who presents with BPSD* provides a guiding framework which outlines risk assessment, comprehensive assessment, developing a behaviour support plan as well as the implementation and evaluation of intervention strategies. Each of the following General BPSD and behaviour specific modules begin with key messages and a two-page summary to enable quick reference to the information in the body of the module. The main body of each module provides specific information relevant to the following aspects for each BPSD:

- A description of the behaviour or psychological symptom and how it presents in dementia
- Causes
- Differential diagnosis
- Measuring the behaviour or psychological symptom
- Prevalence
- Effects on the person with dementia and others living with them or involved in providing care
- Results from the literature search
- Intervention strategies and the quality of the supporting evidence available for specific interventions for each behaviour or psychological symptom
  - *Psychosocial and environmental interventions*
  - *Biological and pharmacological interventions*
- Limitations of the evidence presented
- Conclusions/Principles of care.

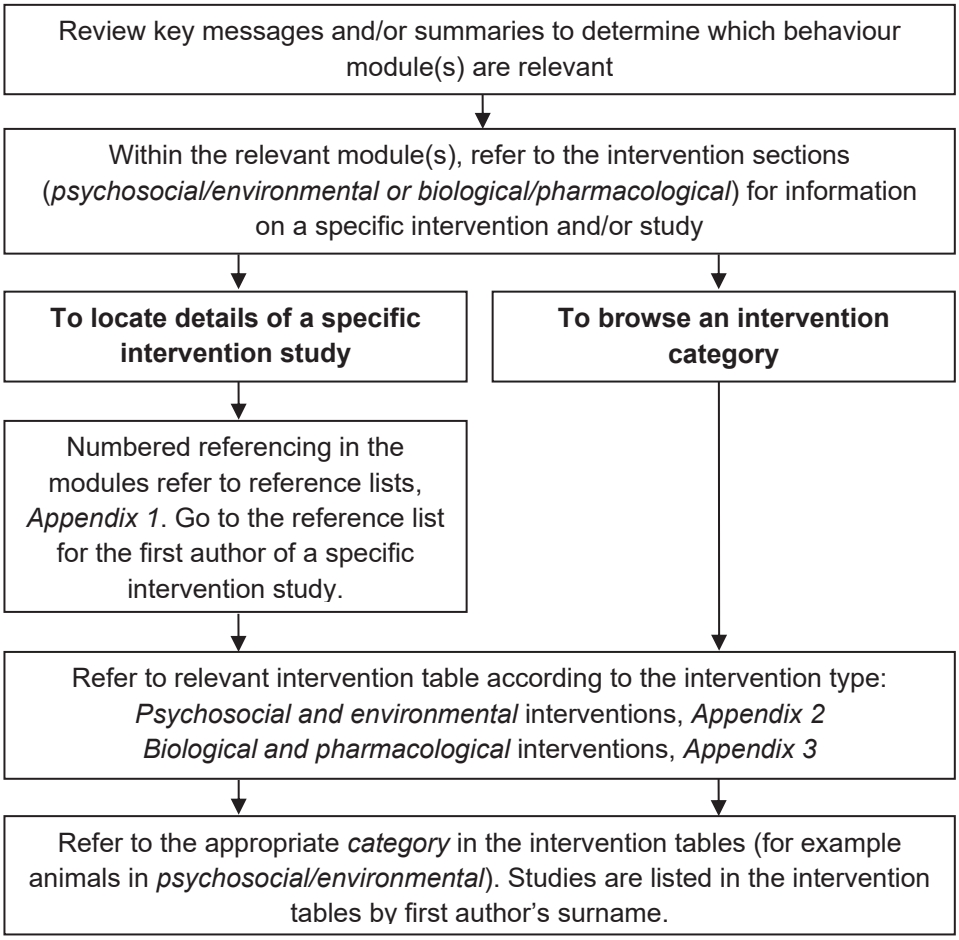
To better guide clinical practice, all intervention studies outlined in the modules have been assessed to determine the strength of the evidence for the findings reported and, where possible, effect size. See *Appendix 4: Methodology* for further details. Interventions are classified under *psychosocial and environmental* or *biological and pharmacological*. Within these groups, interventions have been further categorised for ease of access. See *Module 2: General BPSD* for definitions of categories. Details of intervention studies and the quality ratings are included in *Appendices 2 and 3*. To illustrate the information presented in the modules, each includes an example of a brief clinical scenario encompassing presentation, assessment, strategies and outcomes. All appendices are provided in electronic format only.

Person-centred care (PCC) principles

In providing assistance, service providers and clinicians need to be mindful of the rights of, and their obligations to, people with dementia[7,8]. People with dementia must give informed consent for interventions, or where this is not possible, proxy consent should be sought. The following broad principles should apply to all efforts in addressing changed behaviours and psychological symptoms:

- The rights of people with dementia and BPSD are recognised and protected.
- The goal for addressing BPSD is to maximise quality of life and safety within the least restrictive environment for each individual.
- BPSD are recognised as a form of communication.
- The significant impact of BPSD on the person with dementia, care partners, families, staff who provide care and others around the person is recognised.
- Collaboration with all people affected is the preferred approach to addressing BPSD.

To access information related to a specific intervention category or a specific study, follow the steps outlined below.



Cultural Competency

Cultural competency involves the recognition and respect of the aspects that make the person with dementia and/or their family and community diverse. It emphasises the need for health care systems and providers to be aware of, and responsive to, peoples' cultural perspectives and backgrounds[9, 10]. Understanding how these contribute to the interpretation of, and ability to access, appropriate and timely care as well as incorporating these considerations into clinical practice is important in reducing health inequalities. The person's country of origin, family and cultural background, preferred language, education, religion, belief system and socio-political outlook must be acknowledged with an appreciation of how these aspects influence care. Culturally competent clinicians and service providers must understand the difference between culture and language, which do not always go hand-in-hand.

While language is a significant barrier for many people from CALD backgrounds, it is also important to recognise cultural diversity and potential cultural barriers within English speaking populations. Refugees and others from marginalised backgrounds may present with additional unique and complex needs around forced displacement and trauma[11]. Service providers may not recognise where culturally relevant and sensitive services are required. Awareness of the nuances across and within different groups is also important. For example, those with a common language may be from different cultures based on geography and/or religion[12].

A culturally competent approach requires attention to three important variables[13].

- *Ethnocentrism:* Our individual viewpoint tends to be based on our own socio-cultural background. Although this typically occurs at an unconscious level, and is therefore difficult to distinguish, it informs how individuals view so-called 'normal' or 'abnormal' behaviour. This includes all those involved in care, and clinicians need to develop a conscious awareness of the viewpoint they and others impose. Accommodating the person with dementia's experience and differing perspectives on symptoms, diagnoses and acceptable treatments can then follow. Also see Brooke et al, 2018 *The intersection of culture in the provision of dementia care: A systematic review*[14].
- *General understanding of illness within community:* This differs across cultures depending on whether there is an emphasis on individualistic (ego-centric) or collectivistic (socio-centric) focus on health and wellbeing. Core individual differences, such as family dynamics, political views, educational background, religion, socio-economic status and sexuality can inform how health, wellbeing and illness are individually and collectively viewed.
- *The person with dementia and their family's health literacy:* Supporting the person with dementia and their family to improve their health literacy may help to decrease their fear of dementia, illness, assessment and treatment, enabling additional input into their health management. Education about biomedical methods should respect and add to their beliefs, not supersede them. A combined biomedical and culturally or spiritually relevant approach may improve the therapeutic relationship and compliance.

The following are important for culturally competent and sensitive service provision:

- *Knowledge of local context:* People from similar cultural and/or religious backgrounds should not be regarded as a homogenous group and stereotypical assumptions about the person with dementia should not be made on that basis. Each community is unique, as is the connection between community members and the broader community. Clinicians should gain an understanding of, and working relationship with, local communities, as this may assist in understanding service access issues, the needs of local communities and the local Lores and culture.
- *Communication:* Effective assessment for addressing changed behaviours or psychological symptoms in people with dementia from Aboriginal and/or Torres Strait Islander or CALD backgrounds may require additional attention to communication strategies. Communication not only encompasses language but also recognition of culturally appropriate methods of communication i.e., preferred ways of addressing Elders, body language and other cultural cues. See *Module 1* for more information on culturally competent communication.
- *Assessment tools:* Clinicians undertaking assessment for addressing BPSD may need to exercise judgement in determining the appropriateness of specific assessment tools. This may include discussion with the person, carers, family and/or service providers regarding the relevance of assessment and assessment tools. Culturally appropriate instruments, where available and appropriate, should be used. See *Module 2* for more information on culturally appropriate assessment tools including the KICA and the RUDAS.
- *Raising Awareness:* Where communities may have difficulty accessing services, raising awareness around dementia, BPSD and services available is important. People may lack understanding that dementia and BPSD are not part of normal ageing and that 'culturally safe' options to address BPSD are available.
- *Auditing and benchmarking:* Auditing of an organisation's written materials, resources, referral processes, telephone manner, performance appraisals, benchmarking and service provision for cultural competency should identify areas where services need improvement.

The Purnell Model of Cultural Competence[15, 16] and framework provides a tool for use in health settings to promote culturally competent assessment of cultural perspectives of health and

health care. The model outlines the 12 cultural domains and associated concepts that impact on health care provision for people from a CALD background (building on the points outlined above), including, but not limited to heritage, family roles and organisation, nutrition, spirituality and health care practices. The model can guide clinical practice including assessment from a culturally competent perspective and the development and implementation of individualised, person-centred interventions.

Additional cultural considerations for service provision to those with dementia in Aboriginal and Torres Strait Islander communities

Aboriginal and Torres Strait Islander peoples are made up of many diverse communities and language groups within Australia. Variations in cultural norms are relative to multiple factors which include size of the community, geography, climate, urbanisation, language as well as traditional, historical, political and social influences. While Aboriginal and Torres Strait Islander cultures incorporate extensive diversity, for the purposes of this Guide, aspects related to Aboriginal and Torres Strait Islander peoples will be considered together. Further investigation is required if these aspects are to be more descriptive and specific even within the context of the variations in cultural norms and multiple factors. These investigations are outside the scope of this Guide.

While significant heterogeneity presents across Aboriginal and Torres Strait Islander peoples living in urban, regional and remote locations, commonality arises from a shared history of dispossession, disadvantage and poor health outcomes. An understanding of the unique needs of communities at a local level is essential to providing holistic, culturally appropriate services. An awareness of cultural differences is only the first step toward cultural competency. Aboriginal and/or Torres Strait Islander cultural competency requires an awareness of culture within the context of community/family and the life experiences which guide the additional considerations for working with families living with and/or caring for a person with dementia. Clinicians' usual manner of assessment may not be culturally appropriate for Aboriginal and/or Torres Strait Islander peoples and cultural competency is essential for the comprehensive and appropriate assessment of BPSD.

The following resources provide further information specific to service access issues, the needs of Aboriginal and Torres Strait Islander communities and cultural competency.

Australian Institute of Health and Welfare (AIHW), Dementia in Australia: *Understanding dementia among First Nations people, 2023*[17] (accessed October 2023).

Australian Institute of Health and Welfare: Australian Institute of Family Studies, *Cultural competency in the delivery of health services for Indigenous people*, 2015[18] (accessed October 2023).

The Living Longer Stronger resources have been created for health professionals who treat Aboriginal clients with chronic health conditions. Aboriginal Health & Medical Research Council (AH&MRC), *Living Longer Stronger Resource Kit*, 2020[19] (accessed October 2023).

Australian Human Rights Commission, *Face the facts: Aboriginal and Torres Strait Islander peoples,* 2014[20] (accessed October 2023).

The Aboriginal Practice Checklist provides a basic framework for management and staff to evaluate their agency's policies and practices in relation to Aboriginal clients and partner agencies. Aboriginal Health and Medical Research Council of NSW. *Aboriginal practice checklist: a cultural assessment tool for MERIT teams*, 2010[21] (accessed October 2023).

The NSW Aboriginal Mental Health and Wellbeing Strategy 2020-2025 supports and assists NSW Health services in delivering respectful and appropriate mental health services in partnership with Aboriginal services, people and communities. NSW Department of Health. *NSW Aboriginal Mental Health and Well Being Policy 2020-2025,* 2020[22] (accessed October 2023).

Additional cultural considerations for service provision to those with dementia in CALD communities

When working with CALD communities it is important to recognise the myriad of different understandings of wellbeing, health, illness, mental health and dementia, knowledge of service availability and pathways to access. These factors will vary widely and affect the way people interpret their situation and react to care and treatment. Clinicians' approach to assessment and addressing BPSD may not be culturally appropriate or relevant for CALD communities. Therefore, culturally competent care and clinical practice is vital for comprehensive and appropriate assessment and for addressing BPSD within CALD communities.

The following resources provide further information specific to service access issues, the needs of people from CALD backgrounds and cultural competency.

Federation of Ethnic Communities' Councils of Australia. *Cultural Competence in Australia: A Guide*, 2019[23] (accessed October 2023).

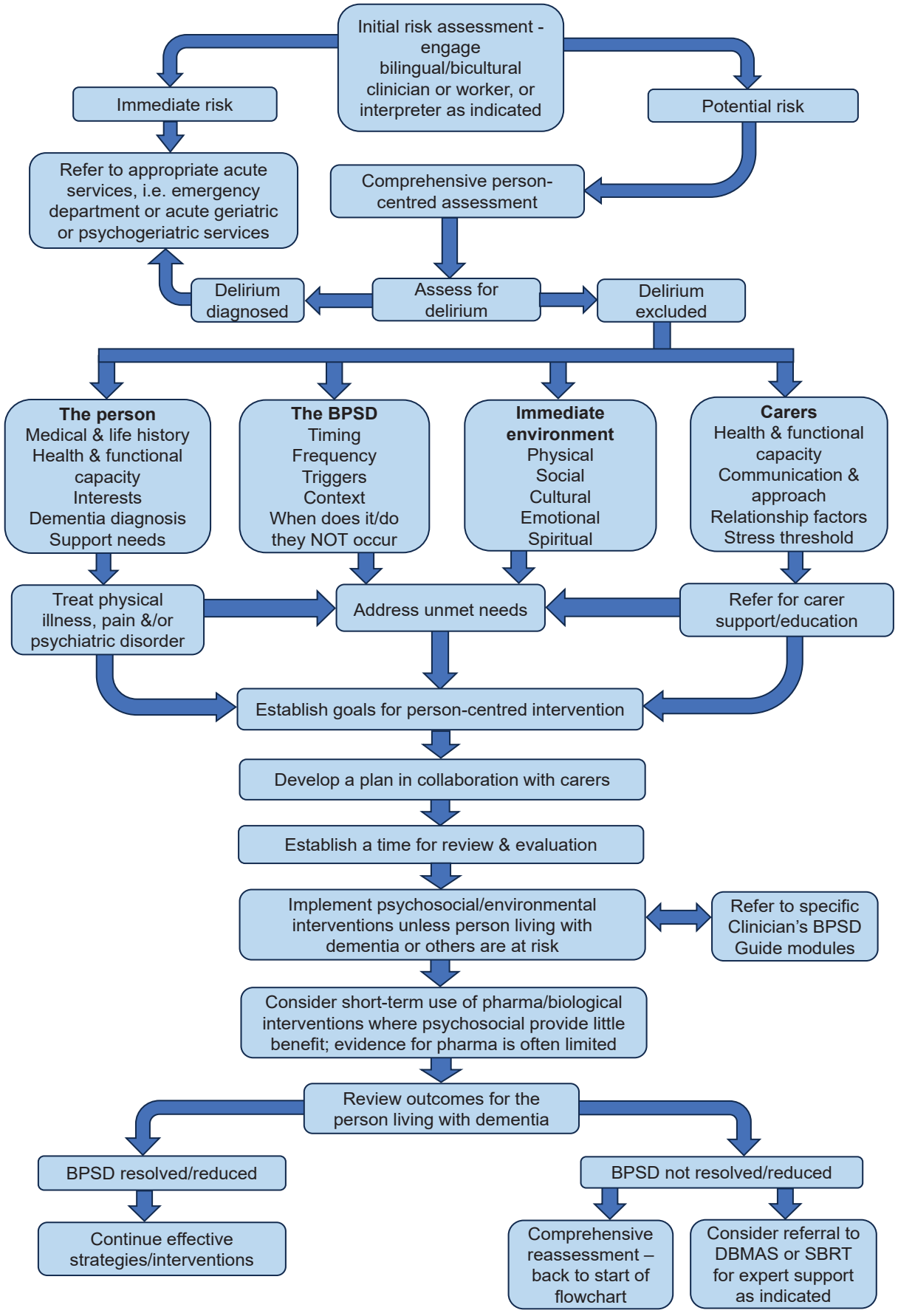
Dementia UK. *Cultural and religious awareness within dementia care*, 2021[24] (accessed October 2023).

Australian Institute of Health and Welfare. *Reporting on the health of culturally and linguistically diverse populations in Australia: An exploratory paper*, 2022[12] (accessed October 2023).

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

1. Addressing BPSD

Figure 1.1 Process for addressing changed behaviours or psychological symptoms



Addressing BPSD

Clinicians should seek to understand the context, meaning and perspective of dementia and the changed behaviours or psychological symptoms exhibited for the individual person. It is important to be aware of the different ways dementia and BPSD may be understood when collecting information around the situation (see Introduction module for further information). A person from an Aboriginal and/or Torres Strait Island or culturally and linguistically diverse (CALD) background may have a different understanding and experience of dementia and BPSD. See additional considerations and relevant historical aspects highlighted throughout this module. These basic principles are important:

- A person-centred approach that reflects this diverse range of causative factors is likely to be the most effective way of addressing BPSD.
- Interventions which focus on addressing the underlying contributing factors rather than the BPSD are likely to be more effective.
- A thorough and detailed assessment of the person with dementia and the BPSD in combination with the interpersonal and physical environment is essential to generate potential strategies and interventions.
- The skills and knowledge of a variety of professional disciplines may be beneficial in working toward a positive outcome.

Initial risk assessment

The most immediate consideration is the degree of risk, or the potential harm or danger, inherent in the situation for the person with dementia, their care partner, family members, others living with the person, care staff and/or members of the public[1, 2]. The purpose of a risk assessment is to determine whether, without an immediate or prompt response, likely harm will come to the person with dementia or others. The degree of risk should be assessed without delay:

- *Immediate risk*: There is immediate medical, mental health, physical and/or environmental risk to the person with dementia or others.
- *Potential risk*: The risk to the person with dementia or others is potential at this time. Strategies to prevent future increased risk should be integrated into the care plan.

When determining the degree of risk the following aspects should be taken into account:

- the nature and severity of the presenting changed behaviours or psychological symptoms
- the context
- the resources available within the care environment to address the situation.

Areas of risk

When conducting a risk assessment, the potential medical, mental health, physical and/or environmental areas of risk should be considered. See *Table 1.1* for possible areas of risk to, the person with dementia and other people.

Medical and mental health

Delirium is potentially a significant risk. *Module 2* provides further information on differential diagnosis of delirium and guidelines for the recognition and investigation of delirium as well as additional resources. The person with dementia may be at risk from untreated medical causes of the BPSD or co-morbid mental illness that may progress rapidly without urgent treatment, including depression or psychosis. If the person with dementia is severely depressed, suicidal or not eating and drinking, urgent referral should be made to a psychogeriatrician or psychiatrist. If the person is psychotic and acting on hallucinations or delusions in a way that may be dangerous, immediate referral to a psychiatric emergency/crisis team or hospital emergency department is indicated.

Physical

Changed behaviours such as aggression, wandering and sexual disinhibition can pose a risk of physical harm to the person with dementia and/or others. Similarly, physical, financial, emotional and/or sexual abuse raise concerns. Dangerous situations involving physical risk must be approached with caution. For situations with high risk of physical harm, avoid escalation and protect all involved by:

- not arguing or attempting to reason with the person with dementia
- maintaining contact with local assistance via phone if possible
- if necessary, remove others in the immediate area from the person with dementia and seek assistance from others nearby and/or contacting local emergency services such as police or ambulance.

Environmental

High risk to the person with dementia can be associated with an unsafe environment. This can include extreme carer stress or the risk of loss of accommodation, particularly in residential care settings. BPSD can place the person with dementia in imminent danger of being moved from their current accommodation. The *Aged Care Act 1997* (Cth)[3] is the main law that sets out the rules for government-funded aged care and the *Residential Care Manual* (2014)[4] provides legislative guidance on the rights of people living in residential care. At the time of writing, the Department of Health and Aged Care is developing a new Aged Care Act. Subject to being passed in Parliament, the new Act is planned to commence on 1 July 2024. For the latest information on Australian Government law see https://www.legislation.gov.au.

Communication

Effective and respectful communication is an essential aspect in assessment of the person with dementia to develop a behaviour support plan to address BPSD. This is particularly important when aiming to provide culturally competent assessment to people with dementia and their families from Aboriginal and Torres Strait Islander or CALD backgrounds[5-8]. Spoken language can be a significant barrier when seeking health advice and interacting with services and body language, or non-verbal communication signals, may vary in meaning and importance[9].

***General principles for culturally competent communication*[10]**

- Confirm the person's preferred mode of address (e.g., Mr, Ms, Mrs, Aunty, Uncle) and the correct pronunciation of their names.
- Based on observation and some direct questioning, ensure your communication and interaction style is appropriate to the person's cultural background. This includes the way you approach the person, your tone of voice and whether eye contact is made or not.
- If providing any written form of communication, ensure that is it is in the person's preferred language as well as clear and easy to understand. Be mindful that some people may be illiterate in their first spoken language so written resources may be inappropriate.
- Recognise and accommodate the influence of the social and cultural context on communication.

For further information on culturally competent communication refer to the following:

Australian Government Style Guide, Inclusive language - *Aboriginal and Torres Strait Islander peoples*, 2023[11] (accessed October 2023).

Australian Government Style Guide, Inclusive language - *Cultural and linguistic diversity*, 2023[12] (accessed October 2023).

When collecting information regarding dementia and BPSD from Aboriginal and Torres Strait Islander peoples or people from CALD backgrounds, the person, carers and/or family members may not be fluent or comfortable speaking in English[8, 13]. Enlisting the services of an Aboriginal or Torres Strait Islander health worker or a bilingual/bicultural clinician or worker is recommended. See below for additional information. Where these options are unavailable, an interpreter may be required to ensure optimal information exchange and comprehensive assessment[14, 15].

Table 1.1 Potential areas of risk for the person with dementia and others

MEDICAL/MENTAL HEALTH	PHYSICAL	ENVIRONMENTAL
Medical • delirium • urinary tract infection • silent infection • constipation • inability to recognise or report pain and/or other symptoms • reduced appetite, food and/or fluid intake resulting in dehydration, malnourishment and/or electrolyte imbalance • atypical disease presentation, i.e. lack of common features • polypharmacy • medication noncompliance, overdose or toxicity • co-morbidity • presence of other chronic diseases • alcohol abuse **Mental health/ co-morbidities** • depression • suicidal ideation • psychosis • psychiatric history • historical trauma • post-traumatic stress disorder (PTSD) • anxiety	**Physical harm to the person living with dementia** • lack of awareness of exposure to danger • injury as a result of physical aggression directed at others or inanimate objects • wandering from care setting unaccompanied • changes in perception • misinterpretation of environment, including other people • increased risk of falls • extreme carer stress leading to assault • neglect or abuse i.e. physical, financial, sexual • self-destructive behaviours • impulsive behaviours **Physical harm to others** • physical aggression • sexual disinhibition • response to delusions or hallucinations • situation exacerbated by alcohol • physical abuse	**Unsafe environment** • safety hazards in care environment • carer's capacity and/or tolerance compromised due to illness or lack of support • avoidable transfer to hospital resulting in increased confusion and/or disorientation • changes to physical environment • leaving the home unsecured • allowing strangers into the home **Loss of accommodation** • transfer from current accommodation • transfer to more restrictive care setting

Communicating with Aboriginal and Torres Strait Islander communities

Culturally appropriate language may vary considerably within and across Aboriginal and Torres Strait Islander communities throughout Australia. Services must be competent in culturally appropriate terminology which is acceptable to the communities in their local area[16].

Effective communication requires respect, listening, patience, confirmation and clarification[8]. Historical factors mean pre-existing partnerships with organisations and/or communities are essential to develop trust[17, 18].

In Aboriginal and Torres Strait Islander communities an appropriate spokesperson may be nominated to act as an interpreter and/or support person for the person with dementia. In some situations, younger family members (under 18 years) may be nominated by an Elder or older Aboriginal or Torres Strait Islander person to interpret.

Assessment and diagnosis should ideally involve a relevant Aboriginal or Torres Strait Islander person. An Aboriginal and/or Torres Strait Islander cultural translator or health worker could assist in the assessment process beyond the concept of interpreters as used in a conventional role[19, 20]. See *Care Environment* section of this module for further information. Consent to access a cultural broker must, of course, be obtained from the client, carer and/or key person beforehand.

At times, it is appropriate to consider the separation of Men's business and Women's business. In some communities a female Aboriginal or Torres Strait Islander health worker will not be able to discuss health issues or assist with an assessment of a male client[8]. Likewise, younger clinicians attending BPSD assessment may be hindered by their youth when working with Elders.

Communication: CALD considerations for working with bilingual and bicultural clinicians and workers

Culturally appropriate assessment requires working with language-concordant and proficient bilingual/bicultural clinicians as a first preference[21, 22] and/or bilingual/bicultural workers as a second preference. Where the person with dementia's preferred language is not English it is important for the clinician or worker to have competency in both languages (bilingual).

Bilingual or bicultural workers[23], or community health navigators[24] may be those working with members of the CALD community who may not have formal qualifications but are able to provide information, education, care and/or support to mainstream clinicians. Bilingual workers can act as a communication facilitator, however, in some instances they cannot act as interpreters due to confidentiality or other issues. In this situation an interpreter may be required.

A bicultural clinician or worker has a high level of competence in and/or is from a similar cultural background to the person with dementia so that they can facilitate communication and information exchange in a culturally competent manner[23]. This is not restricted to language, as the person with dementia may be proficient in English and come from a different CALD background, thus requiring a cultural broker as opposed to a bilingual clinician/worker.

Pathways to accessing bilingual/bicultural clinicians and workers will vary considerably by State and Territory depending on the support structures in place and networks that have previously been established between and across services. Clinicians should familiarise themselves, and establish connections, with services and resources for different language and cultural groups in their area.

Working with an interpreter during assessment

Clinicians should avoid where possible, relying on family and/or friends to act as interpreters during a specialist assessment. See *Interpreters: specific Aboriginal and Torres Strait Islander considerations* box below for additional information. A duty of care to the person with dementia requires that the assessment be as objective as possible[10]. However, in doing so family and friends can feel they have a diminished role in the assessment and care planning process. It should be emphasised that family and friends have an important role in other parts of the

process, for example interpreting in everyday communication. Ensure that the person and their significant others are informed, both verbally and in writing in their preferred language, of their right to access an interpreter at no cost to them at any time during the assessment. Consistent access to interpreters that the person with dementia and their family know and trust may be most helpful[25, 26].

Prior to involving an interpreter, it is necessary to determine the English language proficiency of the person with dementia. While you should always enquire whether the person would like an interpreter, it can be difficult to determine if an interpreter is required. This can occur when the person and/or their family are not familiar with the role of an interpreter. Alternatively, if their English is limited, or there is a cultural perception that it is better not to cause problems or "be a bother", the person with dementia may agree to all suggestions or requests. They may indicate that they can speak English without completely understanding what they are agreeing to. It is important to include other ways of determining whether the person with dementia requires an interpreter prior to undertaking an assessment:

- Language use and fluency may be screened using language items from adapted tools such as the Marin Short Acculturation Scale for Hispanics (SAS-H)[27] and the Singaporean SAS[28].
- Ask the person to answer three open ended questions that require full sentence answers, rather than just yes or no, in English.

It may also be necessary to establish the English language proficiency of carers. Interpreting services may be necessary to ensure comprehension and opportunity for feedback. A sensitive approach is indicated as family members may not require or wish to engage an interpreter and may find it offensive if asked.

Telephone versus in-person interpreters

It is *strongly recommended* that where an interpreter is required, an in-person interpreter should be present during a clinical assessment[10, 29]. A telephone interpreter service may not be suitable for clinical assessment of a person with dementia because:

- it may be difficult for the person to grasp the concept of interpreting when they cannot see the interpreter in person;
- impaired hearing will further limit communication as the interpreter needs to check with the person with dementia that they are hearing all those involved. When the clinician pauses, the interpreter may need to repeatedly reassure the person with dementia that the clinician is still present but silent;
- if dementia severity is such that the person is dysphasic, accurate interpreting over the telephone may be impossible. When the client's speech is confused and/or disjointed, simultaneous interpreting is required which is not practical or appropriate over the telephone and/or;
- a telephone assessment precludes the vital assistance of non-verbal cues which aid the assessment of BPSD and assessment of the comfort of the person with dementia.

In a situation where it is not practical to conduct an assessment in person, possibly due to remoteness, lack of transport or inability to travel, an assessment via telephone or video may be the only viable alternative[15]. Clinicians should aim to avoid distracting sounds such as shuffling papers, scraping chairs and/or nearby phones not on silent mode.

Key considerations when working with interpreters and people with dementia

The interpreter: It is important to assess the appropriateness of the interpreter for the assessment of the person with dementia and BPSD[10].

- Check the preferred language/dialect and gender with the person with dementia.
- Interpreters may be fluent in the person's language but be from an incompatible cultural or ethnic group.
- Confidentiality can be an issue in small communities or cultural groups. Be sensitive to the situation and the individual needs of all involved in the assessment process.
- Availability of interpreters varies with geographical locations and degree of remoteness. Booking well in advance for major medical appointments may be necessary.

Briefing the interpreter prior to the assessment[10, 17, 30]:

- Discuss strategies including length of speaking segments, turn taking, dealing with interruptions and the inappropriateness of simultaneous interpretation as this may confuse or overwhelm the person with dementia.
- If undertaking a cognitive assessment, highlight the importance of gathering objective information and not using cues or prompts which may bias results.

Seating arrangements: For optimal communication between all parties, seating should be arranged in a triangle providing a free line of vision and equal distance between all. This also provides visual access for the interpreter to pick up on all linguistic/verbal and paralinguistic/ non-verbal features of the person with dementia's speech, which might otherwise be missed if the interpreter is seated to the side. Where this is not possible due to confined space or potential safety concerns around an unpredictable person with BPSD, the interpreter should be seated to the side of the person or slightly behind. If this seating arrangement impedes effective communication, concerns should be raised promptly to allow a further attempt at the seating arrangement.

Medical terminology: Translation can be difficult when working with interpreters who lack experience within the health system. Mental health terminology can be complex and different contexts, history and meanings exist within cultural groups which can impact on the interpreting process. Where possible, interpreters should be accredited in mental health terminology.

Allow adequate time: Be aware that a thorough assessment involving an interpreter may need approximately double the time of an assessment for a person who is fluent in English. Be cognisant of the potential for fatigue in the person with dementia and the interpreter during the assessment and take breaks when appropriate.

Use short, easily comprehensible sentences: Rephrase questions or statements when the clinician has not been understood, avoid overuse of jargon or technical terms, provide concise information, speak clearly but not overly slowly and repeat as required. Be aware of not raising your voice to enhance understanding, as this may be offensive.

Avoid interruptions where possible as they can be very challenging for the interpreter and distressing for the person with dementia. Clinicians need to be patient during periods of interpretation and refrain from interrupting wherever possible.

Verbal and non-verbal cues: As language cues may be lacking for the clinician, they could be reliant on non-verbal cues to gauge if the person with dementia is comfortable. Be alert to signals such as facial expressions and body language. Verbal cues such as volume, pitch and tone of voice differ from one language to another and can be difficult to read accurately. If indicated, ask the interpreter about language and cultural idioms as well as non-verbal cultural cues.

Cultural sensitivities and nuances: When using a third party, such as an interpreter, it is important to ensure that cultural factors and sensitivities are sought and clarified early in the assessment process and respected as the assessment progresses.

Interpreters: specific Aboriginal and Torres Strait Islander considerations

A family/spokesperson may indicate that an interpreter is not required although the person with dementia may benefit from having one present[31]. While the use of consistent interpreters, in the conventional sense, for assessment is recommended, practical limitations exist[25, 26, 29, 31].

- Many different Aboriginal and Torres Strait Islander languages exist and there is no generic language used across Australia[11]. In some communities Aboriginal and Torres Strait language(s) may be the main language spoken at home[31, 32]. Respectful language depends on what different communities find appropriate[11].
- Past government resettlement programs can mean that numerous different language groups occupy one geographical region.
- Some traditional languages are lost and specific interpreters may be unavailable. In some northern areas of Australia, a Kriol interpreter may provide an alternative.

Interpreters: specific CALD considerations

- Clinicians may need to be sensitive to the interpreter's country of birth e.g., a Serbian interpreter may be able to interpret in Croatian, but their speech will have a different accent. Their name may also indicate their nationality and this may lead to trauma or distress in the Croatian client.
- Confidentiality issues may be more common within more recent migrant groups, which tend to be smaller. The person with dementia, interpreter and/or family may be known to each other. In this situation, a telephone interpreter who is external to the person's community, possibly from interstate, may be preferred.
- It may be appropriate in some instances to seek guidance from relevant ethnic community groups on these issues and to identify suitable interpreters or cultural links.

Comprehensive Assessment

Collecting information from a variety of sources, including care partners, care staff and/or family members, will assist in developing a comprehensive picture of the person with dementia and the BPSD. Assessment requires information to develop a description and investigation of the potential underlying factors prompting changed behaviour(s) or psychological symptom(s). *Appendix 4* provides suggested questions to facilitate assessment.

A comprehensive assessment should incorporate a ***person-centred approach***. The following aspects may influence the BPSD as well as the development and implementation of an effective behaviour support plan[33, 34]:

- The characteristics of the **person** living with dementia such as their personal history, including migration, language, cultural background, type of dementia, medical co-morbidities and current medication regime are important.
- A description of the **changed behaviour(s) or psychological symptom(s)** is needed.
- The **care environment** includes the physical, social and cultural dimensions of the person's immediate and extended environment. The care setting is also relevant, and where the person is in residential or acute care, the policies, systems, workforce skills and the organisation's management may be relevant.
- Characteristics of the **carers** and the **care relationship**. Knowledge of dementia amongst care staff and families, degree of experience, their attitudes to caring for those with dementia and the ability to apply their knowledge will vary and may contribute significantly. Likewise, staff members' abilities, training, cultural competency, approach and care practices are important.

A person-centred approach to dementia care and assessment

Developed by Kitwood and the Bradford Dementia Group[35, 36], person-centred care (PCC) provides a holistic framework and guiding principles for understanding the person with dementia, rather than focusing solely on addressing the BPSD[37]. Restrictive practices and/or neglect of psychological, social and cultural needs can compromise a person's sense of personhood (recognition, respect and trust gained through social interactions and relationships) and wellbeing[38, 39]. There may, however, be circumstances where the use of restrictive practices is considered necessary as a last resort measure to protect the health, safety, and dignity of the individual or others. Recognising these circumstances, the use of restrictive practices is strictly regulated and monitored in Commonwealth-funded residential aged care services (RACS). Care that emphasises a biomedical perspective can undermine personhood[35], contribute to what Kitwood terms *malignant social psychology*[38], and lead to emotional stress and increased BSPD[40].

Central to PCC in dementia is a focus on supporting relationships and communication[36]. PCC encompasses four key elements **VIPS**:

1. **V**aluing people with dementia (V)
2. Treating people as **I**ndividuals (I)
3. Looking at the work from the perspective of the **P**erson with dementia (P)
4. A positive **S**ocial environment in which the person with dementia can experience relative wellbeing (S)[37]

The *Person-centred Care Assessment Tool (P-CAT)* is a questionnaire that can be used to assess the extent to which care is person-centred within RACS[41, 42]. By employing a person-centred approach to all aspects of dementia care, including assessment and addressing BPSD, a holistic understanding of the person and their life can be incorporated into strategies that are tailored and appropriate for the individual.

The person living with dementia

Discovering as much as possible about the person with dementia is vital. Many aspects of the person's health and life experiences may contribute to the BPSD:

Dementia diagnosis: If a formal diagnosis has been made this can provide important information on the type of dementia, length of time since diagnosis and/or symptom onset, as well as associated cognitive and functional losses. Where the prevalence of particular BPSD varies significantly with dementia type, this is reported in the following modules.

Aspects of the person's life story or personal history: Collecting a social history and life story can assist in developing a comprehensive picture of the person with dementia. This may include details about the person's interests, routines, social networks, social roles, cultural background, spirituality, sexuality and special or traumatic events in their lives. A life story book or talking photo album, particularly where English is not the person's first language, can assist family members and/or care staff to develop a picture of the person with dementia from their early life to the present.

Physical and mental health: Aspects of the person with dementia's physical and mental health may contribute to BPSD. Co-morbid mental health issues including depression or delirium should be considered. See *Risk Assessment* section of this module for further information. Ensure all physical needs of the person have been addressed, including pain or discomfort. Acute and/or chronic pain may be present but underreported by the person[43, 44]. Their perception of, and ability to, articulate the presence of pain may be altered by cognitive impairment and/or BPSD. Delirium and/or BPSD can present as markers for the manifestation of pain[45]. Tools for verbal and nonverbal assessment of pain may be required to determine the potential contribution to BPSD. See *Module 2* for further information on delirium and pain in relation to BPSD.

Holistic wellbeing: An Aboriginal and Torres Strait Islander perspective

Aboriginal and Torres Strait Islander communities traditionally view physical and mental health holistically[46, 47] in a way that encompasses social and emotional wellbeing[48-50]. The social and emotional wellbeing of community members is maintained when the interconnected elements of spiritual, physical, emotional, social and cultural life are balanced. Health encompasses the social, emotional and cultural wellbeing of the whole community in which each individual is able to achieve their potential thereby bringing about the total wellbeing of their community[51]. Factors, including ancestry, connection to land, culture, identity, physical health and family relationships can affect social and emotional health[47, 52, 53]. Addressing dementia and BPSD in this group requires an understanding of ill health from a cultural perspective, including approaches to wellness.

Holistic perspective of health in CALD communities

Depending on their cultural and spiritual perspectives, people with dementia from CALD backgrounds and their families may view health holistically. As such, the physical, social, spiritual and emotional dimensions of health are interconnected aspects that can impact on BPSD and quality of life[54].

Description of the changed behaviour(s) or psychological symptom(s)

Gathering a detailed description of the changed behaviour or psychological symptom and the context in which it occurs is necessary for assessing the situation and establishing an appropriate behaviour support plan. As family carers and/or care staff are in frequent contact with the person living with dementia they are one of the most important sources of information. Consider the following:

- Assist the carer or care staff to describe the BPSD as accurately as possible and the context in which it occurs. For example, describing a person as agitated does not describe how this is manifested e.g., motor restlessness, calling out, picking at clothes.
- Be aware that perceptions of BPSD may vary with carer(s) knowledge and experience.
- Family carers and care staff may not understand that dementia interferes with the person's ability to control their behaviour.
- Carers may need assurance that changed behaviours are not intentionally provocative or directed at them personally.
- Where carers have trialled potentially effective interventions, provide validation and encouragement for their approach; no strategy will work every time.
- Collaborative conversation around strategies previously tried without success may help to develop further options to trial.
- Carers may have suggestions as to what may be prompting the BPSD.
- Understandings of individual BPSD can vary; explanations of how terms used in everyday language, such as aggression, anxiety or agitation apply in the context of dementia may be helpful.
- Encourage specific descriptions of the presenting BPSD e.g. the person "withdraws from other people", "swears at others", "becomes tearful easily" or "hits out at carers".

Table 1.2 Factors which may contribute to changed behaviours or psychological symptoms

PERSON WITH DEMENTIA	COMMUNICATION	ACTIVITIES OF DAILY LIVING	THE CARE ENVIRONMENT
Life history: • family and friends • pets • significant events • anniversaries • sexuality • migrant or refugee experiences • culturally relevant traditions and events • past trauma: - war-time experiences - Stolen Generation - dislocation from *Country* and culture - trans-generational and intergenerational trauma - loss of family member, home or Land/*Country* **Physical/sensory health:** • fever • oral pain • unrecognised and/or untreated pain • constipation • urinary tract infection • chest infection • other illness • adverse effects of medication • poor or interrupted sleep • headache • fatigue • impaired vision and/ or hearing without compensation • irritating itch **Mental health:** • depression • anxiety • post-traumatic stress disorder • other mental health issues **Emotional and spiritual health:** • attachment to Land/ *Country* • loss of sprit or searching for spirit • spiritual/religious beliefs, e.g., curses, external wrongdoings, payback as reasons for disease and/ or BPSD	• speaking too quickly • speaking in a condescending manner • not making eye contact where appropriate • not using person's preferred name/title • arguing with the person • correcting mistakes indicating failure • trying to reason with the person with dementia • language spoken is not the person's preferred language • not following appropriate cultural communication protocols • not using correct form of address and/or mannerisms • Little/no family involvement in communication • no other speakers of person with dementia's first language: - loneliness - isolation - frustration	**Meals:** • table setting too cluttered • more than one course served at a time • inflexible mealtimes • table companions cause irritation/agitation • changes in personnel • oral pain not recognised • person's eating space not defined • insufficient contrast between tablecloth and crockery • culturally inappropriate or food not liked **Personal care delivery:** • care plan not maintained or followed • care delivery rushed • lack of gentle approach • too many staff involved • gender roles • person not supported to participate • uncomfortable bathroom i.e., cold, claustrophobic and/or noisy • no choices offered or too many, too complex or inappropriate choices • poor selection of personal hygiene type e.g., bed bath may be more appropriate than shower • previous personal hygiene practices may be relative to past environment and resources available • previous dental hygiene practices ignored • over/under expectations of abilities by carers • care staff talking over the person • painful limb movements • lack of bilingual or culturally competent care staff where required • cultural and spiritual needs not integrated into care	**Indoor environment:** • overwhelming size • no orienting cues • lighting/sun glare • cluttered environment • contrasting floor surfaces suggest steps • noisy environment • too many others or isolated within the group • personal space not personalised • difficultly finding their room or space • culturally or spiritually inappropriate or offensive objects • lack of space to pray or participate in spiritual/ religious activities • lack of privacy **Outdoor environment:** • lack of points of interest or colour • insufficient shade • does not encourage sitting, engagement or walking • not readily visible or accessible from indoors • physically unsafe • paths lead to frustrating dead ends • previous lifestyle choices regarding outdoors • Aboriginal and/or Torres Strait Islander peoples – impact of removal from Land/*Country* **Stimulation levels:** • lack of structured physical activities • fatigue during the day • boredom/ lack of meaningful activities • overstimulation • under/no stimulation available in the environment • lack of company • dislocation from family, community and/or *Country*

The behaviour or psychological symptom

Assessment of the presenting BPSD requires investigation of the following factors:

- frequency, duration and intensity of the BPSD
- details/clarification of events which occurred prior to the BPSD (antecedents)
- locations where the BPSD occur
- people who are involved with the person with dementia when the BPSD occur
- consequences of, and responses to, the BPSD by other people
- circumstances that prevail when the BPSD are NOT occurring
- extent of discomfort that the BPSD cause the person with dementia
- extent of concern for the person and others in the environment, including the emotional effect on those exposed to the situation.

The greatest challenge for the clinician can be determining what is driving the BPSD for the person, particularly when they live alone or incidents are unwitnessed. Underlying causes may be complex, deep-rooted, silent and/or fluctuating. Sound clinical wisdom and practice will largely guide experienced clinicians. The following modules cover a breadth of published literature to supplement clinicians' expertise.

It is useful to collect information in a format designed to identify people, places and times associated with the BPSD. This encourages a more transparent description of antecedents and consequences as well as helping to establish a clearer impression of the situation and reveal recurring themes. *Table 1.2* provides a list of potential factors and cultural considerations, including those relevant to Aboriginal and Torres Strait Islander peoples and people from CALD backgrounds, which contribute to BPSD and should be considered during assessment. Useful sources of information include:

- Discussion with the person's **general practitioner**.
- Discussion with **care partner, care staff and/or family** (if indicated and available) to clarify the person with dementia's current functioning and assess impact on others. Be aware of how different understandings and perceptions of dementia and BPSD can affect descriptions of the situation.
- **Behaviour charting** over several days may provide accurate, objective information and a baseline measure of the BPSD. Three days is typically suggested for most BPSD however, two sessions of 24 hours each with a break in between is recommended for wandering.
- Reports from **other health professionals** where indicated and available to provide multi-disciplinary assessment e.g. psychogeriatrician, occupational therapist, community service providers, speech pathologist, neuropsychologist, physiotherapist, community nurses, podiatrist.
- **Medication review**, including traditional and non-prescription medicines, by an appropriate clinician is essential in most situations.
- **Additional supports** including progress notes in RACS, community services such as Meals on Wheels, in-home or residential respite providers, neighbours, local café or retail outlet staff and/or visiting friends.

Carers and care relationships

The effect of other people on the person with dementia and the way the BPSD affect others will, in turn, influence BPSD. Carers may unintentionally prompt BPSD when they are unaware of their impact on the person. Factors relating to care partners, care staff, clinicians and/or family members include their:

- knowledge, skills, experience, understanding and training in dementia
- Aboriginal and/or Torres Strait Islander or CALD background
- knowledge of, and history with, the person with dementia
- general emotional and physical health
- informal and formal support, as well as services received
- attitude and empathy
- communication and approach toward the person with dementia
- stress levels
- other demands on them
- the nature and quality of their relationship with the person with dementia.

Family and community structures in Aboriginal and Torres Strait Islander communities

Family and community structures, as well as social connectedness, are often extremely strong within Aboriginal and Torres Strait Islander communities[55].

- In some regions different Aboriginal and Torres Strait Islander Nations are considered separate communities, hence an Aboriginal or Torres Strait Islander person may be part of one community or many different communities. Understanding the person's connection to their community and *Land* is important. A service catchment area may include many different communities who are traditionally from that area or not.
- Knowledge of the local situation will assist services to appreciate the differences and/or tensions between the families/communities/groups in a geographic area and how this can impact on service provision.
- The concept of family is not restricted to westernised bloodlines and the community is typically not limited to family/blood connections but may include wider kinship.
- Families tend to be large. Home visits to an Aboriginal or Torres Strait Islander person with dementia may include many family members but the key person or people to provide information need(s) to be identified.
- The decision-maker for the person with dementia may not be a blood relative or geographically close. Access and contact may be limited as a consequence, but their role must be respected, nonetheless.
- Clinicians need to be aware of the status of an older Aboriginal or Torres Strait Islander person within their community and any potential implications dementia and the need for care may have on their status.
- The leadership responsibilities of older Aboriginal and Torres Strait Islander people can come with a heavy workload. The demands of their position may be incongruent with their health status and/or capacity.
- Because services may be dealing with the entire family or kinship, there are often multiple carers involved and some may be young.
- Aboriginal and Torres Strait Islander people with dementia are typically younger at age of onset, hence issues for their children and/or grandchildren may also be relevant.
- Aboriginal and Torres Strait Islander older people are often carers for the extended family and hence, greater flexibility in approaches to care is required.
- An Aboriginal or Torres Strait Islander person with dementia may present across multiple services in different areas when care is shared between many family members. Usual service guidelines on communicating with relevant others may require clinicians to maintain contact with other services out of area.
- Awareness of the local history of the place, family and communities is important in an understanding of the cultural losses experienced by the local Aboriginal and Torres Strait Islander people. This is relevant to the subsequent strengths and resilience that has developed within families and communities.

Additional considerations for CALD carers and families

The expected roles of family members in the care of people with dementia varies across different CALD communities and families, as does the importance placed on the care of elderly family members[56-60].

- There can often be multiple carers for CALD people with dementia, with the entire family sometimes involved. However, the decision-making role may not necessarily be undertaken by a primary carer. For example, if a father who previously made all the family decisions has dementia the wife will typically continue her role as carer, in relation to his physical needs. Their son or another male family member however, may assume the decision-making position. This can include family members living overseas.
- It is important to understand the multiplicity of carers and decision-makers when dealing with a person with dementia from a CALD background, as this will influence the information gathered and care relationship.
- As in all communities, denial can occur within the family around the symptoms of dementia. The propensity for this increases when family members are living overseas. It is important to involve families throughout the assessment process.
- Home visits to the person with dementia may involve many family members who wish to be present. Be aware that family members may have conflicting views about care. It is essential to identify the key person or people to supply information.

The care environment

The care environment includes the physical, social and cultural dimensions of the environment in which care is provided. These factors are interconnected and can impact on BPSD:

Physical factors

The physical and sensory environment is well documented as a contributing factor in BPSD. It includes a diverse range of elements including noise, access to outdoors, safety, security, glare, lighting, layout, size, furnishings, space for wandering, number of people, traffic through the area and time of day. Observation and discussion with the person's support network will provide opportunities to uncover elements of the physical environment which are disorienting, limiting, unsupportive, confusing, not enabling, over stimulating, under stimulating and/or impacting on the person with dementia in some way.

Social and cultural factors

The social and cultural dimensions of the living environment include all people who come into contact and interact with the person with dementia, and the shared values and practices of the group(s) in the person's immediate environment.

- The quantity, quality and type of activities offered to the person with dementia and the level of meaningful social interaction they receive contribute to BPSD, as can the way care and support are provided, including routine, flexibility and interaction with the person.
- The overall cultural appropriateness of the person's environment can contribute to BPSD. Family or multicultural health workers/advisors may be able to provide input.
- The support provided to informal and formal carers can affect the time and resources available to provide care. Discussions with care partners, service providers' management, different levels of staff, family members and clinicians may be necessary to assess these aspects of the care environment.

Additional considerations related to the care environment for Aboriginal and/or Torres Strait Islander Peoples

Services specifically tailored for Aboriginal and Torres Strait Islander peoples are limited, emphasising the need for service providers to ensure care is delivered in a culturally competent manner[55].

- In acknowledging the holistic view of health, care services for an Aboriginal or Torres Strait Islander person with dementia must be linked to services for other family and community members of all ages who are also affected by the dementia and BPSD.
- Person-centred care for those with dementia in Aboriginal and Torres Strait Islander communities needs to be family-centred and community-centred.
- In some cases, stereotypical and/or racist labels, attitudes or behaviours may be subtle but can, nonetheless, reinforce stigma and pose a major barrier to providing culturally safe, trauma-informed care.
- Hospitals and RACS are typically not set up to accommodate large extended family groups visiting. Where possible, a room with alternate access will minimise disruption to other patients or residents. Likewise, an additional telephone line will allow increased contact with kinship and community.
- Aged care and dementia services must integrate family, culture and community into their service delivery model[61].
- Separation from *Country*, family and community, particularly when admission to an aged care home is necessary[62], can precipitate BPSD. Many Aboriginal and Torres Strait Islander people do not live on their *Land*, although this may not be by choice.
- Enabling physical and/or visual access to the outdoors can be important for an Aboriginal and/or Torres Strait Islander person.
- Separate men's business and women's business may need to be considered around staff rostering.
- Hospitals, and by association RACS, have historically been seen as places to die, not places of healing[8].
- Emotional and spiritual aspects of the care environment warrant careful consideration.

Additional considerations related to the care environment for people from CALD backgrounds

Where residential aged care is indicated, an attempt should be made to arrange admission to an ethno-specific aged care home before accepting a place in a mainstream facility. As this is often not possible, mainstream residential staff members need to have a basic awareness of the potential antecedents that could make the person with dementia uncomfortable and/or possibly trigger BPSD.

Culturally competent workers:

- Bilingual and/or bicultural clinicians or workers should be engaged in the provision of care in community and residential care settings wherever possible. See *Communication* section of this module for further information.
- This can assist with building trust, promoting self-care for carers and recognising the significance of culture in the care of the person with dementia[23].
- It can be useful to identify whether a RACS has bilingual staff who speak the same language (and dialect) as the person with dementia and how much contact they have with the resident. Consideration should be given to scheduling rostered hours to maximise coverage of those who speak the client's language throughout any 24-hour period.
- Building this link may assist with providing successful interventions. While the involvement of a bilingual and/or bicultural worker can make a service more acceptable to the older CALD client, this is not always the case, particularly in relation to RACS[23].

It is important to be aware of potential impacts of dementia care provided by a culturally diverse workforce[63]. Racism from the person with dementia directed toward workers and vice versa in RACS and community care settings may be a concern. The possible implications of this for care and for BPSD are significant. For example, racism can be an issue which precipitates BPSD in older war veterans when care is provided by staff of "Asian appearance" or those with an apparent German accent.

Analysis of the comprehensive assessment

Analysis of the information gathered during assessment should help to identify factors which separately or collectively suggest potential causes of the BPSD and provide a basis for a behaviour support plan.

Frameworks for analysis

A framework or structure to assist analysis of the assessment information can be useful. Examples of such models follow.

The **Progressively Lowered Stress Threshold (PLST)** model is based on the proposal that the person with dementia is increasingly less able to manage stress as dementia progresses. This approach focuses on supporting the person by facilitating the use of retained skills and abilities while reducing environmental triggers for BPSD[64-67]. This is a useful model for analysing assessment information against stressors such as:

- fatigue
- change of routine, environment or carer
- internal or external demands that may exceed functional capacity
- misleading stimuli or inappropriate stimulus levels
- affective response to perceptions of loss, including anger or depression
- physical stressors, such as acute illness, adverse reactions to medication, infection, pain or discomfort.

The **Need-Driven Behavioural** (NDB) model[68, 69] is suitable for use independently or in combination with the PLST model. The NDB model proposes that BPSD are an indication of unmet needs, as the person with dementia becomes progressively less able to meet their own needs. The model considers the influence of *background factors* including neurological, cognitive, health and psychosocial aspects as well as *proximal factors* such as aspects of the person and the impact of environmental factors[70].

A **Concept Mapping** approach incorporates visual representations of the structure and relationship between linked ideas or knowledge[71, 72]. This model aims to improve understanding of the complex, interacting factors contributing to BPSD and hence, facilitate development of an effective behaviour support plan[73, 74].

The **A_B_C** model utilises behavioural observation and analysis to develop strategies for modifying BPSD[75]. **A** refers to the antecedent or potentially triggering event that precedes the changed behaviour or psychological symptom, **B** is the behaviour and **C** is the consequence of the BPSD. The importance of observing and recording BPSD is emphasised. The goal of the **A_B_C** approach is to reinforce appropriate behaviours while discouraging those that are dysfunctional.

Behaviour support plan

A behaviour support plan, based on analysis of the comprehensive assessment and clinical judgement, should be prepared in partnership with the person's support network to ensure their cooperation and understanding. The aim of a behaviour support plan is to eliminate the

inappropriate use of restrictive practices in aged care. It is important for behaviour support plans to include best practice support strategies, be responsive to individual's needs, and provide individualised support[76]. Dementia Support Australia (DSA) offers resources and templates to assist in the development of behaviour support plans. See *Restrictive practices and BPSD* section of *Module 2* for further information on restrictive practices.

According to section 15HA of the Quality of Care Principles 2014[77], the following responsibilities relate to behaviour support plans:

(1) If: (a) an approved provider provides aged care to a care recipient; and (b) behaviour support is needed for the care recipient; the approved provider must ensure that a behaviour support plan for the care recipient is included in the care and services plan for the care recipient.

(2) The approved provider must ensure that the behaviour support plan:

(a) is prepared, reviewed and revised in accordance with this Division; and (b) sets out the matters required by this Division and Divisions 3 and 4.

(3) In preparing the behaviour support plan, the approved provider must take into account any previous assessment relating to the care recipient that is available to the approved provider.

A behaviour support plan should include the following elements:

- a description of the changed behaviour or psychological symptom for which the person needs support
- baseline measurement of frequency and severity of BPSD
- any urgent actions implemented after the initial risk assessment
- possible precipitating factors
- details of strategies/interventions previously trialled
- aspects of care, treatment, daily routine, environment and carer relationships relevant to providing support
- resources, steps, strategies and changes necessary to implement the plan
- details of who has provided consent, where required
- timetable and milestones for implementation
- tools for monitoring and evaluating the BPSD, any changes and outcomes of interventions;
- date for review.

Part 4A of the Quality of Care Principles 2014[78] set out the requirements that must be met for the use of restrictive practices in residential aged care settings. These specific requirements do not apply to aged care services delivered in a home or community setting. However, under 15NB(2A)[78], a provider of services delivered in home or community settings must meet certain requirements for the use of a restrictive practice. These requirements include (but are not limited to) documenting in the care recipient's care and services plan the behaviours that are of concern and the circumstances for when the restrictive practice may be used. If the use of the restrictive practice does not meet the requirements as set out in section 15NB(2A), the provider must submit a reportable incident notification, under the Serious Incident Response Scheme to the Aged Care Quality and Safety Commission.

Planning and communicating the intervention

Interventions that are individually tailored to the person living with dementia will likely be most effective in moderating BPSD[79]. An intervention which is soothing and calming to one person with dementia may be perceived as an invasion of personal space by another[80]. The strengths and limitations of the person's environment are equally important[81]. Interventions may involve psychosocial, environmental, biological and/or pharmacological methods or a combination

of strategies, dependent on the outcome of the assessment[82]. Expert consensus guidelines recommend psychosocial/environmental interventions as a first-line approach when the person and/or others are not at risk and short-term pharmacological/ biological interventions only when necessary[83].

Pharmacological strategies should be considered if non-pharmacological interventions have been unsuccessful. Consultation with the person with dementia and their carers/person responsible, as well as evidence-based guidelines, is essential prior to medication being prescribed[84]. The least harmful medication at the lowest dose should be used and time limited. Pharmacological interventions must be reviewed systematically and discontinued where no efficacy is evident[85]. See *Appendices 2 and 3* for tables of summarised intervention studies.

When planning and communicating interventions it is important to involve care partners, care staff and family in the development of clear goals/outcomes for each aspect of the intervention. Consider the following aspects:

- ability of informal and formal carers to implement the changed approach based on their current skills and resources
- education and additional support required to assist application of new knowledge and strategies
- expectation of the time for changes to impact on the BPSD, and
- where indicated, the estimated time needed for a pharmacological agent to provide therapeutic benefits.

Review and evaluation

It is important to follow-up, review and evaluate the effectiveness of an intervention or strategy after change has been implemented. Ongoing monitoring of the situation enables interventions to be adjusted as needed. The time frame for introducing an intervention or strategy and evaluation of the effectiveness will depend on the individual circumstances. Decide on an appropriate date for review and evaluation when implementing the behaviour support plan. The measure of effectiveness to be applied will be determined by the BPSD and the individual situation. Appropriate measures may assess reductions in the frequency, intensity and/or the impact of the BPSD on the person and others. The Dementia Outcomes Measurement Suite (DOMS) provides validated tools for health care professionals to assess various aspects of dementia, including BPSD. Where BPSD are not resolved see Dementia Support Australia (DSA) for referral to the Dementia Behaviour Management Advisory Service (DBMAS) or Severe Behaviour Response Teams (SBRT) for further expert support.

Additional considerations for dementia and BPSD in Aboriginal and/or Torres Strait Islander communities

Aboriginal and/or Torres Strait Islander peoples are a diverse group, comprised of many language groups. While most Aboriginal and/or Torres Strait Islander peoples live in non-remote areas, the population is widely spread across Australia. As a result, a higher proportion of Aboriginal and/or Torres Strait Islander people live in remote areas[86], than their non-indigenous counterparts, where access to health and support services is typically limited[14, 47, 87]. To understand the multitude of factors that may affect dementia and BPSD within Aboriginal and Torres Strait Islander communities it is important to recognise their unique cultural and historical background[88, 89].

Pre-contact Aboriginal and Torres Strait Islander communities

While estimates vary, current research indicates that Aboriginal and Torres Strait Islander peoples have lived in Australia upwards of 60,000 years[90]. It is estimated that prior to colonisation, around 250 different language g roups i ncorporating 6 00 d ialects w ere spoken by Aboriginal peoples[91]. Aboriginal people were semi-nomadic and lived in communal family

groups[92]. Membership of family groups was founded on birthright, shared language and cultural obligations and relationships, with the roles and relationships between different family members predetermined. Family groupings were diverse, with their own ancestry, history and culture[93]. Cultural beliefs placed an emphasis on the role of social, spiritual and religious dimensions in shaping day-to-day living and the environment[94, 95].

Torres Strait Islander peoples are a diverse population, reflecting differing conditions across the islands as well as interactions between populations in Papua New Guinea and the Cape York Peninsula. Torres Strait Islander peoples tended to live in communal villages, relying on inter-island trade of mainly agriculture and/or fishing. They were skilled sailors and navigators[92]. Fundamental to the belief systems of both Aboriginal and Torres Strait Islander peoples is *Land*[92]. Dreaming creation stories provide the basis for individual and collective spiritual connections to particular *Country*[96]. In contrast to European understandings, *Land* is not owned. Aboriginal and Torres Strait Islander people maintain a strong symbolic and spiritual experience of, and connection to, the *Land* "based on a philosophy of oneness with the natural environment"[92].

The impact of colonisation and oppressive legislation

The impact of colonisation by European settlers in 1788 was marked by death, displacement and disease for Aboriginal and/or Torres Strait Islander peoples. They experienced the dispossession of *Land*, introduction of diseases and direct warfare with colonisers[97]. Under federation, States and Territories introduced oppressive and racist policies and laws to restrict the rights of Aboriginal and Torres Strait Islander peoples and alienate them from their culture[92, 98]. Whilst such legalisation was abolished in the late 1960s, direct and indirect effects of policies continue to impact the lives of Aboriginal and/or Torres Strait people today[99, 100].

One of the most notable ongoing effects of colonisation arises from the forced removal of generations of Aboriginal and Torres Strait Islander children from their families, known as the Stolen Generations[99]. Children who were forcibly removed from their communities and culture were placed in institutions[99]. Reports indicate that during the first half of the 20th century, when the practice was formalised by Government policy, as many as 1 in 3 Aboriginal and/or Torres Strait Islander children may have been removed from their families[101]. Exact numbers are not known as records were often lost or destroyed. Important cultural, spiritual and family ties have been lost leaving a lasting, ongoing impact on the lives and wellbeing of Aboriginal and Torres Strait Islander peoples[102]. The ongoing effect on families and individuals can be profound and can contribute to psychological and/or psychiatric symptomatology[91]. Childhood trauma has also been identified as a risk factor for dementia[103]. Stolen Generations survivors are now aged 50 plus and eligible for aged care services[101].

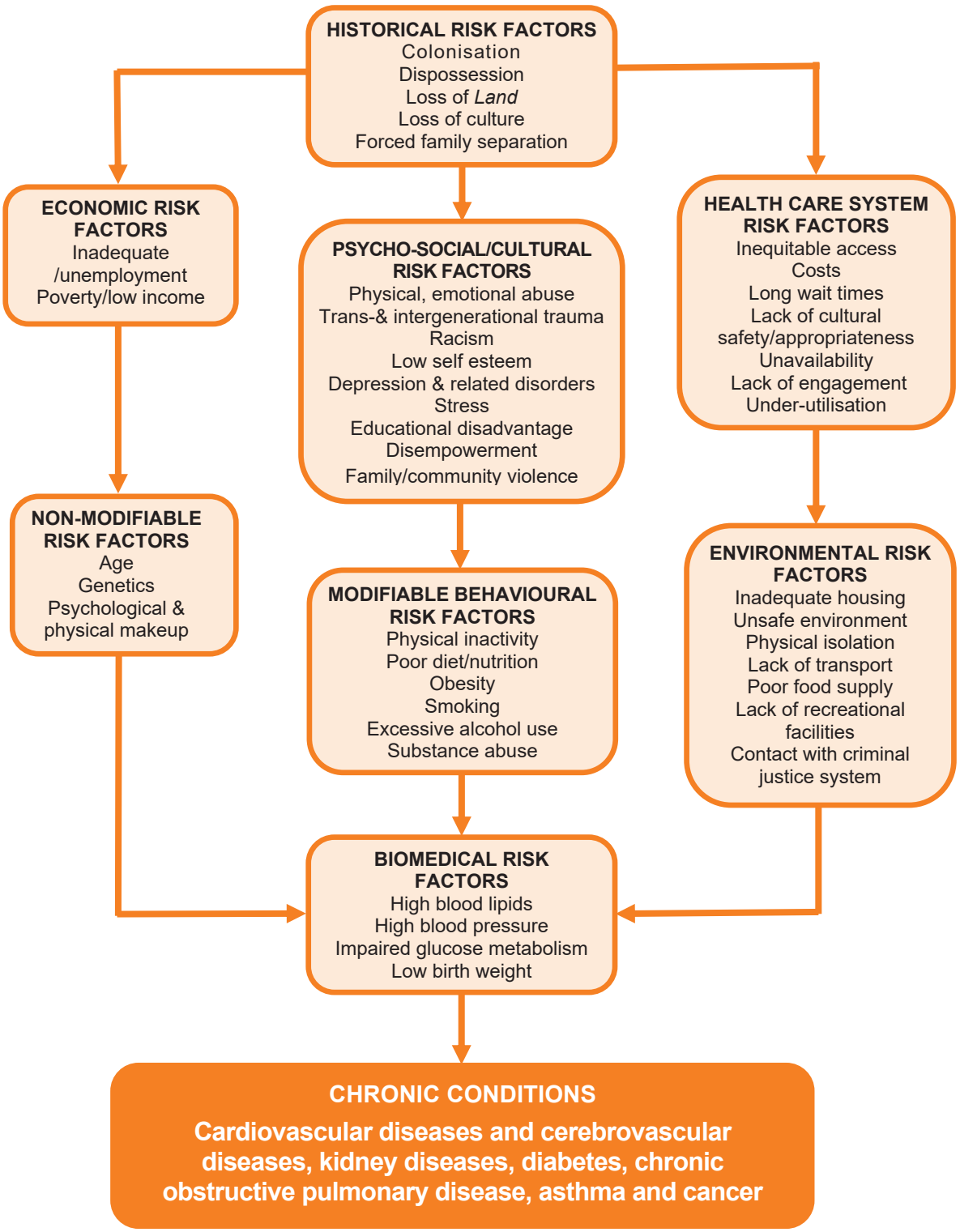
Past practices have been officially recognised, documented and acknowledged by Australian Governments and policy changes since the 1960s have targeted increased access to services for Aboriginal and Torres Strait Islander peoples[104, 105]. Although a National Apology has been issued[106] and attempts have been made to address earlier injustices and inequities, the legacy prevails and the impact continues [107]. Depression in dementia may occur in the context of lived experience or family history of the Stolen Generations and/or other complex grief and trauma. Monitoring changes in social and cultural connection are important to support social and emotional well-being[46]. The fear of transfer from community and/or *Country* to RACS is one example of the enduring relevance[62].

Service access issues for Aboriginal and Torres Strait Islander peoples

As a result of colonisation, successive oppressive government policies, and the significant loss of land, Aboriginal and Torres Strait Islander people are the most economically and socially disadvantaged population groups in Australia[51, 91, 108, 109]. Compared to non-Indigenous people, Aboriginal and Torres Strait Islander people continue to experience poorer outcomes across all major determinants of health and wellbeing including housing, education, employment and engagement with the justice system[51, 91, 109, 110].

Trans-generational trauma is the impact of past and current historical and political events on current generations. This remains a significant issue for Aboriginal and Torres Strait Islander communities[104, 111]. Past government policies created dependence which ultimately led to the breakdown of vital social structures and values[23]. This is apparent in the levels of family violence, substance misuse and family dislocation experienced within the community[23, 104].

Figure 1.2 Factors contributing to chronic disease in Aboriginal and Torres Strait Islander peoples, adapted from NSW Health, 2005 [101, 112-115]



Research has consistently shown that while Aboriginal and Torres Strait Islander people are less likely to consume alcohol compared with the non-indigenous people, those that do consume alcohol are more likely to do so at harmful levels[116]. The excessive consumption of alcohol often relates to trans- and intergenerational trauma and resultant cultural dislocation[112]. Systemic and individual racism continues to maintain the legacy of colonisation and impact on current generations of Aboriginal and Torres Strait Islander communities[112]. Experiences of racism negatively impacts on individual physical and mental health. Older Aboriginal and/ or Torres Strait Islander peoples who access services have reported negative experiences including stereotyping and racial discrimination and as a result are less likely to access services when needed[114].

In line with a social determinant perspective of health, *Figure 1.2* provides an overview of the multiple factors that shape chronic health outcomes for Aboriginal and/or Torres Strait Islander peoples. These include socio-economic factors and a higher incidence of chronic conditions associated with an increased risk of dementia, such as cardiovascular disease, renal disease and diabetes[103, 117, 118]. Dementia can occur against a background of trauma and multiple losses. Where the losses are not acknowledged by society (e.g. by offering appropriate services) disenfranchised grief carries an increased risk of complex grief reactions and potential complications which can impact on BPSD. An older Aboriginal and/or Torres Strait Islander person with dementia may also have unfinished business around historical issues which can impact on the presentation of dementia and BPSD[119-121].

Poor health outcomes secondary to social disadvantage, experienced by Aboriginal and Torres Strait Islander peoples, require that health interventions address issues shaped by the broader social and economic conditions such as poverty, education, racism and housing[1229]. Strategies that address the historical and ongoing impacts of colonisation and marginalisation which contribute to racial inequality and disadvantage must be considered[113]. An understanding of the social determinants of health affecting Aboriginal and Torres Strait Islander peoples is essential to developing effective partnerships and working toward reducing the impact of past practices. Clinicians who are cognisant of the multiple historical factors impacting on the individual presentation of BPSD in an Aboriginal or Torres Strait Islander person may approach the situation more appropriately[114].

Despite the negative impacts of colonisation and associated practices which continue in their communities, the significant resilience of Aboriginal and Torres Strait Islander peoples must be acknowledged. Connection to culture is reported as a key to many Aboriginal and Torres Strait Islander people's identity and strength. Culture is a foundation on which wellbeing can continue to be built[123]. Strength within communities is evidenced by:

- a strong commitment to family and community
- valuing cultural heritage and spirituality
- continued connection to *Land*
- traditional knowledge of ancestry, health, wellbeing and *Country*
- traditional methods of storytelling and healing
- traditional *Lore* and ceremony
- traditional roles of Healers, Elders and community carers[32, 52, 124].

These aspects provide a source of strength for an Aboriginal and/or Torres Strait Islander person with dementia and their family and it is important to recognise and acknowledge them in addressing BPSD as part of person/family/community-centred care. It is equally important to recognise that historical issues have broken down the resilience of some Aboriginal and/or Torres Strait Islander communities, leading to significant health and socio-economic issues[51, 87] as well as a lack of trust in health care and service providers[46].

Additional considerations for dementia and BPSD in CALD communities

An estimated 30% of people living in Australia were born overseas[125]. Awareness of the potential impact of culture and migration experiences on care needs[126], as well as the course of dementia and BPSD, is important. Older migrants and refugees may have an increased risk of dementia related to trauma from displacement and social isolation after arrival in a new country. Many immigrants are also from low- and middle-incomes countries with lower education and literacy levels and limited knowledge of dementia[127, 128].

History of migration and settlement in Australia

Under Australia's Immigration Restriction Act [1901], more commonly referred to as the White Australia policy, the intake of non-European migrants was actively limited and British migrants promoted[129]. However, in response to population decline, Australia launched a post-World War II immigration program from 1945 to attract displaced European migrants[130]. In the 1970s, following the formal abandonment of the White Australia Policy, large numbers of non-European migrants were encouraged to settle in Australia. These migrants were primarily from Asia and the Middle East. Further, Australia's Humanitarian Program[131] supported refugees and others in refugee-like situations escaping areas of war and/or violence[132] to resettle in Australia since 1977[133]. The 1951 Refugee Convention defines a refugee as "someone who is unable or unwilling to return to their country of origin owing to a well-founded fear of being persecuted for reasons of race, religion, nationality, membership of a particular social group, or political opinion"[134]. Some immigrants have arrived in Australia older as part of our Humanitarian Program however, successive waves of migration have also resulted in multiple different ethnic groups reaching old age over the decades[132].

Factors that may trigger or contribute to BPSD

It is important to gather as much information about the person's life history. Not only their cultural background, but also their migrant and settlement experiences as these can impact on BPSD. When carers and service providers have no knowledge or understanding of these experiences, BPSD can be inadvertently exacerbated.

Migrant settlement experiences

Many older migrants in Australia arrived and settled in the White Australia policy period. During this time, migrants were forced to assimilate, often suppressing their own culture and language to adopt "Australian ways". Consequently, in later life older migrants may have lost those connections with their past. Many migrants faced racism and discrimination when they attempted to settle into Australian society. For example, on arrival, many migrants unwillingly had their names and surnames changed by government authorities to make them more appropriate for Anglo-Saxon society. With the onset of dementia, older migrants may revert to their previous name and not respond to the name that family and friends know them by.

Discrimination at the time of migration and settlement can result in a loss of identity, which may remain unresolved. Older migrants with dementia may relive previous traumatic experiences and memories. Stories from early life may have remained untold for many years due to the associated shame, pain and/or trauma. Children of migrants with dementia may thus be unable to report the person's history that is potentially contributing to BPSD. They may feel that they no longer know their parent or misunderstand the situation when the BPSD appears to be out of context.

Some migrants to Australia may have learnt English for the workplace, however following retirement they may not have used English regularly for many years. With increasing age and/or dementia, the need to interact within the health system arises but they may find it difficult to communicate. As a result, older migrants may be labelled as resisting care, uncooperative or exhibiting BPSD, when in fact they are struggling to communicate.

Refugees, asylum seekers and war-time experiences

Refugees and refugee-like people often come from situations where they have encountered significant traumatic events such as war, persecution or harassment by government authorities, torture, rape, witnessing loved ones suffer violence as well as the disappearance and death of family and friends, including children[126]. Torture is used to control individuals and societies by fear, impacting whole population groups not only those being tortured[135]. Fear can cause the person to hide their past, making it difficult for clinicians and care staff to recognise their additional needs. While refugees and asylum seekers may be from diverse cultural backgrounds, they often share experiences, including the hardships of flight, stigma, discrimination, isolation, financial insecurity and protracted asylum processes, including detention[136].

- Past experiences can influence the presentation of changed behaviours or symptoms in dementia such as hoarding food, feeling like they are being watched, hallucinations, misinterpreting people/strangers as threatening and/or re-experiencing the trauma over the loss of a child or family member[132]. Service providers may be seen as representatives of government agencies.
- Because of past fears, for many refugee and refugee-like people, it can be difficult to trust people who work for government authorities and, at times, even their own family[132].
- Refugees in residential care may be reminded of previous trauma by uniforms, corridors and queuing as well as shared spaces for sleeping, eating and bathing.
- Common traumatic triggers for Holocaust survivors include showers, hospital identification bands, medical procedures, surgery and medical gowns[137].
- Hospitalisation is often a traumatic experience for people with dementia from CALD backgrounds, which may worsen BPSD.

It can be challenging for care staff and even family to recognise that a person with dementia is a refugee who requires additional support, particularly when they hide their past. The person may deny their past experiences because of fear of being stereotyped as poor, uneducated or viewed as a victim[132]. It is important for clinicians to explore why the person may be acting this way and how it may be culturally relevant. Clinicians may also need to work with service providers and residential or community care staff to develop an understanding of the reasons and triggers for the BPSD.

Post traumatic stress disorder (PTSD)

PTSD can occur as a result of a refugee, refugee-like or war survivors' past and impact in later life. High quality evidence reports the overall prevalence of PTSD in refugees and asylum seekers is around 31%[138]. While many older refugees adapt well to new circumstances, cognitive changes with normal ageing or dementia can undermine this ability to adapt[132]. This can mean that ageing and/or dementia may be an additional risk factor for those who have experienced trauma in the past and this can trigger changed behaviours[139, 140]. With cognitive decline, PTSD severity in Holocaust survivors may worsen as repressed memories come to the fore[137]. People with dementia have a diminished capacity to hold back traumatic memories, which can result in a re-emergence of PTSD symptoms, including feelings of anxiety and depression[137, 141]. It is important to distinguish between PTSD and BPSD as they may occur independently or in combination[139].

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

2. General BPSD

MODULE 2: General BPSD

Key messages

- Behaviours and psychological symptoms associated with dementia (BPSD) impact significantly on the person living with dementia, care partners, family, clinicians, care staff and others in their environment.
- Manifestations of BPSD involve changes to the person's perception, thought content, mood and/or behaviour and these are influenced by a wide range of factors.
- BPSD occur in people living in the community and residential aged care services (RACS), as well as during hospital admissions, with prevalence rates reportedly ranging between 56% and 90%.
- BPSD can be conceptualised as a response to deficits in care (unmet needs), as a reaction to lowered stress thresholds (due to dementia), as a manifestation of brain pathology or changes in brain chemistry and/or a reaction to biological factors.
- Ethical considerations, in particular the well-being of the individual, should be a primary consideration in the implementation of interventions.
- Psychosocial, environmental, pharmacological and biological interventions for addressing BPSD are not mutually exclusive.
- Effective psychosocial/environmental strategies include therapeutic social/leisure activities, cognitive rehabilitation, reminiscence, music, exercise, animals, sensory stimulation, education for carers and/or staff, touch therapies, models of care and multicomponent interventions.
- Potential side-effects must be carefully weighed against potential benefits to the person with dementia when considering pharmacological interventions for addressing BPSD.
- Pain and trauma can affect the presentation of BPSD and must be considered in the approach to treatment.
- Restrictive practices, including chemical restraints, must only be used as a last resort, for as short a time as possible, and after all legislative requirements have been met.
- Monitoring and evaluating the impact of an intervention is an integral part of addressing BPSD.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

General BPSD Summary

What are general BPSD and what do they look like in dementia?

- Behaviours and psychological symptoms associated with dementia (BPSD) are defined as 'symptoms of disturbed perception, thought content, mood, behaviour frequently occurring in patients with dementia'.
- They are also commonly referred to as behaviours of concern, responsive behaviours, challenging behaviours and non-cognitive or neuropsychiatric symptoms of dementia.
- The most common BPSD are included in this *Guide*: aggression, agitation, anxiety, apathy, delusions and hallucinations, depression, disinhibition, nocturnal disruption, vocal disruption and wandering.
- BPSD occur at different levels of severity with the stages of disease progression. The seven-tiered model of BPSD by Brodaty and colleagues demonstrates the wide range of symptoms and severity encompassed by the term BPSD.

Prevalence of BPSD

- Reported prevalence rates of BPSD range between 56% and 90%.
- BPSD occur in community, hospital and RACS however, they tend to occur more frequently in residential aged care homes.
- The prevalence of dementia in Aboriginal and Torres Strait Islander people in some areas is reportedly three to five times higher than that of the general population, suggesting the potential for high rates of BPSD.
- The culturally and linguistically diverse (CALD) population is ageing at a greater rate than mainstream communities; their prevalence of dementia is predicted to rise significantly.
- The most frequently occurring BPSD are apathy, depression and anxiety although individual BPSD fluctuate over time, with many behaviours occurring episodically.

Effects of General BPSD

- BPSD impacts significantly on the person living with dementia and their carer(s).
- The quality of life of the person with dementia can change considerably, particularly during relocation to a residential aged care home.
- Pain and other unmet physical and/or psychosocial needs may provoke BPSD.
- BPSD contribute to stress and burn-out of residential care staff, particularly where support from management is lacking.
- The degree of carer burden can impact significantly on their ability to address BPSD.

Differential diagnosis

It is important for clinicians to distinguish between BPSD and delirium for appropriate treatment options to be implemented. Delirium superimposed on dementia is often under-recognised. Delirium can be identified by an abrupt onset of behaviour which is out of character for the person with dementia.

The potential impact of pain on BPSD

People with dementia are at risk of under-recognised, underestimated and under-diagnosed pain and the risk increases with dementia severity as their capacity for self-report diminishes. Poorly managed pain can contribute to BPSD.

The potential impact of trauma on BPSD

Many people with dementia have experienced trauma, grief and loss in their life. Knowing how

trauma affects the person can assist in understanding their needs and the presentation of BPSD.

Measuring BPSD

- Tools for measuring BPSD globally include the Neuropsychiatric Inventory (**NPI**) and the Behavioural Pathology in Alzheimer's Disease (**BEHAVE-AD**).
- Aboriginal and Torres Strait Islander, and CALD populations require the use of assessment tools specifically developed for these populations, wherever possible.

Results

Outcomes from the literature search (2012-21) resulted in 113 psychosocial/environmental and 69 biological/pharmacological intervention studies reporting outcomes for general BPSD, that met our quality inclusion criteria.

Addressing BPSD

- The treatment of BPSD can include psychosocial, environmental, pharmacological and/or biological interventions. It is important to note that these are not mutually exclusive.
- Unless the person with dementia is very distressed or at risk of harm to themselves or others, introduce psychosocial methods first and attend to environmental contributors to the BPSD.
- Individually tailor interventions to the person and monitor symptoms for a suitable period before considering pharmacological therapy, as symptoms may resolve spontaneously or in response to psychosocial interventions.

Adverse effects of psychotropic medications

The use of psychotropic medications, particularly antipsychotics, in dementia has been associated with higher risk of functional and cognitive decline as well as serious adverse effects. Their use should be reserved for severe symptoms that are unresponsive to nonpharmacological strategies.

Restrictive practices and BPSD

A restrictive practice restricts a person's rights or freedom of movement for the primary purpose of influencing their behaviour. People with dementia are reported as being more likely to be subjected to the inappropriate use of restrictive practices. Serious adverse physical and psychological outcomes have been associated with restrictive practices, including the death of residents in residential aged care homes.

Limitations

Limitations in the methodology of the studies included in this review inevitably affect the validity of the outcomes reported. Difficulties arise when studies treat BPSD as a homogenous group and individual behaviours are not reported separately.

Conclusions/Principles of care

There has been a substantial increase in the number, quality and complexity of studies investigating the efficacy of interventions to address BPSD, particularly psychosocial and environmental approaches.

- Research findings suggest that interventions have a limited effect when applied as a generic treatment for general BPSD, largely attributed to the diverse aetiology of BPSD.
- Person- and behaviour-specific interventions that are tailored to individual situations tend to be the most effective and are recommended.
- Potential side-effects and drug interactions need to be carefully weighed against the potential benefits to the individual person with dementia when considering pharmacological interventions for addressing BPSD.

What are BPSD and what do they look like in dementia?

Behaviours and psychological symptoms associated with dementia (BPSD) include emotional, perceptual and behavioural disturbances. BPSD are defined by the International Psychogeriatric Association (IPA) Taskforce on BPSD[1] as "symptoms of disturbed perception, thought content, mood, behaviour frequently occurring in patients with dementia" [2]. Psychological symptoms of dementia include anxiety, depression and psychosis whereas behaviours include aggression, apathy, agitation, disinhibition, wandering, nocturnal disruption and vocal disruption. The Diagnostic and Statistical Manual (5th ed.; DSM-5-TR) [3] classifies dementia as a major neurocognitive disorder. Although the presence of cognitive decline and functional impairment are necessary and sufficient for the diagnosis, associated neuropsychiatric symptoms or BPSD can significantly impact on all aspects of dementia. A diagnosis of Alzheimer's dementia might be coded as "major neurocognitive disorder due to Alzheimer's disease, with behavioural disturbances, severe" [4]. The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11 6D86) [5] includes a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" as well as eight individual BPSD under "Neurocognitive disorders, Dementia".

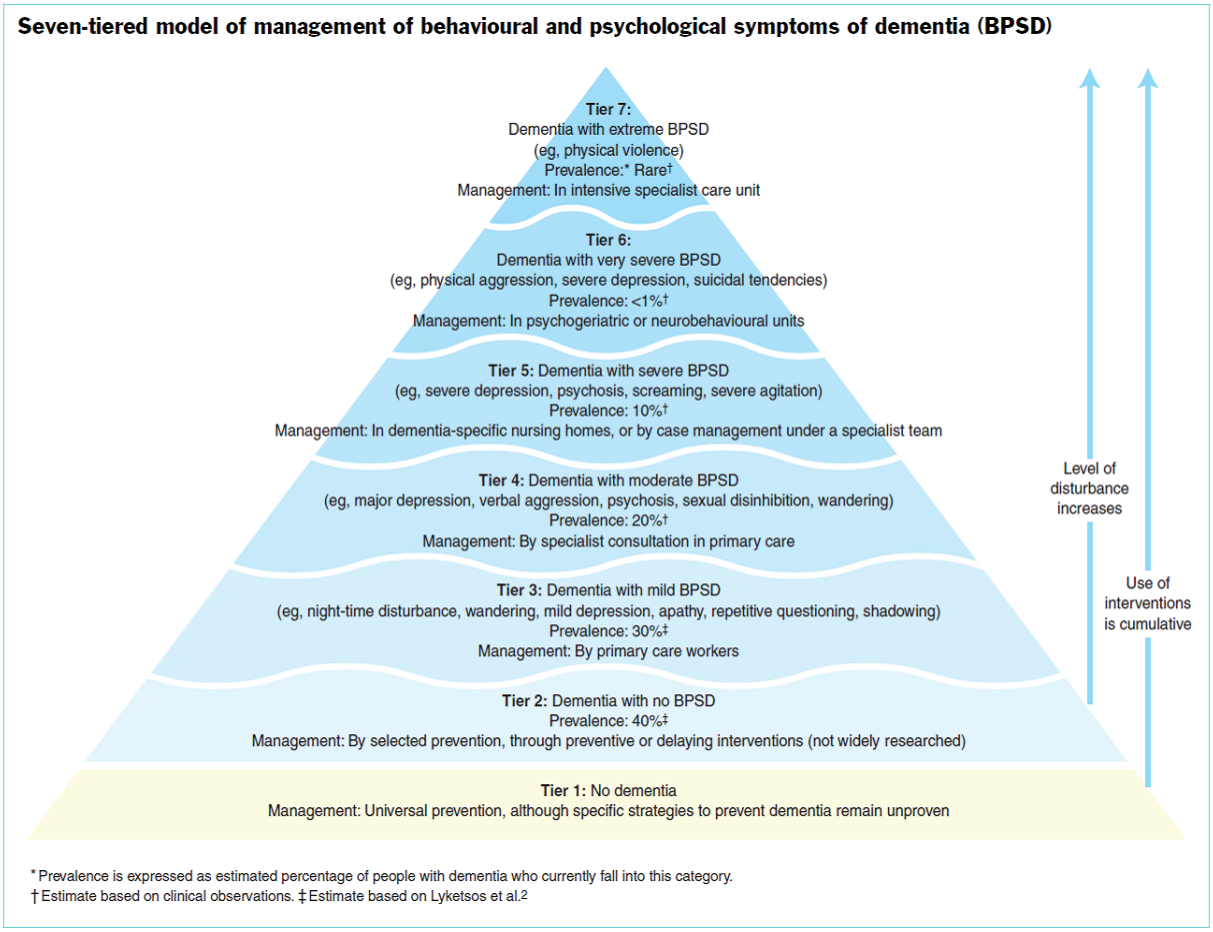
A change in behaviour is typically identified by observation of the person with dementia, particularly when they are at risk of harm or others are affected. The change often indicates a need for assistance or care[6-9]. BPSD are also known as changed behaviours, behaviours of concern, responsive behaviours and non-cognitive or neuropsychiatric symptoms of dementia. Brodaty and colleagues[10] proposed a seven-tiered model (see *Figure 2.1*) for describing BPSD severity and strategies appropriate at each level. The seven tiers reflect an ascending order of symptom severity and descending order of prevalence indicating that the term BPSD covers a wide range of symptoms and severity.

Prevalence of BPSD

While individuals vary, BPSD will occur at some point in up to 90% of people with dementia, during the course of the condition[11-14] and often occur concurrently[15]. BPSD are common amongst both community and residential care dwelling people with dementia with higher rates occurring in residential settings[9, 16]. Reported prevalence rates of BPSD overall range between 56% and 90%[16], with agitation reported as the most common in residential aged care services (RACS) [17]. Aspects of the psychosocial/physical environment are associated with an increased risk of BPSD in residential care [4, 18, 19].

The most frequently occurring BPSD, apathy, agitation, depression and anxiety[11, 20] tend to increase in severity with disease progression, however BPSD can present at any stage [21]. Different BPSD have different trajectories and the baseline characteristics of the person with dementia, including sex and dementia type, predict the subsequent course of symptoms[15]. Alzheimer's disease (AD) has been associated with lower levels of neuropsychiatric symptoms than other types of dementia and frontotemporal dementia (FTD) with greater levels. Dementia with Lewy bodies (DLB) is associated with more hallucinations[17]. Severity of dementia and male sex are also associated with higher levels of BPSD[15, 18]. Prevalence rates should be viewed with caution as figures can vary according to different settings, how symptoms are perceived, tolerated and measured as well as severity thresholds[10].

Figure 2.1 reproduced with permission from Brodaty et al[10]



Prevalence of dementia in Aboriginal and Torres Strait Islander communities

Aboriginal and Torres Strait Islander people are estimated to have dementia prevalence rates that are three to five times that of the general population [22-29] suggesting the potential for high rates of BPSD.

Dementia prevalence for Indigenous Australians in remote and rural communities are among the highest in the world [24, 27, 30]. Biases can occur in estimates used to describe Aboriginal and Torres Strait Islander peoples from measurement issues arising from incomplete and/or inaccurate data. Unreliable data collection can be due in part to how Indigenous Australians are identified as well as the number of Aboriginal and Torres Strait Islander people living in remote locations[31]. Concerns arise where these data are used to identify and address health inequalities. Continuing efforts to overcome data limitations will improve quality, and better support dementia prevalence estimates for Aboriginal and Torres Strait Islander peoples across Australia[32].

Prevalence of dementia in culturally and linguistically diverse (CALD) communities

More than seven million people living in Australia indicated they were born overseas and six million reported speaking a language other than English, in the 2021 Census[33, 34]. According to the 2016 ABS Census, 37% of all Australians aged 65 and over were born overseas, 20% of those aged 65 and over were born in non-English speaking countries and 18% spoke a language other than English at home[35]. The most represented overseas regions of birth for CALD people aged 65 and over were North-West Europe (40.9%) followed by Southern and Eastern Europe (26.7%)[35].

One-third (33%) of people using mainstream aged care services were born overseas. Approximately 40.6% of people using home care, 31.9% of people in permanent residential aged care and 34.4% of those in respite care in 2022 were from a CALD background[36].

Many of the population groups born overseas reported a higher prevalence of dementia than the Australian-born population. Highest numbers were indicated for those from Italy, Iraq and Vietnam[33]. Information on the prevalence of BPSD within and across CALD communities across Australia is lacking. Reporting on numbers of CALD populations in Australia is complex with multiple definitions of the term CALD including country of birth, ancestry, parents' birthplace, language(s) and/or religious affiliation[34].

Effects of BPSD

Impact on the person with dementia

- BPSD significantly impact on the quality of life, prognosis, health outcomes, mortality risk and care needs of the person in community, acute and residential settings[4, 13, 37, 38].
- BPSD predict earlier admission to residential care[39, 40] and relocation frequently triggers or increases changed behaviours in people with dementia.
- BPSD may be a manifestation of adverse effects of medication or unmet physical, medical and/or psychosocial needs[13, 41].
- Clinical changes in the person may trigger BPSD, however BPSD can interfere with the identification of underlying potentially treatable symptoms or conditions such as depression, pain, infection or constipation which can mimic BPSD[42].
- Numerous factors can impact on care partners' tolerance for, and ability to respond to and cope with, BPSD[37, 43] which in turn impact on the person with dementia.

Impact on carers

- Addressing BPSD can be challenging for family carers, community care workers and residential care staff alike, resulting in considerable stress and/or distress[37, 44].
- While the role of care partner is often willingly accepted due to the relationship between the carer and the care recipient, this does not negate the difficulties encountered in addressing changed behaviours and psychological symptoms.
- BPSD are associated with increased burden in care partners and carer depression[45-47].
- Carers' social and psychological resources, as well as their perception of the BPSD, have been identified as predictors of institutionalisation[48].
- Some dementias, such as FTD, and young onset dementia are associated with a lack of suitable resources and support, which can increase carer burden[49-51]. The presence of BPSD can also lead to exclusion from much needed support services or RACS.
- An awareness of the care relationships, knowledge and experience of formal and informal carers as well as resources available is essential when developing a behaviour support plan.
- BPSD contribute to high stress levels and burn-out in residential aged care staff[52, 53]. Support from management and enhancing staff's ability to relate to residents as individuals predict staff members' perception of, and responses to, BPSD[54, 55].
- People with dementia who present with BPSD reportedly have higher healthcare utilisation and associated costs when compared with those who don't[56].
- Dementia Support Australia (DSA) provide expert support for addressing BPSD via referral to the Dementia Behaviour Management Advisory Service (DBMAS) or Severe Behaviour Response Teams (SBRT).

Impact on Aboriginal and Torres Strait Islander family and carers

Because people presenting with BPSD are typically cared for within Aboriginal and Torres Strait Islander families and communities, the situation can be close to crisis point at the time of accessing services. Kinship obligations may contribute to tolerance and care of those with BPSD. Although caring duties may be shared, the ongoing sense of duty to provide care can continue even when community members are close to crisis point.

Aboriginal and Torres Strait Islander peoples reportedly do not access services at a rate consistent with the levels of distress in the communities, and they may have difficulty accessing culturally appropriate care services[57]. Availability of appropriate services are further limited in remote areas which also reduces support for family carers. Older Indigenous Australians generally wish to remain in their communities and on *Country* for as long as possible[58-60]. Strategies to address BPSD must be planned and implemented within the context of the person with dementia's family, carers and community[25].

Impact on CALD carers

Carers of people from CALD backgrounds may focus on how to keep the person with dementia safe and happy[61-64]. This could involve keeping the person occupied, praying for them, and staying at home to avoid dementia-related stigma and discrimination in the community[62-64]. As CALD carers are often more concerned with the person with dementia's physical and other needs and neglect their own self-care and emotional wellbeing, assisting carers with these aspects is important[61, 63, 64].

Carers' migration visas are increasingly becoming a strategy to avoid transfer of a person with dementia to an aged care home in migrant communities. Migrant families who cannot care for their elderly family members with dementia may be able to obtain a carer's visa for a relative to come to Australia and provide the necessary care at home. Currently very little support is available for migrants on a carer's visa. They may have limited English, few caring skills and tend to become socially isolated because they are caring in the home for long periods of time.

Carer strategies

Carer education and/or support which advances knowledge and ability to cope with BPSD may reduce carer burden and potentially delay RACS admission[48, 65, 66]. A review of the literature relevant to this area is beyond the scope of this Guide. Further information on the effectiveness of carer interventions is available through other comprehensive reviews[67-70]. The International Psychogeriatric Association[2] suggests that interventions that target carers of those presenting with BPSD, incorporating the following aspects, are the most effective:

- ensure a focus on the carer as well as the person with dementia
- focus on training and skill building in addition to education and support
- recommend strategies that are multidimensional, flexible, and tailored to the needs of the carers and the person with dementia
- providing information to carers and family that includes techniques for addressing specific BPSD, actions for ensuring the safety and wellbeing of the person with dementia, strategies for dealing with difficulties of activities of daily living (ADLs), and avenues for securing personal assistance, physical aid, financial entitlements and respite services
- collaborate with a professional, such as a nurse working with the carer
- add pharmacotherapy where indicated, only after assessment by a doctor and where other psychosocial strategies have been unsuccessful
- include follow-up assessments.

For further information to support carers of people with dementia see:

Australian Government Productivity Commission 'What Works' review 2018[71] (accessed October 2023).

The *CareForDementia* app (2023)

CareForDementia on the App Store (apple.com)
CareForDementia - App on Google Play

Differential diagnosis - delirium

Serious risk of harm to the person with dementia can arise from underlying, untreated physical or medical precipitants of BPSD. This can manifest as delirium superimposed on the dementia which is often under-recognised[72, 73]. Delirium is a serious condition associated with avoidable functional decline, increased morbidity, prolonged hospitalisation, higher risk of admission to RACS and increased mortality[74-76]. Delirium can be identified by an abrupt onset of changed behaviours, out of character for the person, which develops over hours to days.

Aggression, hallucinations, fluctuating clouding of consciousness, misinterpretation of events, disorganised thinking and sleep disturbance may be evident[77, 78]. A person with delirium may present as agitated and restless (hyperactive delirium), quiet and withdrawn (hypoactive delirium) or they may move between these two subtypes (mixed delirium)[76, 79]. Signs of hypoactive delirium such as slowed responses, reduced mobility and movement, worsened concentration and reduced appetite may be missed[78, 80]. Attempting to control the symptoms with medication can delay diagnosis and lead to poorer outcomes for the person with dementia[81, 82]. Prevention of delirium is the most effective strategy[83, 84]. Where this is not possible, early identification of potential causes and treatment improves outcomes for the person[72].

Detecting delirium

The Confusion Assessment Method (**CAM**)[85] is a scale that enables clinicians or trained lay interviewers to detect confusion due to delirium, with or without dementia. It is suitable for use in older people who are at risk of developing delirium. The CAM has been adapted for use in varied settings including RACS[86] and by telephone[87] and a modified approach for using the CAM with people with moderate to severe dementia is reported[78].

A brief, app-directed 2-step protocol has also been developed, tested and validated in hospital settings to screen for delirium superimposed on dementia UB-CAM Delirium Screen[77, 88-90].

The Delirium Rating Scale-revised-98 (**DRS-R-98**)[91] is a 16-item tool for initial and repeated measurements of delirium symptom severity. Ratings are based on all available sources of information including the person, family, staff and documentation. The DRS-R-98 has excellent inter-rater reliability.

For further information and access to the CAM and the DRS-R-98 refer to the Dementia Outcomes Measurement Suite (DOMS)[92].

For further information on delirium and comprehensive guidelines refer to the following:

National Institute for Health and Clinical Excellence (NICE) *Delirium: diagnosis, prevention and management in hospital and long-term care Clinical Guideline 103*, 2023[76] (accessed October 2023).

Australian Commission on Safety and Quality in Health Care (ACSQHC) Delirium Clinical Care Standard, 2021[72] (accessed October 2023).

ACI Aged Health Network (ACI & CDPC) *Key Principles for Care of Confused Hospitalised Older Persons*, 2015[74] (accessed October 2023).

Table 2.1 Basic guidelines for the recognition and investigation of delirium

INITIATE DIAGNOSIS	Key points
Do not delay	**Delay in initiating diagnosis increases morbidity and mortality.** This is particularly so if it is compounded by inappropriate psychotropic medication with significant probability of side-effects.
Recognise clinical features	**Delirium is often difficult to recognise and assess in an older person.**
Investigate signs/ symptoms of underlying conditions	**Signs and symptoms may be unreliable.** Many older people have multiple comorbidities.
Examine, assess and test	**The presence of nitrites in urine does not preclude further investigation as it does not necessarily indicate a urinary tract infection.** Many frail older people have asymptomatic bacteriuria. There is a risk of other causes being overlooked.
Review medications	**Medications are the most common reversible cause of delirium.** The risk of delirium increases with the number of medications. **Medication reviews are important.**
RECOGNISE	**Presentation**
Clinical features of delirium (see diagnostic criteria: DSM-5-TR)*	✓ Acute onset ✓ Disorganised thinking ✓ Fluctuation which is erratic, and with occasional lucid intervals ✓ Hallucinations ✓ Altered level of consciousness ✓ Inattention ✓ Disrupted sleep-wake cycle ✓ Delusions (paranoid) *No symptom is pathognomonic (specifically indicative) and some of these symptoms can occur in dementia without delirium. Acute onset may not be as evident and the picture may be less florid in the elderly. Older people may have hypoactive delirium in contrast to a typical hyperactive presentation or it may fluctuate between hypo- and hyper-activity.
INVESTIGATE	**Delirium is often difficult to assess in an older person**
Symptoms and signs of underlying conditions	… because common features such as pain and fever may be absent: • A myocardial infarct can occur without chest pain, with abrupt behavioural change as the only indication. • Acute onset of incontinence or retention of urine may indicate a urinary tract infection. • Fever, cough and breathlessness could indicate a chest infection. • Syncopal episodes may indicate cerebrovascular or cardiovascular causes e.g., hypotension, arrhythmia or infarct. • Speech disturbance and paralysis may indicate a stroke or TIA, but confusion may be the only obvious, observable sign.

ASSESS	Careful physical examination, as one would carry out for any acutely ill patient, is essential; check vital signs

Accurate medical history:

- History of onset and course of behavioural changes
- Symptoms suggestive of underlying cause e.g., infection
- Changes in patterns of urination or defaecation
- Previous level of function and cognitive status
- History of acute or chronic pain
- History of acute or chronic sleep deprivation/impaired sleep
- Sensory impairments/decline e.g., visual loss or hearing impairment
- Past medical history including recent illnesses, anaesthetics, etc
- Previous psychiatric history (if any) e.g., mania can mimic delirium and vice-versa

Medication review is essential, especially looking at:

- Current medication including any recent changes in drug or dose
- Prior history of any drug reaction
- Drug toxicity including agents with strong anti-cholinergic activity - tricyclic antidepressants, oxybutynin, benztropine, olanzapine, chlorpromazine, antihistamines and incontinence medications
- History of substance use or recent cessation of heavy alcohol consumption, illicit drugs, opioids, benzodiazepine or other medications

Complete physical examination including:

- Temperature, pulse and blood pressure
- Pain assessment
- Hearing and vision (check aids)
- Dehydration and hypoperfusion states e.g., blood loss, hypoxia and bladder or bowel obstruction.
- Urinalysis - nitrites could indicate UTI, ketones if diabetic

Neurological assessments:

- Cognitive assessment e.g., Abbreviated Mental Test Score or Mini Mental State Exam
- Neurological examination including speech assessment
- Attention assessment e.g., Serial 7s, months of year backwards

Basic screening tests:

Metabolic (renal impairment, dehydration)

- Urea, electrolytes and creatinine
- Liver function tests
- Abdominal X-ray if faecal impaction suspected
- Ultrasound if urinary retention suspected

Infection

- Full Blood Count
- Mid-stream urine
- Chest X-ray
- COVID-19 swab
- Blood cultures, if indicated (bacteria, yeast, fungal)

Endocrine

- Calcium, magnesium, phosphate levels
- Thyroid function tests
- Blood sugar

Malnutrition, malabsorption

- B12, Folate

Acute myocardial infarction

- Cardiac enzymes
- Electrocardiogram (ECG)

Additional tests may be indicated:

- If focal neurological signs, recent fall or head injury, stroke, haemorrhage ⇨Cerebral CT scan
- To exclude seizures e.g., temporal lobe epilepsy ⇨ EEG
- If meningism, headaches and fever ⇨ lumbar puncture
- If trauma possible ⇨ X-ray of suspected fracture site

The potential impact of pain on BPSD

Pain frequently occurs with comorbid conditions in the older person with dementia. Approximately 50% of people with dementia and up to 80% of aged care home residents with dementia reportedly experience pain regularly[93-95]. Those with dementia are at risk of under-recognised, underestimated and under-diagnosed pain[8, 96-101] and the risk increases with dementia severity as the capacity for self-report diminishes[102, 103]. A lack of appropriate assessment, misinterpretation of symptoms and/or inadequate pain management is the likely consequence[103].

Poorly controlled pain can impact on many aspects of the person with dementia's quality of life[104] and contribute to BPSD[41, 95]. Pain can also be misdiagnosed and mistreated as BPSD. The presence of pain has been associated with agitation, anxiety, resistance to care, aggression, depression, poor sleep, socially inappropriate behaviour, delusions and vocalisation in people with dementia as well as the inappropriate use of restrictive practices[2, 7, 105-112].

People with dementia are frequently dependent on carers to recognise their pain and respond appropriately. This may require skilled assessment and interpretation of the signs and symptoms of pain, many of which will be nonverbal. Some indications such as grimacing, moaning or rubbing a body part may be more typically considered pain-related however, BPSD may be unrecognised as an indicator of pain[103, 113, 114]. Further, because BPSD can occur in response to numerous and varied triggers unrelated to pain, assessment may be compounded by other factors. Strategies to overcome barriers to pain assessment in people with dementia include knowing the person, education and training, and using adequate tools for pain assessment[114, 115].

A person with dementia may experience considerable anxiety or fear around pain. An awareness of cultural and language differences may assist in managing pain in those with dementia from Aboriginal and Torres Strait Islander[116] or CALD[117] backgrounds. Acceptable ways of expressing pain can vary across cultures and the influence of life experience, educational opportunities and socio-economic differences can be significant[118]. Also see *Introduction Module* for information on engaging interpreters.

Treatment for pain can be more effective when the underlying mechanism is targeted. Types of pain can be classified according to the following mechanisms[119]. Some pain will fit into more than one category:

- **Nociceptive** pain is the most common type of pain. It is due to stimulation of pain receptors which may arise from tissue inflammation, mechanical deformation, injury or disease such as osteoarthritis, fractures, musculoskeletal problems[120], decubitus ulcers and intra-abdominal conditions[117, 121].
- **Neuropathic** pain is due to damage to the peripheral and/or central nervous system and may be described as burning, itching, tingling, electric or shooting. Examples include diabetic neuropathy, central post-stroke pain, sciatica, phantom limb pain and neuralgia[121, 122].
- **Psychological or psychiatric factors** can play a major role in the onset, severity and maintenance of pain. Somatic complaints such as pain may be associated with depression. Diagnosis and subsequent treatment of the underlying psychopathology is indicated.
- **Pain related to mixed or unknown mechanisms** such as recurrent headaches or widespread pain can be unpredictable and more difficult to manage. A combination of approaches may be required.

Assessment of pain

Potential interventions for pain management include pharmacological, physical, psychological, complementary and alternative therapies. While the person with dementia should be given the opportunity to attempt self-report first[123], severity ratings will assist in determining the effectiveness of strategies trialled. Specific instruments are required for the assessment of pain in those with dementia, particularly where the capacity for self-report has been lost. New technologies under

development, as well as a computerised clinical system and pain assessment app to explore pain and vocalised expressions in people with dementia who are unable to self-report, have been tested in an Australian RACS[114, 124]. The following instruments report at least adequate reliability and validity, are readily available, easily administered, widely used in dementia care and have been translated into languages other than English:

- **The Pain Assessment in Advanced Dementia Scale (PAINAD)** is a clinically relevant tool for the assessment of pain in five areas: breathing; negative vocalisation; facial expression; body language and consolability[123, 125-127].
- **Pain Assessment Checklist for seniors with Limited Ability to Communicate (PACSLAC)** is appropriate for use in severe dementia. A familiar carer records their observations of behaviours associated with pain on four subscales: facial expressions, activity/body movements, social/personality/mood and physiological indicators[125, 128, 129].
- **The Abbey Pain Scale (ABS)** is based on observation, knowledge of the person's typical functional abilities and medical history. The ABS was developed for the assessment of pain in those who are unable to self-report. Six domains are included: vocalisation and facial expressions as well as body language, behaviour, physiological and physical changes[125, 130].

For further information on addressing pain refer to:

National Ageing Research Institute: Melbourne and Australian Pain Society. *Pain Management Guide: Toolkit for Aged Care*, 2nd Edition 2021[117] (accessed October 2023).

The Australian Pain Society website includes resources for older people with pain and for those with special needs (accessed October 2023).

The potential impact of trauma on BPSD

Many people with dementia have experienced trauma, grief and loss in their life. Trauma can occur as a single incident, or a series of incidents that a person finds emotionally disturbing, physically harmful or life-threatening[131]. Trauma can be particularly relevant in those with dementia from Aboriginal and Torres Strait Islander communities[132], migrants and refugees escaping violence and other minority groups subject to discrimination. Knowing how trauma affects people can assist in understanding their needs and potential causes of BPSD. Symptoms of trauma, dementia and BPSD can overlap, thereby confounding diagnosis. These include:

- memory and thinking problems
- difficulty communicating
- anxiety, anger and irritability
- stressed by changes in routine or having to make decisions
- depression, tearfulness, withdrawal
- emotional outbursts, aggression
- poor sleep and/or appetite[133].

Where trauma responses are misidentified as BPSD, potentially major consequences to the person's health and well-being can result[134]. Aspects of past traumatic experiences can also present for the first time during a delirium[135, 136]. The impact of past trauma may be lifelong and influence the person's presentation and experiences in health care settings[137]. More recent changes, associated with ageing, can also cause a sense of loss which can be traumatising for people with dementia. These can include the loss of independence, dignity, health, sense of purpose, loved ones, friends, home and/or pets[138].

The stigma associated with dementia may also contribute to the person's trauma. Further, trauma survivors can be reluctant to access aged care services due to past experiences which prompt distrust and fear of institutional care[139]. A person with dementia may have difficulty

expressing their feelings or explaining how historical or recent trauma is currently affecting them. Subsequent distress can present as BPSD[140, 141].

Trauma-informed care (TIC) incorporates attempting to: understand and acknowledge the impacts of past trauma, avoid triggers and provide a sense of choice and control in care[142]. TIC includes aspects of person-centred care (PCC) [143] but aims to go beyond PCC to recognise where trauma can change the experience of receiving care for some people with dementia[144]. TIC helps clinicians to support the person to feel as safe as possible in care. The goal is to address their needs in a way that avoids re-traumatising them and/or prompting BPSD[145].

For further information on trauma-informed care refer to:

Phoenix Australia. *Trauma and Aged Care Support and Information Hub* 2020[146] (accessed October 2023).

Australian Indigenous *HealthInfoNet* Edith Cowan University 2021[147] (accessed October 2023).

Inner West Sector Support and Development & the Ethnic Communities Council of NSW. *An introduction to trauma-informed care* (in multiple languages) 2021[131] (accessed October 2023).

Measuring BPSD

A description of BPSD should include the context in which the changed behaviours and/or psychological symptoms occur as well as the frequency, intensity, duration and impact of the changes. *Appendix 4* provides suggested questions to facilitate comprehensive assessment. Given their heterogeneous nature, it has been suggested that clusters of BPSD symptoms provide a framework for assessment and treatment[11, 21, 148, 149]. The following tools are widely used for the assessment of general and individual BPSD. Guidelines and scoring information enable the delivery of assessment results and ease of use. These and many other instruments for measuring individual BPSD have been translated into languages other than English.

Neuropsychiatric Inventory (NPI)

The Neuropsychiatric Inventory is used to assess psychopathology in the person with dementia and helps distinguish between the different causes of dementia. It is based on informant report for the preceding four weeks and is appropriate for patients in acute, community and residential care settings[150]. The NPI assesses the severity, frequency and level of carer stress associated with each symptom via individual subscales. The NPI-Clinician rating scale (NPI-C) [151] has expanded on many of the original items and enables care staff to act as the informant, providing an alternative to obtaining information from an informal carer. Other versions of the NPI include the NPI-nursing home (NPI-NH) [152] and a briefer version which can be completed by family carers, NPI-Questionnaire (NPI-Q) [153].

The reliability and validity of the NPI, overall is well established[154, 155] however, individual NPI symptom domains can be more clinically relevant than the total NPI score[156]. Interventions may be effective for managing one, or more, neuropsychiatric syndromes. Evaluating the effect of an intervention on individual symptoms or a cluster of symptoms is more likely to give an accurate depiction of efficacy in addressing neuropsychiatric syndromes. For more information and access to the NPI refer to the Dementia Outcomes Measurement Suite (DOMS) [92, 157].

Behavioural Pathology in Alzheimer's Disease (BEHAVE-AD)

The Behavioural Pathology in Alzheimer's Disease scale[158] measures global BPSD in people with AD through informant interview, based on the preceding two weeks. The BEHAVE-AD includes 25 items grouped into 7 major categories. It is appropriate for patients in acute, community and residential care settings. The instrument has well established psychometric properties and it is recommended for the measurement of global BPSD in clinical and research settings[157, 159]. The Empirical Behavioral Pathology in Alzheimer's Disease (E-BEHAVE-AD) [160] was later developed as a 12-item clinician-rated instrument.

While the BEHAVE-AD was developed for use in people with AD, it has been used in vascular dementia (VaD), DLB and FTD[161-164]. Caution is necessary when measuring BPSD in FTD as the BEHAVE-AD may not adequately measure symptoms which occur less frequently in AD such as apathy, disinhibition and emotional inappropriateness[157]. For more information and access to the BEHAVE-AD refer to the Dementia Outcomes Measurement Suite (DOMS)[92].

Culturally appropriate dementia assessment tools for Aboriginal and Torres Strait Islander peoples

A lack of timely diagnosis of dementia, and consequently BPSD, in Aboriginal and Torres Strait Islander communities impacts on presentation, management and the provision of timely and appropriate support[165]. Mainstream dementia assessment tools may be inappropriate for Aboriginal and Torres Strait Islander peoples. For example, they often include concepts of functioning related to career and employment, concepts of independence framed as positive rather than valuing the level of family dependence, or examples that may have little meaning, especially in a remote context[157]. It is important that cultural, emotional and spiritual aspects are accounted for in screening instruments.

The Kimberley Indigenous Cognitive Assessment (KICA-Cog)[166] is a validated dementia assessment tool for older Aboriginal and Torres Strait Islander peoples. The KICA-Cog has 16 questions that test memory, comprehension, language abilities and limited executive functions and thus the KICA-Cog predominantly assesses memory and language skills. The KICA-Cog has been tested with rural and remote Aboriginal peoples and the urban and regional modification in those living in urban and regional settings. It has been further adapted for use with Torres Strait Islander peoples. The scales are reportedly culturally acceptable with good psychometric properties[23, 167-169]. In efforts to support timely dementia diagnosis in remote communities, the KICA-screen has been tested, and reported as acceptable, via telehealth[170]. The KICA-Cog with additional tools and resources to aid administration and exclude other possible causes of cognitive decline are available at

https://www.iawr.com.au/kica [171](accessed October 2023).

Culturally appropriate dementia and BPSD assessment tools for CALD groups

It is important to ensure culturally appropriate assessments are undertaken with people with possible cognitive impairment from CALD backgrounds. For older CALD people, particularly those for whom English is not their preferred language, screening tools must be culturally equivalent, not merely translated from one language to another[172, 173] and have good psychometric properties[174, 175]. It is important that clinicians are aware of the limitations of some existing screening and assessment tools for cognitive impairment in their use with patients from non-English speaking and culturally diverse backgrounds[176]. The Australian Dementia Outcomes Measurement Suite (DOMS) project found that the Rowland Universal Dementia Assessment Scale (RUDAS)[177], the Modified Mini Mental Exam (3MS)[178] and the General Practitioner Assessment of Cognition (GPCOG)[179] were suitable and valid tools in most health care settings for people from CALD backgrounds[157].

The Rowland Universal Dementia Assessment Scale (RUDAS)[177] is a cognitive screening tool that was designed to reduce the effects of cultural and/or linguistic diversity on the assessment of baseline cognition in people from CALD backgrounds. The 6-item scale was developed and validated in CALD populations and was found not to be affected by gender, years of education or language. It can be directly and easily translated into a number of languages without having to change the structure or format of any item[177]. Access the RUDAS here.

Results

Intervention studies from a literature search (2012-21) were classified as *psychosocial and environmental* or *biological and pharmacological*. We found 113 psychosocial/environmental and 69 biological/pharmacological studies that reported outcomes for general BPSD and met our inclusion criteria. Of the psychosocial/environmental interventions, 75 studies were rated as moderate quality and 38 were rated as strong. Positive outcomes were reported for 44

studies; the *models of care* category included the greatest number with 19. Of the biological/ pharmacological interventions, 43 studies were rated as moderate quality and 26 were rated as strong. Positive outcomes were reported for 36 studies and the *cholinesterase inhibitors/ memantine* category included the greatest number with 33. See *Appendix 5: Methodology*, for further details on the method employed in conducting the literature search and *Appendices 2* and *3* for tables of summarised interventions.

Addressing BPSD

Psychosocial and environmental interventions

Clinicians should attempt to prevent and/or minimise BPSD in the first instance through a supportive and proactive approach[41, 180]. Psychosocial and environmental intervention trials from this literature review are grouped according to the following categories:

- *Animal interventions* (or animal-assisted therapies) involve interaction between the person with dementia and trained animals, plush animal toys or robotic animals including *Paro*.
- *Cognitive rehabilitation/stimulation interventions* include cognitive behavioural therapy, cognitive stimulation, mindfulness, creative storytelling, psychostimulation and problem adaptation therapy and cognitive training programs.
- *Education/training* includes psychoeducation programs for people with dementia and/or their carers and staff training programs which target BPSD.
- *Exercise interventions* entail some form of physical activity such as walking, chair-based exercises and/or movement which targets balance, mobility and/or strength.
- *Models of care* refer to specific care protocols or services, including respite care interventions, dementia care mapping, dementia-specific units, interventions incorporating *Montessori* principles and environmental modifications.
- *Multicomponent interventions* are intervention trials that compare a combination of interventions from many or all of these categories, in a variety of care settings.
- *Music interventions* engage the use of sounds, melodies and/or rhythmic movement through live music, singing, playing musical instruments or listening to music.
- *Reminiscence-based interventions* involve using life histories and past experiences to promote positive memories including memory apps for mobile phones, ipads and computers and reminiscence sessions incorporating many different types of props.
- *Sensory interventions* provide people with dementia with sensory stimulation including aromatherapy, massage and multisensory stimulation (such as *Snoezelen*).
- *Therapeutic recreation* refers to a range of leisure activities including cooking activities, humour therapy, gardening, doll therapy, art, games or social interaction.
- *Touch therapies* refer to a range of activities including acupressure, acupuncture and massage, with some incorporating aromatherapy.

See *Appendix 2* for table of psychosocial and environmental interventions.

Biological and pharmacological interventions

Biological and pharmacological interventions from the literature search include pharmacological treatments, light therapy and transcranial magnetic stimulation (TMS) therapies. See *Appendix 3* for table of biological and pharmacological interventions.

Table 2.2 Generic and trade names for medications relevant to BPSD

Generic name	Trade names
Cholinesterase inhibitors (ChEIs) and memantine	
Donepezil	Aricept, Arazil, Aridon
Galantamine	Galantyl, Gamine, Reminyl
Memantine	Ebixa, Memanxa
Rivastigmine	Exelon
Typical antipsychotic (neuroleptic)	
Haloperidol	Serenace
Atypical antipsychotics (neuroleptics)	
Amisulpride	Solian, Sulprix, Amipride
Aripiprazole	Abilify, Abyraz, Tevaripiprazole
Clozapine	Clopine, Clozaril
Olanzapine	Zyprexa, Lanzek, Ozin, Zylap, Zypine, Pryzex
Quetiapine	Seroquel, Syquet, Quetia, Tevatiapine, Kaptan
Risperidone	Ozidal, Resdone, Rispa, Risperdal, Rispernia, Rixadone
Ziprasidone	Ziprox, Zeldox
Antidepressants	
Agomelatine	Valdoxan, Domion, Nortim, Ardix Agomelatine
Citalopram	Cipramil, Celapram, Celica, Ciazil, Citalobell, Talam
Clomipramine	Anafranil, Placil
Duloxetine	Cymbalta, Duloxecor, Dytrex, Tixol
Escitalopram	Cipralex, Lexapro, Esiprama
Fluoxetine	Prozac, Auscap, Lovan, Zactin, Fluotex
Milnacipran	Joncia, Acmil, Milnace
Mirtazapine	Avanza, Axit, Mirtazon, Mirtanza
Nortriptyline	Allegron, Nortritabs
Paroxetine	Aropax, Extine, Paxtine, Paroxo, Paxtinea
Sertraline	Zoloft, Eleva, Sertra, Setrona
Trazodone	Oleptro
Venlafaxine	Efexor, Elaxine, Enlafax
Hormone	
Melatonin	Circadin
Psychostimulant	
Methylphenidate	Artige, Attenta, Concerta, Ritalin
Anxiolytics	
Diazepam	Valium, Ducene, Antenex, Propam
Lorazepam	Ativan
Oxazepam	Alepam, Murelax, Serepax
Cannabinoids	
Dronabinol	Marinol, Syndros
Nabilone	Cesamet

Adverse effects of psychotropic medications

While an individualised medication risk-benefit assessment should always be undertaken, psychotropic medications are not the preferred treatment for changed behaviours or psychological symptoms in people living with dementia[181]. Their use should be reserved for severe symptoms that are unresponsive to nonpharmacological strategies[182, 183]. Where any pharmacological interventions are indicated these should be prescribed in combination with appropriate psychosocial interventions[183-185]. Before a psychotropic medication is prescribed, a comprehensive discussion must be undertaken with the person with dementia, their support person and/or substitute decision-maker about potential individual harms and benefits. This discussion should be documented in the person's medical record, behaviour support plan and progress notes, as applicable. Health literacy should be considered, and an interpreter engaged where indicated[183]. See *Working with an interpreter during assessment* section in *Module 1* for information on using interpreters. Further, informed consent from the person with dementia and/or their substitute decision-maker as well as approval by the person's attending physician or specialist must be obtained. A protocol should be put in place for monitoring, review, reduction and/or discontinuation when indicated.

No antipsychotic medication is both safe and effective for the treatment of BPSD in dementia[186, 187]. Antipsychotic use in dementia has been associated with higher risk of functional and cognitive decline as well as greater risk of somnolence, extrapyramidal symptoms, abnormal gait, oedema, urinary tract infections, incontinence, falls, cerebrovascular adverse events and mortality[41, 183, 188-190]. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[183, 191]. For example, where the person presents with psychotic symptoms and agitation, treating the psychotic symptoms may also reduce agitation. See *Table 2.3 Side effects of antipsychotics/neuroleptics* for more information.

Research provides some support for the reduction and/or discontinuation of antipsychotic treatment when indicated. Ballard and colleagues[192] compared neuropsychiatric symptoms in two groups of people with AD who had been previously prescribed antipsychotics for BPSD for at least three months. The treatment group continued medication and the control group commenced placebo. No significant difference in NPI scores was found between groups at 6 months but at one year there was some evidence suggesting those with severe neuropsychiatric symptoms at baseline (NPI≥15) benefitted from continuing treatment. Those who continued antipsychotics had more rapid decline and greater mortality. More recently, a longitudinal study across 23 RACS evaluated the sustained reduction of antipsychotic use for BPSD, supported by education for health care professionals[193]. Withdrawal of antipsychotics was not accompanied by drug substitution or a significant increase in the use of prn antipsychotic or benzodiazepine medications. No increase in NPI BPSD scores or adverse outcomes occurred. In most situations antipsychotic use should be short-term (up to 12 weeks) for severe neuropsychiatric symptoms only, to minimise potentially serious adverse effects[194].

For further information on psychotropic medications refer to:

The Guideline Development Group. *Clinical Practice Guidelines for the Appropriate Use of Psychotropic Medications in People Living with Dementia and in Residential Aged Care* 2022[183] (accessed October 2023).

Australian Government Aged Care Quality and Safety Commission and the Wicking Dementia Research and Education Centre. *Psychotropic medications used in Australia: Information for aged care* 2020[195] (accessed October 2023).

Dementia Training Australia. *Appropriate use of antipsychotics when prescribed as a restrictive practice* 2023[196] (accessed October 2023).

Table 2.3: Side effects of antipsychotics/neuroleptics adapted from Moretti et al 2006 [197]

Drug	Extra-pyramidal effects	Prolactin	Anti-cholinergic effects	Seizure risk	Ortho-stasis	Weight gain	Sedation	Haemato-logical effects	Elevated blood sugar	Elevated cholesterol
Clozapine	0	0	+++	+++	+++	+++	+++	+++	+	+
Risperidone	0/++	0/++	+	+	++	+	+	0	0	0
Olanzapine	0/+	0	++	+	++	++	++	0	+	+
Quetiapine	0/+	0	+	+	+	+	++	0	+	+
Ziprasidone	0/+	0/+	+	+	+	?	+	0	+	+
Aripiprazole	0/+	0	+	+	+	+	+	0	+	+
Amisulpride	0/+	0	+	0	+	+	+	0	0	0
Haloperidol	+++	+	0/+	+	+	+	+/++	+	+	0

Pimavanserin and Brexpiprazole are omitted from this table. Pimavanserin was voted by FDA committee (17/6/2022) as not demonstrating "substantial evidence for the effectiveness … for the treatment of hallucinations and delusions associated with Alzheimer's disease". Pimavanserin can cause prolongation of QT interval which can be associated with cardiac arrhythmias and, as with other antipsychotics, it carries a black box warning of increased risk of death (Schneider 2022; https://doi.org/10.1176/appi.ajp.20220519). Brexpiprazole carried similar warnings about increased risk of death (FDA Briefing Document, NDA 205422/S-009, 14th April 2023)

Key

0 no effect
\+ present
++ present and major side effect
+++ present and very important side effect

Restrictive practices and BPSD

A restrictive practice is any practice or intervention that restricts a person's rights or freedom of movement for the primary purpose of influencing their behaviour. People with dementia are reported as being more likely to be subjected to the inappropriate use of restrictive practices[198-200]. The Federal Register of Legislation defines five types of restrictive practices under Part 4A, Division 2, 15E of the Quality of Care Principles 2014 of the updated Aged Care Act 1997:

- Chemical restraint is a practice or intervention that is, or that involves, the use of medication or a chemical substance for the primary purpose of influencing a care recipient's behaviour, but does not include the use of medication prescribed for:
  (a) the treatment of, or to enable treatment of, the care recipient for: (i) a diagnosed mental disorder; or (ii) a physical illness; or (iii) a physical condition; or
  (b) end of life care for the care recipient.
- Environmental restraint is a practice or intervention that restricts, or that involves restricting, a care recipient's free access to all parts of the care recipient's environment (including items and activities) for the primary purpose of influencing the care recipient's behaviour.
- Mechanical restraint is a practice or intervention that is, or that involves, the use of a device to prevent, restrict or subdue a care recipient's movement for the primary purpose of influencing the care recipient's behaviour, but does not include the use of a device for therapeutic or non-behavioural purposes in relation to the care recipient.
- Physical restraint is a practice or intervention that: is or involves the use of physical force to prevent, restrict or subdue movement of a care recipient's body, or part of a care recipient's body, for the primary purpose of influencing the care recipient's behaviour; but does not include the use of a hands-on technique in a reflexive way to guide or redirect the care recipient away from potential harm or injury if it is consistent with what could reasonably be considered to be the exercise of care towards the care recipient.
- Seclusion is a practice or intervention that is, or that involves, the solitary confinement of a care recipient in a room or a physical space at any hour of the day or night where:
  (a) voluntary exit is prevented or not facilitated; or
  (b) it is implied that voluntary exit is not permitted; for the primary purpose of influencing the care recipient's behaviour.

Sourced from the Federal Register of Legislation at 8 October 2023[201]. For the latest information on Australian Government law go to https://www.legislation.gov.au.

Restrictive practices have been associated with serious adverse physical and psychological outcomes, including the death of residents in RACS[202]. The person's efforts to affect their release can increase their risk of falls and subsequent risk of serious injury[203, 204]. Further consequences include other BPSD such as vocal disruption, depression and agitation as well as decreased mobility, muscle weakening, incontinence, pressure ulcers and significant psychological effects[205-207]. Circumstances may occur where a restrictive practice is considered necessary as a last resort measure to protect the health, safety, and dignity of the person with dementia or others. By implementing strategies to prevent or reduce BPSD, including through comprehensive risk assessment, the inappropriate use of restrictive practices can be minimised[181].

Restrictive practices must be used in the least restrictive manner and cannot be used in residential aged care unless there is a Behaviour Support Plan in place, except in an emergency. The Quality of Care Principles 2014, Part 4A, Division 5, 15HA sets out service provider's responsibilities relating to behaviour support plans[145, 208]. An emergency is defined as "a serious or dangerous situation that is unanticipated or unforeseen and that requires immediate action"[209]. The person's substitute decision-maker must be informed and details of the incident documented afterwards. See Dementia Support Australia for information on developing a Behaviour Support Plan - restrictive practices[210].

Informed consent for the use of restrictive practices in residential aged care must be provided by the person or their restrictive practices substitute decision-maker (RPSDM), where the person with dementia lacks capacity. A RPSDM is a person or body that can give informed consent to the restrictive practice, including the prescription of medication in the case of chemical restraint. Consent must follow the laws of the state or territory in which the person is receiving aged care[211]. Strategies that provide alternatives to the use of restrictive practices should always be considered first. If after careful consideration of all options, the use of restrictive practices is deemed to be in the best interest of the person with dementia, safety measures must be observed. While the decision to use restraint is made to ensure the safety of the person with dementia, the expected benefits must be weighed against the potential harm of any form of restraint prior to implementation.

The requirements set out in Part 4A of the Quality of Care Principles 2014[201] in relation to the use of restrictive practices does not apply to aged care services delivered in the home or community setting. However, providers of services delivered in home or community settings must have an appropriate care and services plan in place that satisfies the requirements set out in the Aged Care Quality Standards for each care recipient. This includes regular review of the plan for effectiveness and in response to incidents that impact the needs, goals or preferences of the person with dementia.

Under Part 4B, Division 4, section 15NB(2A) of the Quality of Care Principles 2014[212], in home or community service providers must meet certain requirements for the use of a restrictive practice. These requirements include (but are not limited to) documenting the changed behaviours and circumstances when the restrictive practice may be used in the care recipient's care and services plan. If the use of the restrictive practice does not meet the requirements as set out in section 15NB(2A), the provider must submit a reportable incident notification, under the Serious Incident Response Scheme to the Aged Care Quality and Safety Commission.

The use of restrictive practices by family or other carers may be a form of elder abuse[145, 213]. See World Health Organization, Abuse of older people[214] for more information. Wherever restrictive practices are used, care providers are responsible for the regular monitoring, reporting and documenting of any signs of distress or harm, adverse events, changes to the person's wellbeing and/or functioning as well as the effectiveness of the restrictive practices[145].

A Cochrane review reported that interventions aimed at reducing the use of physical restraint at an organisational level through changing policy and practice in RACS are probably effective at reducing the number of residents restrained[207]. A background paper prepared for the Royal Commission into Aged Care Quality and Safety[215] identified six main barriers to the use of non-pharmacological interventions to reduce the need for restrictive practices including:

- perceptions about safety, fear of resident injury and legal concerns
- staff and resource constraints in RACS, and organisational culture
- lack of knowledge about effects of restrictive practices and alternative practices
- beliefs and expectations of staff, family and residents
- inadequate review, and
- communication barriers.

People with dementia who present with BPSD often have complex care needs. Providing quality care with a focus on psychosocial and nondrug therapeutic approaches to addressing BPSD requires a well-trained and skilled workforce[216] of adequate numbers, with access to specialised supports. This would help to limit restrictive practices to a last resort option in addressing BPSD[205].

Table 2.4 Strategies for alternatives to restrictive practices adapted from Department of Health and Ageing 2012[217]

Physical Environmental strategies	*Personal areas*: • bed height to suit individual needs, ensure brakes are engaged • appropriate mobility aids close at hand • soft floor mat by the bed to minimise injury from falls • familiar objects from the person's home (photos, furniture, etc) • seating to meet the needs of individual residents • appropriate (silent) alarm systems to alert staff to situations of risk such as a resident who has exited a safe area *Indoor areas:* • minimise disruptive changes to the care environment • reduce clutter and glare • install non-slip or carpet flooring in frequented areas • appropriate signage and visual reminders to aid orientation • provide safe areas for residents to walk/wander • provide quiet areas and where possible, reduce overstimulation due to environmental noise and bright lighting *Outdoor areas:* • increase ease of access to safe and protected outdoor areas
Social and emotional environmental strategies	• encourage visitors (staggered if indicated) and promote appropriate staff/ resident interaction • engage familiar staff • relaxation activities such as therapeutic touch and massage • reality orientation • sensory aids and appropriate stimulation • decrease sensory overload
Psychosocial strategies	*Develop and implement individualised psychosocial strategies*: • rehabilitation and/or exercise • continence program • physical, occupational and recreational therapies • night-time activities for nocturnal restlessness • individual and small group social activities • activities for promoting success through use of overlearned skills (e.g. gardening, folding laundry) • facilitate safe walking/wandering • offer a change of seating arrangements at regular intervals with their consent, for residents who are not independently mobile • falls prevention program
Care approach	• person-centred care (i.e. knowing the person as an individual) • increased supervision and observation by all categories of staff • regular evaluation and monitoring of conditions that may alter behaviour • individualised and structured routines e.g. toileting, naps • check 'at risk' residents regularly • improved communication strategies
Physiological strategies	• comprehensive medical examination and medication review • treat infections, constipation, etc • pain management • consider alternatives to sedation (warm milk, soothing music, etc)

For further information on restrictive practices refer to:

Australian Government Department of Health and Aged Care. *Restrictive practices in aged care – a last resort* 2023[211] (accessed October 2023).

Aged Care Quality and Safety Commission. *Overview of restrictive practices* 2021[209] (accessed October 2023).

Australian Government Federal register of legislation Quality of Care Amendment. *Minimising the Use of Restraints* Principles 2019 (accessed October 2023).

Dementia Support Australia. *The When and What of Behaviour Support Plans* 2022[210] (accessed October 2023).

Dementia Training Australia. *Dementia and Behaviour Support Plans* [218] (accessed October 2023).

Dementia Australia. *Restrictive practices: A submission to the Royal Commission into Violence, Abuse, Neglect and Exploitation of People with Disability* 2020[205] (accessed October 2023).

Limitations

Limitations in the methodology of the studies included in this review inevitably affect the validity of reported outcomes. The best levels of evidence are from randomised, double-blind, placebo-controlled studies, but many psychosocial and environmental studies reviewed for this Guide did not meet this gold standard. Further, the multiple components of psychosocial and environmental interventions often overlap making it difficult to determine which aspect of the intervention is the active ingredient. Psychosocial and environmental studies conducted within RACS also face the added complication of potential contamination across groups. It is virtually impossible to introduce an intervention, beyond the normal routine, which does not impact on all those in the immediate vicinity in some way. Visiting researchers (or others gathering observational data) will likely provide a point of interest amongst residents, their families and/or staff. This negates true control conditions for the purposes of comparison with the intervention condition.

Research into interventions targeting BPSD tends to be hampered by the difficulties of recruiting and retaining numbers to ensure sufficient power to conduct good quality trials. Ethical issues also arise when recruiting and obtaining consent from participants with dementia. Maintaining compliance with interventions and study design in the real world often requires much supervision, support and encouragement by carers. When obvious individual improvements are not evident, carers' motivation and commitment may diminish. Both, formal and informal carers are classically overloaded in their roles thereby increasing the risk of withdrawal from studies. Participant samples often included mixed types and severities of dementia. Primary outcome measures used to collect BPSD data were not always validated, follow-up assessment to determine sustainability of effects was rarely conducted and intention-to-treat analyses were frequently not reported. Further, effect sizes could not be calculated for many studies when means and/or standard deviations were not provided.

Where studies report similar interventions, differing care environments and procedures led to difficulty in determining consistency of delivery. Individual BPSD were typically not the primary outcome in the majority of the included studies. Difficulties arise when studies treat BPSD as a homogenous group and individual symptoms are not reported separately. Equally problems occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. Consistency in gathering and reporting outcomes would better facilitate useful comparisons between studies and aid in developing effective treatment strategies.

Conclusions/Principles of care

While limitations are evident, it is encouraging to see a substantial increase in the number, quality and complexity of studies investigating the efficacy of interventions to address BPSD,

particularly psychosocial and environmental approaches[219], over the past decade. Overall, support for the effectiveness of pharmacological and biological treatment of BPSD is limited and the risk of adverse effects is always a concern. However, ongoing trials of potential treatments provide hope with 19% of current phase 3 trials in people with AD targeting neuropsychiatric symptoms[220]. While an increasing number of psychosocial interventions have been reported as effective[219], efforts to improve the quality of psychosocial/environmental intervention studies continue.

Research findings suggest that psychosocial/environmental and pharmacological/biological interventions have a limited effect when applied as a generic treatment for BPSD, with more studies reporting negative outcomes than positive. The limited benefits of generic interventions can largely be attributed to the diverse aetiology of BPSD, implying that an intervention may be effective in one set of circumstances or for one behaviour or psychological symptom but not another. Individualised interventions that are tailored to specific situations are recommended. To uncover factors that may be contributing to BPSD a thorough process of information gathering and assessment of the person with dementia, the presenting BPSD, and the interpersonal and physical environment is essential. Care partners, family members and care staff often hold valuable knowledge and information about the person's history, personality and preferences. This information is important in developing effective intervention strategies. See individual modules following for further information on interventions targeting specific BPSD.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

3. Aggression

MODULE 3: Aggression

Key messages

- Aggression in dementia is characterised by physically and/or verbally threatening behaviours directed at people, objects or self.
- Aggression can arise from underlying depression, psychotic symptoms and/or unmet needs such as pain and constipation.
- The prevalence of verbal and/or physical aggression reportedly ranges from 20% to 30% of people with dementia living in the community and from 6% to 95% of those in residential care.
- Harm to the person with dementia or others and the inappropriate use of restrictive practices can be serious consequences of aggression.
- The crucial task for the clinician is to attempt to understand what is underlying the aggression for the individual with dementia and to address the cause.
- Individualised psychosocial interventions are recommended as the first line approach although the scientific evidence is limited.
- Intervention trials sometimes report combined agitation/aggression outcomes, which limits the generalisability of findings for aggression.
- Pain management should be considered and some support for the benefit of analgesic medication was indicated.
- Expert consensus guidelines recommend short term use of atypical antipsychotics for physical aggression where necessary for safety.
- Some pharmacological evidence was reported for dextromethorphan-quinidine.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and addressed, reversible causes of the BPSD

    (See *Module 1* for further details)

Aggression summary

What is aggression and what does it look like in dementia?

Aggression in dementia is characterised by physically and/or verbally threatening behaviours directed at people, objects or self. It is often quantified by specific acts which can include:

- verbal insults, shouting, screaming
- obscene language
- hitting, punching, kicking
- pushing, throwing objects
- sexual aggression

Causes of aggression

- Aggression can be a purposive and overt response to a violation of personal space or a perceived threat.
- It often occurs during personal care tasks involving close carer-/staff-resident contact.
- Aggression may be a form of communication and/or a manifestation of unmet needs such as poorly managed pain, constipation, illness, infection and loneliness.
- Underlying depression, psychotic symptoms and/or environmental stressors can also give rise to aggression in people living with dementia.

Differential diagnosis

Aggression can present independently or with agitation. It is also strongly associated with depression and psychosis. Aggression should be differentiated from delirium.

Measuring aggression

Some rating scales have been developed for assessing aggression and other global BPSD scales include items/subscales which measure aggression. The following scales are widely used:

- The Rating scale for Aggressive behaviour in the Elderly (**RAGE**)
- The Overt Aggression Scale (**OAS**)
- The physically aggressive subscale of the Cohen-Mansfield Agitation Inventory (**CMAI**)
- The agitation/aggression subscale of the Neuropsychiatric Inventory (**NPI**) and the aggression subscale of the **NPI-Clinician**

Prevalence of aggression

The prevalence of aggression reportedly ranges from 20% to 30% of people with dementia living in the community and from 6% to 95% of those living in residential aged care homes. The frequency of aggression tends to increase as dementia progresses and functioning declines, until the later stages when it typically declines. Prevalence also varies with dementia types.

Effects of aggression

Aggression is associated with considerable carer burden and stress, reduced quality of life and earlier admission to residential aged care. Although not common, harm to the person with dementia or others can be a serious consequence, as can the inappropriate use of restrictive practices.

Addressing aggression

The crucial task for the clinician is to attempt to understand what is underlying the aggression for the individual with dementia. Interventions targeting the cause will likely assist in avoiding or reducing aggression.

Psychosocial and environmental interventions

- Psychosocial and environmental intervention studies were primarily conducted in residential settings.
- Limited evidence of a decrease in number of episodes of physical aggression was demonstrated for environmental modifications.
- No benefit was found for humour therapy, aromatherapy or care-staff education.
- Individualised, person-centred care based on psychosocial interventions are recommended as a first line approach unless urgent action is required for safety.
- The lack of scientific evidence for psychosocial interventions should not prevent clinicians considering non-drug interventions on an individual basis.

Biological and pharmacological interventions

- Although the adverse effects of pharmacological interventions raise concerns, particularly antipsychotics, situations can arise which place the person with dementia and/or others around them at risk, requiring an urgent response.
- Where physical aggression presents a safety risk, expert consensus guidelines recommend short-term use of atypical antipsychotics although evidence to support their use is limited. Risperidone is the only antipsychotic approved by the Australian Government Pharmaceutical Benefits Scheme (PBS) for this use.
- Some evidence of effect in reducing aggression was reported for analgesic medications and dextromethorphan-quinidine.
- No benefit was found for cholinesterase inhibitors or antidepressants.

Limitations

Limited intervention studies were reported for aggression in dementia and aggression is often not the primary outcome of the included intervention studies. Some intervention trials report combined agitation/aggression outcomes only, which limits the generalisability of findings. Few studies examined the long-term sustainability of benefits after interventions ceased.

Conclusions/Principles of care

- Aggression can have serious consequences for the person living with dementia and others around them.
- The anxiety and fear generated in others by the unpredictability of aggression tends to isolate the person with dementia.
- Individualised psychosocial interventions are recommended as the first line intervention, although the evidence is limited.
- Effective pain management should always be considered.
- Where necessary for safety, expert consensus guidelines recommend the short-term use of atypical antipsychotics for aggression, that is not due to underlying anxiety or depression.

What is aggression and what does it look like in dementia?

Aggression associated with dementia can be extremely challenging and disruptive. The presentation of aggression is not always consistent, but it is characterised by physically and/or verbally threatening behaviours directed at people, objects or self. Aggression is generally perceived as a threat to the safety of those with dementia and the care environment, which includes family carers and/or care staff and other residents[1-3]. Aggression includes:

- verbal insults
- shouting, screaming
- obscene language
- hitting, punching, kicking
- pushing, throwing objects
- sexual aggression[4]. Also see *Module 9 Disinhibition.*

Expert opinion differs regarding whether aggression is a unique syndrome in dementia or a subset of agitation[5, 6]. Verbal and physical aggression are included as part of the IPA criteria definition of agitation, items for aggression are included in several agitation measurement tools as are aggression subscales. The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11) includes Agitation or aggression in dementia under a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" under Neurocognitive disorders, Dementia. Aggression is described as "clinically significant excessive psychomotor activity accompanied by increased tension and hostile or violent behaviour"[7].

Because dementia is a significant and growing health and aged care issue, the Australian Institute of Health and Welfare reports statistics, including those relevant to BPSD, to provide a comprehensive picture of dementia in Australia[5, 8]. Information on physical aggression and verbal aggression are currently recorded as separate and unique behaviours rather than the previously combined agitation/aggression category[8].

PRESENTATION

Bob is a 78 year old man who has lived in residential care for 6 months. He was widowed some years ago but he has a caring family who visit regularly. Prior to moving into the aged care home Bob lived alone. He frequently expresses his frustration at sharing 'his home' with so many other people. At times he has become verbally aggressive with staff and other residents. Some staff are aware of Bob's frustrations and the early indicators of his distress. These staff members are usually able to diffuse these situations before they escalate.

A resident who has recently been admitted to the aged care home is often very talkative. On one occasion when this resident was talking continuously, Bob slapped his hand over their mouth and said, "Would you just shut up?". In response the resident hit out at Bob who then grabbed them by the shirt and pushed them into the closest chair. At this point the other resident was yelling and others nearby were visibly distressed. Bob stormed off, cursing loudly.

Causes of aggression

Aggression can be a purposive and overt response to a violation of personal space or a perceived threat[9, 10]. Not unexpectedly, it often occurs during personal care tasks involving close contact[1, 4, 11] and during interactions with other residents in residential care settings[9, 12]. The neurobiological underpinnings of aggression in dementia are not well understood, but reduced serotonergic activity[3, 10], reduced cholinergic transmission[13], as well as frontal lobe/executive dysfunction[14] have been implicated. Aggression can occur because of underlying pain, poor relationship quality between the person and carer, dysphoria, depression or psychotic symptoms[15-17].

Aggression may be a manifestation of unmet physiological and/or psychological needs. Within the Need Driven Behaviour (NDB) model,

aggression can be a form of communication as the person is less able to effectively articulate their needs. People living with dementia and their care partners have said that aggression may be a manifestation of confusion, frustration, insecurity, self-loathing and fear[18]. Aggression, and the negative impact of aggressive interactions on relationships with care staff and family carers, can be a source of sadness for people living with dementia[19]. People with lived experience of dementia have highlighted the importance of others trying to understand the underlying reasons for their changed behaviour. They have indicated that helpful responses include being treated respectfully and accepting the reality of the person living with dementia[20]. For example, being aware that resisting/lashing out could be a natural reaction, for anybody, if a stranger tried to remove their clothing and place them in a shower[20].

There is no single cause of aggression in dementia. Common precipitants include:

- pain or discomfort
- hunger and/or thirst
- dehydration
- boredom
- fear
- past and ongoing trauma
- environmental temperature
- frustration
- violation of social norms
- constipation
- medical illness
- infection
- depression
- loneliness
- perceived lack of respect
- problematic social relationships
- resident-to-resident tension
- sensory overstimulation.
- environmental stressors including staff and/or carer communication
- misinterpretation of intention or context due to vision and/or hearing impairment
- misinterpretation of intention or context due to cognitive impairment
- subjective/objective violation of personal space[15, 21, 22]

Differential diagnosis

The term aggression is often used interchangeably with agitation[5]. It can present independently or with agitation[2, 23] and the underlying mechanisms of aggression may be unique. Aggression is strongly associated with depression[14, 24] and psychosis[24, 25]. Aggression can also indicate a delirium[26, 27].

Measuring aggression

Scales specifically developed for assessing aggression, and global scales which include items which measure aggression, are available for clinical purposes:

- The Rating scale for Aggressive behaviour in the Elderly (**RAGE**) is a carer or care staff rated 21-item scale to quantify type, frequency and pattern of physical aggression, verbal aggression and antisocial behaviour over the preceding three to five days[28]. The reliability and validity of the RAGE is well established[29] and it is available in multiple languages.
- The Overt Aggression Scale (**OAS**) is a staff-rated scale which assesses the severity and duration of verbal and physical aggression against objects, self and others[30]. Modified versions have been used with older adults and people with dementia[31-33]. These include the OAS-M[34] for outpatients, the MOAS-1[35] for institutional settings and the MOAS-2[36] for drug studies. The psychometric properties of MOAS in dementia have not been thoroughly evaluated[37].
- The Aggressive Behavior Risk Assessment Tool for Long-Term Care (**ARBAT-L**) is a 6-item tool completed by aged care staff to assess risk of aggression in people in long-term care settings[38]. Items include history of physical aggression, cognitive impairment, anxiety and current physical aggression/threatening. The tool has satisfactory predictive sensitivity and specificity[39].

- The Aggressive Behavior Scale (**ABS**) is a 4-item scale completed by a trained observer or aged care staff to measure severity of aggressive behaviour based on verbal and physical abuse, socially inappropriate behaviour and resistance to care over a seven-day period[40]. The tool has good to excellent psychometric properties in older adults[37, 41].
- The Ryden Aggression Scale (**RAS**) is a 25-item staff or family carer-rated measure of physical, verbal, and sexual aggression over the past year[42]. Psychometric properties for the RAS are adequate to good[37, 41]. The adapted RAS-2[43] rates the presence of 26 aggressive behaviours over a 24-hour period.
- The physically aggressive subscale of the carer-rated Cohen-Mansfield Agitation Inventory (**CMAI**) includes physical and verbal behaviours assessed during the preceding fortnight[44].
- The Agitation/Aggression subscale of the Neuropsychiatric Inventory (**NPI)** is completed during an interview with the carer who rates the frequency and severity of the person with dementia's aggression as well as their own subsequent distress[45]. The reliability and validity of the NPI overall is well established[46].
- The NPI-Clinician (**NPI-C**) has revised the original NPI subscale, allocating aggression a discrete subscale with an expanded list of 14 items[47] including spitting, pushing, scratching, passive aggression, intrusive behaviours, destroying property, conflict with others, sexual aggression, dangerous activities and throwing food.
- The Physical and Verbal Aggression subscales of the staff-rated Disruptive Behavior Rating Scales (**DBRS**) assesses severity over one-week based on medical records and staff reports[48]. Strength of subscale psychometric properties varies [37].
- The Aggressiveness subscale of the staff-rated observational Pittsburgh Agitation Scale (**PAS**) assesses severity over a period of 1 to 8 hours[49]. The subscale has good to excellent psychometric properties in inpatient and residential care settings[37].
- The 3-item Aggressiveness subscale of the clinician-/staff-rated Behavioral Pathology in Alzheimer's Disease Rating Scale (**BEHAVE-AD**) assesses verbal and physical aggression[50].
- The 8-item Uncooperative or Aggressive Behavior subscale of the Nursing Home Behavior Problem Scale (**NHBPS**) measures frequency of resistance to care, physical aggression, and verbal aggression over the past three days[51]. The scale has adequate psychometric properties[37].

Prevalence of aggression

Aggression has been reported in approximately 20% to 30% of people with dementia living in the community[52, 53] and 6% to 95% in residential aged care[52, 54, 55]. Not all episodes of aggression are clinically significant[5, 56] but under-reporting can occur, particularly with family carers. Aggressive symptoms in dementia are more common in men[57]. In Australia, physical and verbal aggression are the second and third most common BPSD reported in Dementia Support Australia assessments, respectively, with higher rates for people aged 64 years or less compared to older people[8].

The frequency and intensity of aggression tends to increase as dementia progresses and as cognition, activities of daily living (ADLs) functioning and language abilities deteriorate[3, 58], until the severe stages when it typically declines. Aggression is reportedly as or more prevalent in those with Alzheimer's disease (AD) than those with vascular dementia (VaD)[3, 56]. There is a high prevalence and incidence of aggression in Parkinson's disease dementia (PDD) and related disorders[59]. People with frontotemporal dementia (FTD) present with a higher incidence of more intense physical aggression than those with AD[60, 61].

ASSESSMENT

To reduce the escalating aggression that is placing Bob and others at risk, potentially contributing factors must be identified:

- *Overstimulation (noise, people, activities)*
- *Reduced threshold for coping with stress due to dementia*
- *Pain/discomfort/illness/infection/ constipation*
- *Medication review: interactions, dosage, recently prescribed, adverse effects*
- *Lack of attention to Bob's nonverbal communication*
- *Others expecting too much of him and Bob trying to overextend his capabilities*
- *Altered routines, introduction of new staff, particular staff and/or family members*
- *Unfamiliar/altered/deprived physical environment*
- *Exclude underlying depressive and psychotic symptoms*

Assessing the situation:

- *Encourage Bob to express his needs as far as he is able.*
- *Directly observe what may specifically trigger the aggression.*
- *Ask staff who know Bob quite well if they can assist in identifying his needs or reasons for his aggression.*
- *Consult Bob's life history as well as behaviour and clinical charts for further information with regard to triggers for the aggression.*
- *Assess the immediate environment for potential triggers.*
- *Consult close family members to identify possible triggers for the aggression which may be unknown to staff or not previously documented.*
- *Is pharmacological intervention indicated and/or appropriate for treatment of the aggression?*

Effects of aggression

Aggression, particularly physical aggression, significantly affects those with dementia and others within the care environment. Symptoms are typically very difficult for care partners and residential care staff to address[62-65]. Additionally, there may be cultural differences in how aggression is perceived and addressed[66-69]. Aggression is associated with considerable carer burden and stress, reduced quality of life and earlier admission to residential aged care[16, 54, 70, 71].

One of the worst consequences of aggression is harm to others in the care environment[72-74]. Resident-to-resident aggression, also known as resident-to-resident elder mistreatment, is common in residential care[75, 76] and can lead to serious negative psychological and physical harm including injury and death[77, 78]. Between 12% and 23% of residents engage in mistreatment of others, and approximately 19% to 98% of care staff report observing aggression between residents[76]. Resident-to-resident mistreatment is associated with milder dementia, less functional impairment, the presence of other BPSD, residing in a dementia unit and higher nurse case load[77, 78]. Victims of aggression are commonly females with cognitive impairment and other BPSD, including wandering[9].

In-home and residential care workers frequently experience aggressive responses when assisting people with dementia[73, 79, 80]. Personal hygiene activities, particularly bathing, can provoke anxiety or fear in the person and hence, tend to be the circumstances that most often prompt aggression[11, 81]. Confined spaces and hard surfaces in the bathroom may exacerbate the risks for all involved. When carers are anxious and/or fearful around provoking aggression, they may attempt to complete tasks in the shortest possible time. This can further inflame the situation[10], and neglect important health needs such as oral health and nutrition, subsequently exacerbating poor health and medical conditions[82, 83].

Aggression can also directly influence social functioning and increase isolation due to a loss of opportunities for positive interaction with others[13, 19]. The person with dementia will likely forget the episode long before others do. Aggression directed at primary carers tends to affect the quality of the relationship between the carer and the person with dementia. When this increases carer stress, BPSD can be exacerbated[14, 84]. Additionally, aggression is associated with the use of restrictive practices [85, 86]. The inappropriate use of restrictive practices can negatively influence the quality of life and health of the person with

dementia, cause distress for the person and their family as well as exacerbate the behaviours[87-89] For further information on restrictive practices see *Module 2*.

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded four psychosocial and environmental, and four biological and pharmacological intervention studies with outcomes relevant to aggression in dementia. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012, met quality criteria they have been retained but not included in the summary numbers.

Addressing aggression

The crucial task for the clinician is to attempt to understand what the aggression means for the individual with dementia and to determine cause[90]. Interventions targeting the cause will likely assist in reducing aggression. Where underlying depression or psychotic symptoms are prompting aggression, treatment of these may be helpful. *Appendix 4* provides suggested questions to facilitate comprehensive assessment.

Psychosocial and environmental interventions

Four trials of psychosocial/environmental interventions were conducted in residential settings and one in the community.

Models of care
Quality of research study: 1/1 moderate
Outcome of study: 1/1 limited evidence

The number of episodes of agitation/physical aggression during 24-hour periods in people with moderate to severe dementia significantly decreased, with a medium effect, after three months in a small study of residential care environmental modifications. These included painting walls light beige, installation of "skylike" ceiling tiles, coordination of staff clothing, lighting changes to better differentiate day and night, installation of oversized clocks and streaming soothing music. The evidence is limited because it was not possible to identify the specific aggression component of the positive outcome as agitation/aggression scores were not reported separately[91].

Therapeutic recreation
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A very large study in residential aged care (RAC provided moderate evidence of no benefit for *ElderClown*-delivered humour therapy. Humour therapy decreased duration of angry mood but did not outperform usual care[92]. A medium RCT conducted prior to 2012 which compared live group music with facilitated group reading in people with AD found no benefit for aggression with either intervention[93].

Sensory interventions
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

Strong evidence of no change in CMAI aggression scores was found for twice-daily lavender oil application, with or without hand massage, delivered over six weeks to people living in RAC. Agitation and aggression scores were not reported separately[94].

Education/training
Quality of research study: 2/2 moderate
Outcome of study: 2/2 negative

A very large RAC study provided evidence of no significant decrease in the rate of any physically aggressive behaviour during bath care, including grabbing, hitting, kicking, biting, throwing objects and spitting, after care staff participated in the *Bathing Without a Battle* staff

educational program[95]. A large trial provided moderate evidence of no change in physically aggressive behaviour after either individualised in-home, carer training sessions delivered by BPSD nurses or written instructions with social telephone follow-up over six months[96]. A very large RCT undertaken prior to 2012 demonstrated no benefit for physical or verbal aggression for staff education in practical advice for dealing with BPSD[97].

Psychosocial and environmental interventions are recommended for BPSD as part of an individualised care plan although negative outcomes were reported for four of the five above interventions. There is limited evidence that adapting the living environment may be beneficial[91]. While the evidence for psychosocial interventions is lacking, this should not prevent clinicians from considering these interventions on an individual basis, where they are beneficial to the individual with dementia, enjoyable and culturally appropriate. Aggression in people with dementia can occur in response to many potential antecedents. See *Module 1, Table 1.2* for potentially contributing factors. The identification of triggers or underlying causes can assist in reducing aggression. See *Appendix 2* for interventions reported above.

Biological and pharmacological interventions

Medications are not recommended as first-line treatment for physical or verbal aggression as they can have significant adverse effects. Expert consensus guidelines suggest pharmacological interventions should be used as a second-line approach for aggression when symptoms appear to have a physical or iatrogenic aetiology i.e., stemming from biological causes or in response to medical examination or treatment, symptoms are unresponsive to psychosocial interventions or where residual symptoms are problematic[98, 99]. Although the adverse effects of pharmacological interventions raise concerns, when the person with dementia and/or others around them are at risk, an urgent response may be required. In these cases, expert consensus recommends an atypical antipsychotic for physical aggression that is not due to underlying anxiety or depression[98, 99]. Risperidone is the only PBS approved antipsychotic for this use[99].

Analgesic medication
Quality of research study: 1/1 moderate
Outcome of study: 1/1 some evidence

Pain management should always be considered as a first line treatment for BPSD. A very large RAC study provided some evidence of a very small effect for a stepwise pain management protocol with paracetamol, then buprenorphine, pregabaline and/or morphine daily according to assessed pain needs. Although the study focused on reducing agitation, secondary analysis indicated that CMAI aggressive behaviour factor scores decreased at 8 weeks compared with usual care[100, 101].

Other pharmacological/biological
Quality of research study: 1/1 strong
Outcome of study: 1/1 some evidence

A very large multicentre double-blind placebo-controlled RCT provided strong evidence of a significant decrease in NPI agitation/aggression change scores for dextromethorphan-quinidine which is used to treat pseudobulbar affect or emotional incontinence. Significantly greater benefit than placebo, and compared with baseline, was found in outpatients and RAC-dwelling people with AD at 10 weeks. Insufficient data was provided to calculate the effect size[102]. A large RCT conducted before 2012 provided no evidence for the antiepileptic oxcarbazepine for aggression in people with severe dementia[103].

Cholinesterase inhibitors (ChEIs) and/or memantine
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

The literature published since 2012 provided no evidence for ChEIs or memantine in addressing aggression. Rivastigmine monotherapy failed to change CMAI aggressive behaviour factor scores in outpatients with mild-moderate AD at 24 weeks when compared to baseline[104]. A very

large, pooled analysis of 3 studies published prior to 2012 reported significantly reduced aggression with memantine. While the individual studies showed no benefit, post hoc analysis reported benefit for the combination of agitation/aggression and delusions. There have been no further studies replicating these findings[105].

Antidepressants
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A very large community-based multicentre RCT of people with probable AD and agitation found no benefit for citalopram over placebo. Citalopram add-on to psychosocial intervention did not decrease NPI agitation/aggression subscale scores. Serious adverse effects worsened with citalopram (CitAD)[106].

Atypical antipsychotics

No studies of atypical antipsychotics published since 2012 met our quality criteria for inclusion in this review. A very large RCT conducted prior to 2012, in people with AD, VaD or mixed dementia and aggression, found a significantly greater decrease in CMAI aggression scores for risperidone compared with placebo at 12 weeks[107].

Where atypical antipsychotics are prescribed for the treatment of aggression, guidelines recommend careful monitoring for adverse effects. Their use has been associated with further cognitive decline and risk of somnolence, extrapyramidal symptoms, abnormal gait, oedema, urinary tract infections, metabolic syndrome, incontinence, falls, cerebrovascular adverse events and mortality. See *Module 2, Table 2.3* for associated side effects. Consult local policies and legislation regarding the use of antipsychotic medication.

Although low levels of androgens are associated with cognitive impairment in older men[108, 109], borderline to normal androgen levels in males have been correlated with psychotic symptoms (hallucinations, delusions) agitation, irritability and aggression in men with mild to moderate AD[110]. Studies published before 2012 suggested that treatment with the antiandrogen cyproterone acetate improved this symptom[111], but recent evidence notes associated risk of negative effects on cognition with antiandrogen treatment including cyproterone acetate[110, 112]. Depending on the jurisdiction, it may be necessary to obtain

STRATEGIES/OUTCOMES

- *Bob's frustration appears to be related to his inability to express his discomfort, around overstimulating aspects of his environment, in an appropriate manner. This is likely contributing to the aggressive outbursts. Investigate if Bob has some favourite, soothing music which helps him to relax before his discomfort escalates.*
- *Some staff members reported that Bob can become anxious and more confused when there is a lot of activity happening around him and staff are busy.*
- *Staff identified that Bob has become increasingly socially isolated since his admission to residential care yet he was previously outgoing and enjoyed the company of friends. He is further isolated because other residents and some staff are fearful of his outbursts.*
- *Inservice training sessions with role plays were held to refresh staff knowledge of risk assessment and de-escalation techniques.*
- *Several team meetings were arranged to include all relevant staff members and raise awareness of Bob's subtle, nonverbal signs which may indicate escalating anxiety and/or frustration. This enabled staff to recognise the signs more consistently and more effectively deescalate situations before Bob became aggressive.*
- *Consultation with family members identified activities that Bob previously found relaxing and pleasant such as gardening and playing cards. Family also provided some of his favourite music.*
- *Bob's visitors were encouraged to sit with him in quiet spaces or outdoors.*
- *A trial of small group activities conducted away from the main living area of the aged care home was somewhat effective.*
- *Seating arrangements in the dining room were changed so Bob was able to eat his meals with less talkative residents.*
- *Bob was assisted to participate in gardening in the aged care home raised garden bed and while he was physically limited in what he could do, he apparently enjoyed being around the garden.*
- *Bob's aggression was not eliminated, however follow up assessment indicated that instances were markedly reduced and staff members were better able to address episodes when they occurred.*

consent from a guardianship tribunal or board, not just from the person responsible, before administering hormonal therapy.

Summary

The individual risk/benefit ratio for the person with dementia and the safety of others in the immediate environment must be carefully considered prior to the prescription of pharmacological agents. Where prescribed, the use of such agents should be time-limited, and the situation reviewed frequently[98, 99]. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[99, 113]. For example, where the person presents with aggression and agitation, treating the agitation may also reduce aggression. Additionally, individuals with different types of dementia may respond differently. Those with FTD generally do not respond to ChEIs, and those with dementia with Lewy bodies (DLB) are more vulnerable to the adverse effects of antipsychotics. The potential benefits of effective pain management should not be underestimated in the treatment of aggression[15]. See *Appendix 3* for interventions reported above.

Limitations

There is a paucity of sound research to guide clinicians and carers on addressing aggression in dementia, with most studies offering only moderate evidence. Psychosocial intervention studies are limited in number and effect, and some studies do not differentiate aggression outcomes from those of agitation. Aggression is often not the primary outcome of intervention studies and many studies do not specifically address aggression. Problems occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. While most trials failed to conduct long-term follow-ups, three studies completed short-term follow-up assessments between four and fourteen weeks[92, 94, 100, 101]. Beneficial effects were not maintained after interventions ceased.

Conclusions/Principles of care

In summary, while aggression in dementia is relatively uncommon, there are significant consequences for both the person with dementia and others within the care environment. Reports of positive outcomes, particularly for psychosocial interventions, are lacking. A lack of sound evidence should not prevent clinicians from considering strategies on an individual basis[114]. Limited support was demonstrated for environmental modifications. Expert consensus guidelines recommend the use of multidisciplinary, individualised and multifaceted care including psychosocial interventions and short-term pharmacological treatment only when necessary. Good clinical practice suggests tailoring psychosocial interventions to individuals. For example, a person-centred approach[115] may indicate that for one person reducing aggression may result from changing their personal care routine, for another it may be prescribing analgesics for osteoarthritic pain prior to personal care activities and for a third, it may be arranging outdoor walks with a carer or family member to alleviate feelings of imprisonment.

Where pharmacological treatment is indicated, stepwise pain management and dextromethorphan-quinidine provide some evidence for the treatment of aggression. The risk of adverse effects limits the use of antipsychotics although they may be indicated in urgent situations, in the context of psychosis or a subsequent treatment where other interventions have failed. Clinical trials of biological treatments continue[116].

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

4. Agitation

MODULE 4: Agitation

Key messages

- Agitation in dementia presents as emotional distress with observable, non-specific, restless behaviours that are excessive, inappropriate and repetitive.
- Agitation occurs as a product of the interaction between individual and environmental factors including neurobiological changes, biological causes extrinsic to dementia and unmet biopsychosocial needs such as social isolation.
- Symptoms of agitation can overlap with aggression and delirium which can lead to misdiagnosis.
- Comprehensive assessment is required to differentiate between these conditions, identify underlying issues and develop appropriate strategies to address agitation.
- Agitation is one of the most commonly occurring BPSD, with prevalence rates ranging from 30%-80% according to care setting and 22%-50% according to dementia type.
- Agitation is associated with poorer quality of life, an increase in the inappropriate use of restrictive practices and greater burden on carers.
- Individualised psychosocial interventions are recommended as a first-line approach and short-term pharmacological interventions only when necessary.
- Multicomponent interventions provide the best psychosocial evidence.
- Where pharmacological/biological interventions are indicated rivastigmine, galantamine or light exposure may warrant a trial.
- Some evidence is reported for atypical antipsychotics however, these are not recommended due to safety concerns.
- Expert consensus guidelines recommend multidisciplinary, individualised and multifaceted care.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and addressed, reversible causes of the BPSD

    (See *Module 1* for further details)

Agitation summary

What is agitation and what does it look like in dementia?

Agitation in dementia refers to observable, non-specific, restless behaviours that are excessive, inappropriate and repetitive. It may present as:

- irritability and disruptive vocalisations
- aberrant motor activities such as excessive restlessness and/or pacing
- observed or inferred emotional distress, and/or
- excessive negative physical actions directed at objects, self, or others.

Causes of agitation

Agitation occurs as a product of the interaction between individual and environmental factors:

- neurobiological changes intrinsic to dementia such as neurofibrillary tangle burden and neurotransmitter systems
- biological causes extrinsic to dementia including unmet care needs, pain, medical comorbidities and drug effects
- unmet biopsychosocial needs such as social isolation
- feelings of fatigue, anxiety, frustration and loss of independence.

Differential diagnosis

The International Psychogeriatric Association (IPA) consensus definition is the current standardised definition for diagnosis of agitation in cognitive disorders. Symptoms of agitation in dementia can overlap with aggression. Hyperactive delirium can be misdiagnosed as agitation and both can arise from potentially reversible organic factors. Comprehensive assessment is required to differentiate between these conditions, identify underlying issues and develop appropriate strategies to address agitation.

Measuring agitation

Guidelines recommend the use of the Cohen-Mansfield Agitation Inventory (**CMAI**), the Pittsburgh Agitation Scale (**PAS**) as well as the Agitation/Aggression and Aberrant Motor Behaviour subscales of the Neuropsychiatric Inventory (**NPI**) and the Agitation subscale of the NPI-Clinician (**NPI-C**). Other commonly used instruments include the NPI-Questionnaire (**NPI-Q**), Agitated Behaviour Mapping Instrument (**ABMI**), the Brief Agitation Rating Scale (**BARS**) and the Excited Component of the Positive and Negative Syndrome Scale (**PANSS-EC**).

Prevalence of agitation

- Agitation is one of the most commonly occurring BPSD, with an average prevalence of 30%-80% according to care setting and 22%-50% according to dementia type.
- Highest rates are reported for Alzheimer's disease (AD) and vascular dementia (VaD).
- Not all instances are clinically significant.
- Prevalence rates vary depending on the different definitions of agitation and the instrument used to measure agitation.
- Increased agitation has been associated with faster rate of cognitive and functional decline and poorer activities of daily living (ADL) functioning.

Effects of agitation

Agitation is associated with poorer health-related quality of life for the person with dementia, an increase in the inappropriate use of restrictive practices and premature admission to residential aged care services (RACS). Agitation results in greater burden on care partners and family as well as in-home, community, acute and RACS staff.

Addressing agitation

Potential triggers and/or underlying causes behind the agitation for the individual with dementia should be identified where possible. Where the person with dementia and/or others are not at risk and reversible causes have been eliminated, consider psychosocial and/or environmental interventions as a first option.

Psychosocial and environmental interventions

- 64 studies met our inclusion criteria.
- Psychosocial intervention studies were primarily conducted in residential settings.
- Multicomponent interventions provided the best psychosocial evidence for reducing agitation.
- Education/training interventions incorporated the greatest number of studies and the highest number of strong quality studies however, the majority reported negative outcomes.
- Cognitive rehabilitation/stimulation and reminiscence interventions provided no evidence of benefit.
- Effective interventions should be beneficial to the individual, enjoyable, respectful, culturally appropriate and culturally safe.

Biological and pharmacological interventions

- 20 studies met our inclusion criteria.
- Pharmacological interventions should only be used as a second-line approach in addressing agitation.
- Risk/benefit ratios for the individual person with dementia must be considered.
- Biological and pharmacological intervention studies were conducted in community/ outpatient, residential aged care and inpatient hospital settings.
- The Cholinesterase inhibitors (ChEIs) and/or memantine, atypical antipsychotics, tetrahydrocannabinol (THC)/cannabinoids and brain stimulation categories included four studies each.
- All categories provided some evidence of, at least, limited benefit although adverse effects raise concerns with their use.
- Where pharmacological/biological interventions are indicated, rivastigmine, galantamine or light exposure may warrant a trial.
- Although only one trial of analgesic medication was reported and showed benefit, pain management should always be considered.
- Some evidence was demonstrated for atypical antipsychotics however, current guidelines do not recommend their use due to the associated risks.
- The evidence reported for cannabinoids, citalopram and dextromethorphan/quinidine is too limited for recommendations to be made.

Limitations

Many studies have methodological and sampling issues which impact on the outcomes and/or potentially limit the generalisability of results. A definitive diagnosis of agitation in dementia can be difficult due to overlapping symptoms of aggression, sundowning and other BPSD. Limited evidence of sustainability of effects is available as only one study assessed long-term outcomes and five reported short-term outcomes.

Conclusions/Principles of care

Expert consensus guidelines recommend multidisciplinary, individualised and multifaceted care, including individualised psychosocial interventions as a first-line approach when addressing agitation in dementia and short-term pharmacological intervention only when necessary.

What is agitation and what does it look like in dementia?

Agitation accompanies observed or inferred emotional distress and represent at least one of three categories:

1. excessive motor activity such as restlessness, pacing, rocking or hand wringing,
2. excessive vocalisations such as overly loud speaking, yelling, screaming or using profanities and/or
3. excessive negative physical actions directed at objects, self, or others such as throwing, slamming, hitting, kicking, tearing, scratching, and biting[1, 2].

Other non-specific, disruptive symptoms are indiscriminately classified as agitation and this may arise from a previous lack of consensus around the conceptualisation of agitation. The International Psychogeriatric Association (IPA) developed provisional criteria in 2015[3], and a final definition based on expert clinical and research consensus considering clinical guidelines and the experience of people living with dementia and family advocates in 2023[2]. A standardised definition facilitates identification and assessment using clinical skills and commonly available tools, and a more targeted approach to agitation.

The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11) includes *Agitation or aggression* under a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" under Neurocognitive disorders, Dementia. Agitation is described as "clinically significant excessive psychomotor activity accompanied by increased tension"[4]. Sundowning is a related clinical phenomenon, which refers to the appearance or exacerbation of behaviours in the late afternoon or early evening. Various aetiologies of sundowning have been proposed, including unmet biopsychosocial needs, fatigue, diurnal mood variations, sleep disturbances and inadequate daylight exposure[5-7].

It is important to consider what agitation may mean to the person with lived experience of dementia. When clinicians identify 'agitation', people living with dementia and care partners report that they may be feeling and/or expressing frustration, fear, stress, worry, empathy, panic, anxiety, loss of confidence, intolerance, impatience and/or annoyance[8, 9].

Causes of agitation

The neurobiological underpinnings of agitation are not well understood. Recent research suggests decreased frontal[10] or temporal lobe metabolism[11, 12], increased neurofibrillary tangle burden[11, 13], and altered activity in the neurotransmitter systems due to trauma and/or neuroinflammatory disorders such as COVID may all play a role[14].

PRESENTATION

Kirra is a 70-year old Aboriginal man from a regional town on the NSW north coast. He is dependent on his daughters for many of his care needs. To provide some respite for his family and hopefully provide him with additional company and quality time, Kirra has recently started attending a day respite service.

He is wary of care staff, particularly those who are from non-Aboriginal or Torres Strait Islander backgrounds. During group activities Kirra often leaves the group and walks around the centre, touching items belonging to other attendees and artwork on the walls. The situation can quickly become risky when Kirra intrudes into the personal space of some of the other attendees and they become angry. Kirra's reaction is to become increasingly agitated and, at times, verbally aggressive. If anyone approaches Kirra at this point, he can become combative and the situation continues to escalate.

Kirra's daughters report that he is increasingly reverting to his Indigenous language which limits Kirra's communication with some of his family and the day centre staff. His increasing frustration with being unable to communicate with staff and other centre attendees also prompts agitation. At times he has become agitated to the point where he attempted to leave the day respite centre, increasing the risk to his safety.

Agitation is a product of the interaction between individual and environmental factors including unmet care needs, premorbid personality[15, 16], non-verbal communication impairment[17], medical comorbidities[10, 18], dementia type and younger-onset[19]. Other factors include substance use/misuse including psychoactive drugs[19, 20], poorly managed pain[21-23], iatrogenic causes[24, 25], inappropriate clinician/care staff communication[26], use of restrictive practices[27, 28] as well as unmet biopsychosocial needs such as a lack of sensory stimulation or social interaction[29]. Agitation may also be associated with more affective and psychotic symptoms[30].

People living with dementia and their care partners have described their "agitation" occurring in response to changing circumstances, fatigue, anxiety, frustration with themselves and others, loss of independence and sense of identity, past or ongoing traumas as well as feeling unsupported, misunderstood or unsafe[8, 9, 31, 32].

Differential diagnosis

The IPA consensus definition is the current standardised definition for diagnosis of agitation in cognitive disorders[2]. It reflects symptomatic overlap between agitation and aggression[2] although others suggest that agitation and aggression are different syndromes[33]. Aggression is generally intentional, violent, intense and harmful, lacking the repetitiveness and non-specificity of agitation[33]. Hyperactive delirium can be misdiagnosed as agitation[34] and both can arise from potentially reversible organic factors. Differing aetiologies, and hence strategies, necessitate differentiating between agitation, aggression and delirium, and identifying the underlying issues[2, 10, 35].

Measuring agitation

In line with the inconsistencies in the nosology of agitation, numerous instruments have been developed to assess different aspects of agitation[10, 36]. Informant ratings of agitation converge moderately with ratings based on direct observations[37]. While observation-based assessments may be more time consuming to administer, they are less prone to bias when compared to carer-rated instruments relying on retrospective reporting[38]. Most agitation scales were developed in English[39], however more language appropriate tools have been developed such as a Thai tool (BPSD-T)[40]. The Neuropsychiatric Inventory (NPI)[41] and NPI-Q[42] have been adapted and validated in multiple languages[43]. There is no recommended scale for measuring agitation in older Aboriginal and Torres Strait Islander populations. Australian guidelines recommend the following scales for the measurement of agitation in dementia[44]:

- The Cohen-Mansfield Agitation Inventory (**CMAI**) is a carer-rated questionnaire that quantifies the frequency and disruptiveness of 29 agitated behaviours over two-weeks. Separate scores can be calculated for physically aggressive, physically non-aggressive and verbally agitated behaviours[45]. Additional versions include the 14-item short form (CMAI-SF)[46], and a 36-item version for day centres in community settings (CMAI-C)[47].
- The Pittsburgh Agitation Scale (**PAS**) assesses the presence and severity of agitation symptoms under aberrant vocalisation, motor agitation, aggressiveness and resisting care based on short periods of direct observation. The scale offers greater flexibility to suit different clinical needs[48].
- The agitation/aggression and aberrant motor behaviour subscales of the **NPI** are completed during a clinical interview with the carer, in which they rate the frequency and severity of the agitation/aggression, as well as their own subsequent distress[41]. The reliability and validity of the NPI overall is well established[49]. Individual NPI symptom domains can be more clinically relevant than a total NPI score[43, 50].
- The NPI-Questionnaire (**NPI-Q**) is a 12-item self- or clinician-administered tool that rates symptom severity and distress in 12 domains, including agitation/aggression, over the past month[42]. The NPI-Q does not rate symptom frequency.
- The NPI-Clinician (**NPI-C**) version has revised the original NPI Agitation/Aggression subscale, with an expanded Agitation subscale[51] including unwanted requests for attention,

repetitive questioning, restlessness, fidgeting, complaining, hyperventilating, refusing medication, pacing, aggressively trying to get to a different place, crying with frustration and trying to enlist help inappropriately.

Other scales have also been widely used to measure agitation in research and clinical settings:

- The Agitated Behaviour Mapping Instrument (**ABMI**) is an observational tool that assesses the frequency of 14 agitated behaviours as well as the environment in which these occur over a three-minute period[45].
- The Brief Agitation Rating Scale (**BARS**) is a carer-rated scale that measures the frequency of 10 agitated behaviours over the previous two weeks[52].
- The Excited Component of the Positive and Negative Syndrome Scale (**PANSS-EC**) rates agitation based on excitement, tension, hostility, uncooperativeness and poor impulse control[53, 54].
- Agitated Behavior in Dementia Scale (**ABID**) is a carer-rated tool which assesses the frequency of, and carer's reaction to, 16 behaviours over the previous two weeks[55, 56].
- Neurobehavioral Rating Scale (**NBRS**) is a 28-item observational tool that measures cognitive and noncognitive symptoms in dementia including a 9-item agitation/disinhibition factor score[57].
- The Disruptive Behavior Rating Scales (**DBRS**) is a 21-item scale that rates four dimensions: physical aggression, verbal aggression, agitation and wandering over a week based on observation or chart review[58].
- The Overt Agitation Severity Scale (**OASS**) is an observational tool for rating the severity of 12 physical behaviours based on vocalisation as well as oral/facial, upper torso and lower extremity movements during 15-minute periods[59].

Two novel agitation scales have been developed for use in clinical trials. The NPI-C-IPA and the CMAI-IPA are clinician-rated measures that incorporate International Psychogeriatric Association (IPA) criteria for agitation in Alzheimer's disease (AD). Early analyses indicate good reliability[60].

Prevalence of agitation

Agitation is one of the most commonly occurring changed behaviours in dementia with an average prevalence of 30%-80% according to care setting and 22%-50% according to dementia type, with the highest rates reported for AD and vascular dementia (VaD)[10, 61]. Agitation is the most frequently recorded primary behaviour in Dementia Support Australia assessments at 34.9% in 2022 with highest rates reported for females[62].

Variance in prevalence arises from the different definitions of agitation, the instruments used for assessment and/or the population studied e.g. community or residential aged care services (RACS). Although agitation is observed in the majority of those with dementia, the level of disruption is not always clinically significant[61]. Increased agitation has been associated with faster rate of cognitive and functional decline and poorer activities of daily living (ADL) functioning[63]. In people with younger onset dementia (YOD) living in RACS, increased agitation has been associated with greater dementia severity and greater impairment in insight[19].

Effects of agitation

Agitation is often viewed as one of the most challenging and persistent behaviours manifesting in people with dementia. It is linked to an increased likelihood of neuropsychiatric or medical comorbidity[18, 64, 65], poorer health-related quality of life[66] and significant functional impairment in activities of daily living (ADL)[67] as well as increased burden on family[68], RACS and acute care staff[69, 70]. Agitation can precipitate admission to residential care[71].

Although expert consensus recommends non-pharmacological interventions as a first-line approach for agitation in dementia[72, 73], it is associated with increased use of restrictive practices[18, 28, 74-76].

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded 64 psychosocial and environmental and 20 biological and pharmacological intervention studies with outcomes relevant to agitation in dementia. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained but not included in the summary numbers.

Addressing agitation

It is important to attempt to determine the potential triggers and/or underlying causes behind the agitation for the individual with dementia. Episodes of agitation may be linked with specific environmental stimuli, traumatic memories, incidents or people. A personalised[77], trauma-informed[31, 32, 78] approach can assist in addressing symptoms and increase the potential for therapeutic benefit. Interventions targeting the cause will likely assist in reducing the agitation. Underlying physical symptoms, pain, discomfort and or medication effects must also be addressed. *Appendix 4* provides suggested questions to facilitate comprehensive assessment.

Where the person with dementia and/or others are not at risk and reversible causes have been eliminated, consider psychosocial and/or environmental interventions as a first option. The International Psychogeriatrics Agitation Work Group have published a consensus algorithm contained in the Investigate, Plan and Act (IPA) approach to agitation reduction and prevention[24].

Psychosocial and environmental interventions

Psychosocial and environmental intervention studies were primarily conducted in residential care settings. Although the education/training category incorporated the greatest number of studies and the highest number of strong quality studies, the majority reported negative outcomes. The cognitive rehabilitation/stimulation and reminiscence categories provided no evidence of benefit.

Therapeutic recreation
Quality of research studies: 2/5 strong, 3/5 moderate
Outcomes of studies: 4/5 mixed/limited/some evidence, 1/5 negative

ASSESSMENT

To reduce the presenting agitation that may be placing Kirra and others at risk, potentially contributing factors must be identified:

- *Chronic or acute pain/discomfort/ illness/infection*
- *Medication interactions, dosage, adverse effects, recently prescribed*
- *Exclude underlying depressive and psychotic symptoms*
- *Overstimulation (noise, people, activities)*
- *Lack of attention to culturally-relevant needs and historical trauma*
- *Others expecting too much of him and Kirra trying to overextend his capabilities*
- *Altered routines, new staff, particular staff or family*
- *Unfamiliar/altered/deprived physical environment*
- *Stopping Kirra from what he is doing or wanting to do*
- *Reduced threshold for coping with stress*

Assessing the situation:

- *Encourage Kirra to express his needs as far he is able.*
- *Directly observe what may trigger the agitation.*
- *Ask staff who have got to know Kirra if they can assist in identifying unmet needs, or possible reasons for his agitation.*
- *Consult Kirra's life history as well as behaviour charts for further information with regard to triggers for his agitation.*
- *Assess the immediate environment for possible triggers.*
- *Consult close family members to identify possible triggers for the agitation that may be unknown to day centre staff and not previously documented.*
- *Are pharmacological interventions indicated and/or appropriate for addressing the agitation?*

All therapeutic recreation studies took place in RACS. A large study found moderate evidence of a small to large effect for all *Treatment Routes for Exploring Agitation* (TREA) individualised activities except music, with decreased total agitation related to higher levels and duration of engagement[79]. Some evidence of benefit was demonstrated in a very large study of moderate to strong quality for ElderClown-delivered humour therapy. Agitation also decreased significantly at 13 week post-intervention follow up[80-82].

Evidence was presented of a very small effect for both personalised one-to-one *Montessori* interventions and non-personalised social interaction activities with a decrease in the number of observed agitated behaviours in both groups[83, 84]. *DementiAbility Methods the Montessori Way* tailored activities and therapeutic environment decreased total agitation and disruptiveness scores but structured social activities showed no change[85]. Both studies were of medium size and moderate quality. A medium study of lifelike baby dolls indicated strong evidence of no benefit[86].

Cognitive rehabilitation/stimulation
Quality of research studies: 2/2 moderate
Outcomes of studies: 2/2 negative

Two medium studies, one undertaken in hospital and one in RACS provided moderate evidence of no benefit for brain-activating rehabilitation[87, 88].

Reminiscence-based interventions
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A small RCT that compared weekly sessions using a *Memory Box* personalised computer app and social visits with reading/discussing current events reported moderate evidence for no benefit for either activity after four weeks[89].

Physical activity
Quality of research studies: 1/3 strong, 2/3 moderate
Outcomes of studies: 2/3 mixed/limited/some evidence, 1/3 negative

A medium community study of people with AD and sundowning reported moderate evidence of a small effect after 6 months of walking 30 minutes 4 times weekly with family carers when compared with usual activities[90]. A large hospital-based RCT comparing a small group exercise program of weights/endurance exercises 3 times weekly with a 2 hour social table games program weekly reported some benefit in verbal agitation for exercise over socialising[91]. In contrast, a medium community-based study comparing 45-minute sessions of chair yoga, customised music and gentle chair-based exercise twice weekly provided moderate evidence of no decrease in agitation after 12 weeks[92].

Music
Quality of research studies: 7/7 moderate
Outcomes of studies: 1/7 positive, 3/7 mixed/limited/some evidence, 3/7 negative

All music studies were of moderate quality and conducted in residential care, with one exception undertaken in hospital[93]. Twice-weekly 30-minute individual sessions of active or passive engagement with music, decreased disruptive agitation compared with usual care over 6 weeks for residents with moderate to severe dementia[94]. By contrast, another study of people with moderate to severe dementia provided evidence of no benefit over usual care for streaming preferred music to residents' rooms[95]. A trial of music therapy with percussion instruments reported no benefit over usual care for residents with mild to moderate dementia over 6 weeks[96]. All three studies were medium in size.

Two large studies provided contrasting outcomes for small group music sessions delivered by certified music therapists. One study reported benefit with a small effect for preferred music with instruments after two-weeks, when compared with usual care[97]. An RCT reported no change in agitation and no benefit for music therapy with instruments, singing, and/or dancing over typical recreational activities after 4 months[98].

Daily 30-minute individualised music sessions via cordless headphones reduced agitation with a large effect in people hospitalised for acute BPSD, including those with severe AD, compared with usual care over an average 23-day hospital stay[93]. In a trial that compared twice-weekly 1-hour small group guided interventions, cooking and reminiscence sessions decreased agitation with a medium effect after 4 weeks but interactive music and reminiscence showed no effect for people with moderate to severe dementia[99]. Both studies were medium in size. A further medium RACS study conducted prior to 2012 provided evidence of no decrease in agitation for facilitated live group music compared with facilitated group reading[100].

Animal-assisted
Quality of research studies: 5/5 moderate
Outcomes of studies: 1/5 some evidence, 4/5 negative

All animal-assisted studies were medium in size with one study conducted in the community[101] and the remainder in RACS. Weekly visits focused on entertainment and social interaction with a therapy dog and their human guide did not lower agitation after 10 weeks but performed better than usual care for people with moderate to very severe dementia[102]. Small group interactions with a therapy dog and their handler twice weekly over 12 weeks did not outperform usual group activities for people with severe dementia[103]. No study of the companion robot *Paro* provided evidence for significantly reduced agitation, or provided greater benefit than usual care and activities, for people with dementia living in the community or RACS[101, 104, 105].

Sensory interventions
Quality of research studies: 1/6 strong, 5/6 moderate
Outcomes of studies: 1/6 positive, 2/6 some evidence/possibly positive, 3/6 negative

All sensory studies were medium and conducted in RACS. All provided moderate quality evidence, with one exception[106]. Twice-weekly *Snoezelen*® multisensory room sessions, and the same schedule of individualised music, decreased agitation scores with a medium effect for both interventions at 16 weeks and no difference between groups[107]. Two similar studies compared *Snoezelen*® sessions with therapist-led one-to-one activity sessions over 8 weeks[108] and 16 weeks[109, 110], respectively. Both interventions decreased agitation with a large effect.

Two hours of aromatherapy daily with lavender or lemon balm essential oil attached to clothing did not decrease agitation for residents with moderate dementia after two weeks[111]. Likewise, no change in agitation was reported for weekly lavender and orange essential oil mixture massage over 8 weeks[112] and strong evidence indicated no change for twice-daily lavender oil application with or without hand massage over six weeks[106].

Touch therapies
Quality of research studies: 2/5 strong, 3/5 moderate
Outcomes of studies: 1/5 positive, 4/5 negative

All trials of touch therapies were conducted in residential care with one exception set in hospital[113]. A very large study of lavender oil applied to 5 acupoints 5 times weekly, with and without acupressure, reported moderate evidence for decreased agitation for both aromatherapy and aroma-acupressure after 4 weeks[114]. A medium study of moderate quality provided no good evidence of decreased agitation for acupressure to 4 acupoints in residents with moderate to severe dementia[115]. Likewise, a large RCT provided strong evidence of no benefit for acupressure using real acupoints over sham acupoints, and no benefit over usual care[116].

A medium study that compared daily foot massage with sitting near the person to provide *quiet presence* reported strong evidence that both interventions *increased* agitation after 3 weeks for residents with moderate to severe dementia[117]. Twenty-minute hand and forearm massage provided in a medium hospital inpatient study showed moderate evidence of no benefit over usual care[113]. One medium RCT conducted prior to 2012 indicated limited evidence of benefit for therapeutic touch over placebo touch in people with moderate to severe dementia[118].

Education/training
Quality of research studies: 5/11 strong, 6/11 moderate
Outcomes of studies: 1/11 positive, 2/11 some evidence, 8/11 negative

A very large RCT undertaken in RACS, *Targeted Interdisciplinary Model for Evaluation and Treatment of Neuropsychiatric Symptoms* (TIME), compared brief dementia/BPSD staff education with and without additional supervised education to support tailored treatment measures for BPSD. Strong evidence indicated decreased agitation for enhanced education, with medium effect, when compared with brief education after 12 weeks[119]. Moderate evidence of some benefit for in-home family carer training on the *Progressively Lowered Stress Threshold* (PLST) model with 'phone support was demonstrated over educational materials only in a medium study after three months[120]. A small community study of psychiatrist-led carer education sessions, including *Antecedent-Behaviour-Consequence* (ABC) training[121], provided moderate evidence of some benefit after 3 months[122].

A study comparing BPSD/PCC education, clinical protocol training and clinical support sessions reported strong evidence of no significant benefit over usual care after 12 weeks[123]. Moderate evidence of no benefit was also demonstrated for *Trust Before Restraint* staff training covering decision-making, legislation and PCC over usual care after seven months[124, 125]. A psychologist-delivered education program for RACS staff, *Managing Agitation and Raising Quality of Life (MARQUE),* with implementation/supervision reported strong evidence of no significant difference in agitation when compared with usual care after 8 months[126]. These three studies were very large.

A medium community study of moderate quality reported no benefit for a weekly 2-hour program *Powerful Tools for Caregivers* (PTC) over six weeks[127]. A very large community study reported strong evidence of no benefit for the *Preventing aggression in veterans with dementia* (PAVeD) carers' program, which included weekly in-home visits, pain education and introducing pleasant activities, over enhanced usual primary care after 6 weeks[128].

Two large trials of the *Function-Focused Care for the Cognitively Impaired* (FFC-CI) education for RACS care workers, aiming to optimise function in residents with moderate to severe dementia, showed moderate quality evidence of no additional benefit over 30-minute in-service education sessions after 6 months[129, 130]. A very large RCT of *Dementia Education Programme Incorporating Reminiscence for Staff* (DARES)[131] provided strong evidence of no additional benefit over usual care at 12 or 16 weeks post-intervention[132].

Two very large RCTs conducted in residential care prior to 2012 provided mixed evidence for staff training with support. One study reported a significant decrease in agitation[133] whereas a study of staff training in PCC showed no benefit[134].

Models of care
Quality of research studies: 2/10 strong, 8/10 moderate
Outcomes of studies: 1/10 positive, 4/10 mixed/limited/some evidence, 5/10 negative

All models of care studies were conducted in residential care with one exception undertaken in shared housing[135]. A very large study indicated strong evidence of decreased agitation for staff training plus PCC, and for staff training plus person-centred environment (PCE), with a large effect when compared with usual care after 8 months[136]. *Treatment Routes for Exploring Agitation* (TREA) interventions, including personal care activities, based on the unmet needs model of care[137] demonstrated moderate evidence with a large effect when compared with staff education, in a large study of residents with advanced dementia[138].

Staff dementia training supplemented with *Dementia Care Mapping* (DCM)[139] or *VIPS practice model of PCC* (VPM)[140] demonstrated limited evidence of decreased agitation scores for DCM supplemented training only but outcomes were no more effective than dementia training alone after 10 months[141]. Two cycles[142] and three cycles of DCM[143] showed no significant change in CMAI scores and no benefit over usual care after 8 and 16 months, respectively. These three studies were very large and of moderate quality.

A very large, moderate quality study of *Comprehensive stepwise training for multidisciplinary teams* (STA OP!) showed some benefit for agitation and restless tense behaviour with a large effect compared with dementia/pain management/general skills training after 3 months[144, 145].

Two very large studies were conducted in dementia units. A strong study of multidisciplinary evidence- and practice-based training indicated equivocal evidence of decreased agitation when compared with usual care after 20 months[146]. Another multidisciplinary program *Grip on Challenging Behaviour* demonstrated moderate evidence of no beneficial effect on agitation or psychotropic drug use after six months for people with YOD[147].

Two 12-month studies in smaller-scale living contexts provided moderate evidence of no benefit. A large study of a tailored quality development process for shared houses demonstrated no decrease in CMAI scores and no benefit over shared housing usual care[135]. Small-scale residential living units were associated with more social engagement but *increased* physically non-aggressive agitated and aberrant motor behaviours when compared with traditional larger-scale units in a very large study[148].

Multicomponent interventions
Quality of research studies: 3/9 strong, 6/9 moderate
Outcomes of studies: 2/9 positive, 5/9 limited/some evidence, 2/9 negative

All multicomponent studies were undertaken in residential care with one exception set in the community[149]. A very large study provided strong evidence for guideline-based interventions including assessment and medication optimisation compared with tailored OT-provided activities in residents with moderate-severe dementia, with a small to medium effect[150]. A large study of moderate quality compared the efficacy of weekly aroma-massage, reminiscence or cognitive stimulation sessions for people with moderate to severe dementia. Decreased CMAI scores with medium effect were reported for aroma-massage only after 10 weeks[151].

Moderate evidence in a medium study indicated a small-to-medium effect for decreased NPI agitation and aberrant motor behaviour scores in people with moderate dementia, but no change in NPI irritability, after 8 weeks of twice-weekly 30-minute interactive therapy sessions to stimulate senses and encourage social interaction e.g., relaxation, music, dancing[152]. Creative, meaningful activities with sensory stimulation outperformed usual care in a medium study of residents with severe dementia. Moderate quality evidence with a large effect was reported[153]. Limited evidence of decreased agitation was provided for different combinations of exercise and cognitive training five times weekly in a large, moderate quality study of residents with moderate dementia[154].

Community-dwelling people who learned and performed dual musical-physical tasks simultaneously for 60 minutes twice weekly e.g., singing, walking/stepping to the beat showed moderate evidence of a greater decrease in agitation with medium effect than those who performed non-musical cognitive and physical tasks sequentially e.g. cards, puzzles, then walk with the therapist. This was a medium, 2 month study of people with mild to moderate dementia[149].

A strong, very large trial of staff training in PCC plus tailored activities and/or antipsychotic medication review based on NICE Dementia Guidelines (*Well-being and Health for People with Dementia* (WHELD) intervention)[155] in the context of supporting staff with coaching, supervision and review provided some evidence of a small positive effect on agitation when compared with usual care in residents with moderate to severe dementia[156]. However, another very large study provided strong evidence that implementing all the WHELD components together in the context of ongoing support over 9 months did not significantly change agitation scores in residents with mild to severe dementia[157]. A medium study provided moderate evidence of no decrease in agitation for various combinations of massage, aromatherapy, exercise, acupressure, cognitive training and stretching[158].

Summary

Interventions that aim to address the unmet biopsychosocial needs of the person with dementia showed some beneficial effects for agitation, with multicomponent interventions providing the best evidence overall. Professional consensus recommends psychosocial interventions as part of an individualised care plan. Strategies also include adapting the living environment, allowing people with dementia to participate in structured activities as well as providing training and support to family and formal carers. Where they are beneficial to the person, enjoyable, respectful, culturally appropriate and culturally safe, the suitability of interventions should be considered on an individual basis[24, 159, 160]. See *Appendix 2* for interventions reported above.

Biological and pharmacological interventions

Expert consensus guidelines suggest pharmacological interventions should only be used as a second-line approach for agitation, typically when symptoms appear to have a physical or iatrogenic aetiology i.e. stemming from biological causes or in response to medical examination or treatment, are unresponsive to psychosocial interventions or where residual symptoms are problematic. Biological/ pharmacological intervention studies were conducted in community/outpatient, RACS and inpatient hospital settings. All categories reported provided some evidence of, at least, limited benefit although adverse effects raise concerns with their use.

STRATEGIES/OUTCOMES

- *Any one of Kirra's comorbid illnesses may be causing discomfort or pain. Limited access to health services and transport within the community can preclude regular medical treatment. A medical review was arranged with the assistance of male family members.*
- *When family provided relevant details of Kirra's history, it became evident that his past experiences as a member of the stolen generation and his ongoing fear of institutions may provoke anxiety around being taken out of his community for day respite. Community members initially attended the day respite centre with Kirra, for part of the day, to assist in his adjustment to the unfamiliar environment.*
- *Visual resources and pictorial language aids were developed and/or sourced with the assistance of community members familiar with Kirra's language. Attempts to locate a language-appropriate interpreter were unsuccessful.*
- *An older Aboriginal man who is a nearby neighbour to the respite centre was originally from the same Country as Kirra and he had some knowledge of his language. He was willing to assist with communication when he was available and regularly spend some time yarning with Kirra.*
- *Some staff members at the respite centre had little knowledge of dementia, BPSD and/or trauma-informed care. They became fearful of Kirra, unsure when he may become agitated.*
- *Education was provided to improve the care and support of clients with dementia as well as increase staff members' confidence to effectively fulfil their role.*
- *The respite centre was located on different Country to Kirra's own, causing him distress when he forgot that he would be returning home later in the day. With a better understanding of dementia, staff at the centre were able to more effectively provide Kirra with regular reassurance.*
- *Limited experience and knowledge of dementia and BPSD within Kirra's extended family and community was increasing his daughters' stress and isolation. Local Aboriginal services visited the community to raise awareness of dementia and provide culturally-appropriate education and information on BPSD.*
- *Family assisted the respite centre staff to plan appropriate activities, relevant to Kirra's interests and background. Kirra had previously been a keen painter and he responded positively to staff providing large sheets of paper and safe art materials for new paintings.*
- *Kirra's daughter brought one of her father's earlier artworks to hang in the day centre. These strategies provided staff with opportunities to interact positively and meaningfully with Kirra as well as praise his work.*
- *The frequency and severity of Kirra's agitated episodes reduced. Staff and family agreed that he was largely enjoying his time at the centre. Kirra's daughters benefitted from the regular respite and were relieved that he was now willing to attend without resistance. Staff reported improved confidence in their capacity to de-escalate Kirra's agitation when they observed the early signs of his distress.*

Cholinesterase inhibitors (ChEIs) and/or memantine
Quality of research studies: 2/4 strong, 2/4 moderate
Outcomes of studies: 1/4 positive, 2/4 some evidence, 1/4 negative

Primarily prescribed for cognitive symptoms in dementia, ChEIs may play a role in addressing agitation, although overall the evidence is mixed. One large community study provided moderate evidence that rivastigmine patch monotherapy outperformed rivastigmine patch plus memantine with small to medium effect. Monotherapy decreased CMAI nonaggressive agitated behaviour scores compared with baseline while combination therapy *increased* these scores at 24 weeks[161].

A large community RCT that compared galantamine with risperidone over 12 weeks provided strong evidence of significant decrease in total CMAI scores at 12 weeks for both medications, but a significantly greater decrease for risperidone with small-to-medium effect[162, 163]. A very large 24-week multicentre, RCT withdrawal trial in RACS residents with probable AD compared memantine and antipsychotics i.e. risperidone, olanzapine, quetiapine or haloperidol. Strong evidence of no benefit for either group on CMAI scores at any time point was reported[164].

A very large community RCT of up-titrated memantine in people with moderate-to-severe AD provided moderate evidence of no benefit for memantine over placebo at 24 weeks[165]. One very large RCT published prior to 2012 reported some evidence of reduced agitation for donepezil[166], while a large study of memantine indicated no effect in people with AD[167].

Analgesic medication
Quality of research study: 1/1 moderate
Outcome of study: 1/1 some evidence

A very large RACS study provided evidence of very small to small effect for a stepwise pain management protocol with paracetamol then buprenorphine, pregabalin and/or morphine daily according to assessed pain needs[168-170].

Atypical antipsychotics
Quality of research studies: 3/4 strong, 1/4 moderate
Outcomes of studies: 2/4 limited evidence, 2/4 negative

Two very large mixed-setting, strong quality studies of people with probable AD compared different fixed and flexible doses of brexpiprazole with placebo over 12 weeks. Brexpiprazole outperformed placebo at higher doses only, with a significantly greater decrease in CMAI scores at 12 weeks with very small to small effect[171]. Pimavanserin decreased agitation in post hoc analysis of RACS residents with moderate to severe AD and severe agitation, and in those whose psychosis responded to pimavanserin in a very large study of strong quality[172]. One large RACS study demonstrated moderate evidence of no significant change in CMAI scores for extended or immediate release quetiapine after 6 weeks, in people with AD[173]. Antipsychotic medications are generally not recommended for use in people with BPSD due to the risk of significant adverse events.

Studies published prior to 2012 provided some evidence for olanzapine[174] and risperidone[175] as well as limited evidence for quetiapine[176, 177] in reducing agitation. While individual studies showed no benefit for memantine[178, 179], very large post hoc analyses reported benefit for the combination of agitation/aggression and delusions[180, 181]. No further studies replicating these findings were found.

Antidepressants
Quality of research studies: 2/2 moderate
Outcomes of studies: 1/2 limited evidence, 1/2 negative

Primary and secondary analyses from a very large community, multicentre RCT of moderate quality provided limited evidence for citalopram in people with probable AD after 9 weeks. Cardiac adverse effects and cognition worsened with citalopram[182, 183]. A significant response was also found for the placebo group[184]. A large study published prior to 2012 provided evidence for

decreased agitation with fewer adverse effects for citalopram when compared with risperidone after 12 weeks[185].

THC/cannibinoids
Quality of research studies: 2/4 strong, 2/4 moderate
Outcomes of studies: 1/4 positive, 1/4 possibly positive (no control group), 2/4 negative

A medium RCT provided strong evidence of significantly decreased CMAI scores for nabilone at 14 weeks compared with placebo in people with AD attending a psychogeriatric clinic and/or living in RACS[186]. A medium open-label chart review of dronabinol add-on to psychoactive medication in neuropsychiatric inpatients with severe dementia provided moderate evidence of possible decreased total PAS agitation scores after seven days, when compared with baseline[187].

Two medium multicentre RCTs provided strong and moderate evidence of no efficacy compared to placebo for oral THC in people with mild to moderate AD, VaD or mixed dementia in the community or RACS[188, 189]. By comparison, a medium community study of high and low doses showed moderate evidence that THC *increased* total CMAI scores in people with mild to moderate dementia[188].

Other biological/pharmacological
Quality of research study: 1/1 strong
Outcome of study: 1/1 some evidence

A very large multicentre RCT of clinic outpatients and RACS residents with AD reported a significant decrease in NPI agitation/aggression subscale change scores for dextromethorphan-quinidine at 10 weeks compared with baseline, and significantly greater benefit for dextromethorphan-quinidine when compared with placebo[190].

Brain stimulation therapies
Quality of research studies: 4/4 moderate
Outcomes of studies: 2/4 positive, 2/4 negative

A medium trial of consistent daytime light therapy, tailored to maximally affect the circadian system, reported a medium effect on agitation at 4 weeks. High circadian stimulus periods outperformed lower-level placebo lighting in RACS and assisted-living residents[191]. Similarly, a medium RACS study of time-limited exposure to therapeutic bright light units reported a large positive effect i.e. greater decrease in CMAI-frequency and CMAI-disruptiveness and BARS agitation scores, for bright light exposure at 8 weeks compared with placebo low level light exposure[192, 193].

A large RACS study compared fluorescent warm white and cold white ceiling lights with warm-white conventional lighting over 8 weeks during Autumn/Winter months. No effect on agitation, of one type of lighting over another, was reported in people with severe dementia[194]. A medium RCT conducted prior to 2012 also reported no significant decrease in agitation for bright light therapy[195]. See *Appendix 3* for interventions reported above.

Summary

While biological or pharmacological interventions are never a substitute for good quality, person-centred care, the individual risk/benefit ratio of these agents must be considered. Some positive outcomes were reported for rivastigmine and galantamine. Studies of tailored light therapy and bright light exposure also indicated benefit. Although pain management should always be considered as a first line treatment for BPSD, only one trial of analgesic medication is included indicating some evidence of improvement. In the antidepressant category, limited evidence was reported for citalopram and adverse effects are a concern. Despite some improvement demonstrated for nabilone, dronabinol add-on to psychoactive medication and dextromethorphan-quinidine, the evidence is too limited for recommendations to be made.

Of the atypical antipsychotics trialled, brexpiprazole and risperidone, compared with galantamine, provided limited evidence of benefit. Current guidelines do not recommend the use of atypical

antipsychotics due to safety concerns. Their use has been associated with further cognitive decline and greater risk of somnolence, extrapyramidal symptoms, abnormal gait, oedema, urinary tract infections, incontinence, falls, cerebrovascular adverse events and mortality. See *Module 2, Table 2.3* for side effects associated with antipsychotics. Therapeutic use must be approved by the person's attending physician or specialist and care partners/guardians must be informed prior to initiation.

Although classified as an atypical antipsychotic, pimavanserin is a selective 5-HT receptor subtype 2A inverse agonist and antagonist and, as such, does not induce clinically significant antagonism of adrenergic, dopaminergic, histaminergic, or muscarinic receptors[196, 197]. This mechanism of action is unique and pimavanserin has been associated with lower mortality than atypical antipsychotic use, in community-dwelling people with Parkinson's disease (PD), during the first 180 days of treatment[198]. Pimavanserin is approved for use internationally for psychosis in PD[197, 199] however, it is not PBS listed for BPSD[200] and not routinely available in Australia[201]. As always, treatment decisions should be guided by all available evidence of efficacy balanced with potential adverse effects[199, 202].

Studies of modest quality, that did not meet our criteria for inclusion indicate limited evidence that ECT may provide transient benefits for agitation, unresponsive to other treatments. See *Appendix 3* for interventions. ECT should only be considered in exceptional circumstances with consent and a second consultant opinion[203].

Where biological or pharmacological interventions are prescribed for agitation, their use should be time-limited and reviewed frequently[204]. Additionally, individuals with different types of dementia may respond differently to pharmacological agents. Those with frontotemporal dementia (FTD) do not respond to ChEIs[205, 206]. Those with dementia with Lewy bodies (DLB) may respond to ChEIs and are more vulnerable to the adverse effects of antipsychotics[207-209].

Limitations

Many of the intervention studies reported have methodological and sampling issues which impact on outcomes and/or potentially limit the generalisability of the results. Some studies provided insufficient data to calculate effect sizes, limiting interpretation of extent of the benefit provided by interventions. Additionally, a definitive diagnosis of agitation in dementia can be difficult due to overlapping symptoms of aggression, sundowning and other BPSD[2, 5]. Further, limited evidence is provided of sustainability of effects after interventions cease. Twenty one psychosocial studies conducted follow-up and of these, only two assessed long-term effects at eight and nine months. Four reported any benefit [81, 82, 114, 144]. One biological study conducted short-term, post-intervention follow-up indicating no benefit[169].

Conclusions/Principles of care

In summary, agitation is one of the most common and challenging behaviours in dementia and it is associated with poor health and quality of life outcomes. While intervention studies for addressing agitation are plentiful in the literature, many have limitations regarding quality. A lack of sound evidence should not prevent clinicians from considering strategies on an individual basis[210]. Expert consensus guidelines recommend the use of multidisciplinary, individualised and multifaceted care including psychosocial interventions as a first-line approach and short-term pharmacological interventions only when necessary. Multicomponent interventions provide the best psychosocial evidence for addressing agitation.

Where pharmacological/biological intervention is indicated analgesic medication should be considered. Some evidence was demonstrated for atypical antipsychotics however, these are not recommended due to the associated risks. Although some benefit was found for nabilone, dronabinol, citalopram and dextromethorphan/quinidine, the evidence for these is not strong enough to make recommendations. Rivastigmine, galantamine, memantine or light exposure may warrant a trial. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary symptom[200, 211]. For example,

where the person presents with depression and agitation, treating the depression may also reduce agitation.

Agitation in people with dementia can occur because of many potential antecedents. Use of a person-centred, trauma-informed approach that incorporates the identification and remediation of triggers and/or underlying causes will assist to reduce agitation, potentially increase therapeutic benefit as well as delivery of optimal care and support for the person and those in their support network[31, 32, 77, 212].

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

5. Anxiety

MODULE 5: Anxiety

Key messages

- Anxiety in dementia presents with thoughts of worry, emotions such as fearfulness, physical sensations associated with autonomic hyperactivity and behaviours such as avoidance and restlessness.
- Anxiety is one of the most disabling and commonly occurring psychological symptoms associated with dementia.
- Where anxiety is secondary to another psychological disturbance in dementia the primary problem should be treated.
- Music interventions provide the best psychosocial evidence for anxiety in dementia.
- Reminiscence-based interventions, multicomponent interventions and sensory interventions were also beneficial.
- A multidisciplinary, individualised and multifaceted approach is recommended.
- Evidence of benefit for pharmacological and biological interventions is lacking; refer to Principles of care for guidance.
- Where symptomatic pharmacological agents are prescribed to address anxiety, these should be time limited, closely monitored, reviewed, reduced and/or discontinued when indicated and used with appropriate psychosocial interventions.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

Anxiety summary

What is anxiety and what does it look like in dementia?

Anxiety can be described as an internal state defined by:

- thoughts of worry, anguish, apprehension and/or vigilance
- emotions such as fearfulness, unease or dread
- physical sensations of muscle tension, tremor, fatigue, nausea, hyperventilation/ shortness of breath, headache and/or other pain, insomnia and/or palpitations associated with autonomic hyperactivity
- perceived need to seek help, flee, fight, or freeze
- manifested actions such as hand wringing, pacing or other repetitious activities.

Anxiety in dementia can become exacerbated to the point of phobias and panic attacks.

Differential diagnosis

The presentation of anxiety is not always typical in those with dementia and medical comorbidities. Differential diagnosis can be confounded by overlapping symptoms of anxiety and depression, agitation, and aggression. The presence of anxiety can be difficult to establish when the person with dementia has difficulty expressing themselves, due to impaired language.

Measuring anxiety

Scales most commonly employed to measure anxiety in dementia include the Rating Anxiety in Dementia (**RAID**) scale, the anxiety subscale of the Neuropsychiatric Inventory (**NPI**) and the NPI-Clinician (**NPI-C**), the anxieties and phobias subscale of the Behavioral Pathology in Alzheimer's Disease Scale (**BEHAVE-AD**), the Hamilton Anxiety Rating Scale (**HAM-A**) and the Geriatric Anxiety Inventory (**GAI**).

Prevalence of anxiety

- Anxiety is one of the most commonly occurring psychological symptoms associated with dementia, reported as a symptom in 13% to 67% of people with dementia and as a disorder in 5% to 31%.
- Higher rates of anxiety are associated with greater cognitive impairment in older people living in the community and associated with better cognition in aged care settings.
- The incidence of clinically relevant anxiety is higher in those with Alzheimer's disease (AD) than in those with vascular dementia (VaD) and other dementia types.
- Anxiety is also commonly reported in frontotemporal dementia (FTD) and in people with younger-onset dementia.

Effects of anxiety

- Anxiety is linked to earlier residential care placement, other changed behaviours and psychological symptoms, overestimation of dementia severity and impaired functioning as well as poor quality of life.
- Excess anxiety may lead to the person with dementia 'shadowing' helpers, seeking reassurance as well as constantly searching for carers, companionship and/or assistance.
- Anxiety can contribute to a higher carer burden due to increased dependence.

Addressing anxiety

Individual antecedents for anxiety, where identifiable, should be considered before addressing symptoms. Where the antecedents and frustrations for the individual with dementia can be identified, minimised and/or avoided, anxiety may be reduced or prevented. Keeping the environment uncomplicated, maintaining structure and routine, reducing the need to make decisions, avoiding overstimulation, providing opportunities to succeed and reinforcing retained skills may help to support the person to experience fewer and less severe symptoms.

Psychosocial and environmental interventions

- Psychosocial intervention trials were conducted in residential aged care, community and hospital settings.
- The cognitive rehabilitation/stimulation interventions group incorporated the greatest number of studies. This was followed by therapeutic recreation, and music interventions.
- Music therapy and reminiscence-based interventions provided the best evidence for a psychosocial approach to anxiety in dementia for people living in residential aged care settings. Interventions included listening/playing music, active music interventions, individual autobiographical sessions and using a reminiscence-based personalised computer app.
- Therapeutic recreation and multicomponent interventions provided the best evidence for addressing anxiety in people with dementia receiving services in hospital and community settings. These included board games, small group choral singing, painting and a multicomponent intervention combining socialisation, cognitive stimulation and physical activity.

Pharmacological and biological interventions

- Evidence for pharmacological/biological intervention studies is lacking and no studies met our criteria for inclusion here.
- Refer to Principles of care for guidance.
- Where symptomatic, pharmacological agents are prescribed for anxiety, these should be time limited, closely monitored, reviewed, reduced and/or discontinued when indicated and always prescribed in combination with appropriate psychosocial interventions.

Limitations

There is limited sound research to guide clinicians and carers in addressing anxiety in people with dementia. Diagnosis of anxiety in dementia can also be difficult due to underlying symptoms of depression, agitation and/or aggression. Few trials investigated the long-term effects of reported interventions with only nine studies conducting post-intervention follow-up assessments.

Conclusions

- Recognised expert guidelines are limited for addressing anxiety in dementia.
- Music therapy, reminiscence, therapeutic recreation and multicomponent interventions provide the best psychosocial evidence.
- Environmental factors may also have a part in reducing anxiety symptoms.
- Evidence of benefit for pharmacological and biological interventions to address anxiety is lacking.
- A multifaceted, person-centred and trauma-informed approach to addressing anxiety in dementia is recommended.

What is anxiety and what does it look like in dementia?

Anxiety in the person with dementia may present with facial expressions of worry, distress or fear, complaints of somatic symptoms, agitation, irritability, hoarding, restlessness and/or requests for reassurance and assistance, often related to forgotten information[1, 2]. Irritability and restlessness have been reported as the most frequent symptoms[3]. Anxiety in dementia has been described as principally an internal state defined by:

- thoughts of worry, anguish, apprehension and/or vigilance
- emotions such as fearfulness, unease or dread
- physical sensations of muscle tension, tremor, fatigue, nausea, hyperventilation/shortness of breath, complaints of headache or other body aches and pain[4], insomnia and/or palpitations associated with autonomic hyperactivity
- motivation/perceived need to seek help, flee, fight, or freeze in response to above cognitive, emotional, and physical sensations
- actions such as hand wringing, restlessness, pacing, repetitious activity or repeated questioning[5]
- seeking family/pets/close others, searching for personal/important possessions, hoarding, avoidance, exit seeking, refusal to mobilise, refusal of care, defensive aggression.

The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11) includes Anxiety symptoms in dementia under a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" under Neurocognitive disorders, Dementia. Anxiety is described as "clinically significant symptoms of anxiety or worry"[6]. Longitudinal studies with greater than 10 years follow-up report single episode and relapsing anxiety[7] and substantial within-individual variation over time[8].

Ballard and colleagues[9] propose three main categories of anxiety in dementia: anxiety related to depression, anxiety in the context of psychosis and anxiety in interpersonal situations. Anxiety can become exacerbated to the point of phobias and panic attacks[10, 11] and frequently presents comorbidly with depression[12, 13]. Anxiety is at times used interchangeably with agitation when agitation presents as the motor manifestation of anxiety[5]. Although agitation can be a manifestation of anxiety that the person with dementia can no longer express verbally[14], anxiety and agitation may be distinct constructs in early stages of disease progression[15].

PRESENTATION

Quang is 80 years old. He migrated to Australia after the Vietnam war under the Family Reunion Scheme and now lives with three generations of his family. When he moved in with the family 6 months ago, they noticed that Quang had some cognitive difficulties. The longer he stayed with them, the more his daughter became aware of his functional deficits. This ultimately led to the local GP making a diagnosis of Alzheimer's dementia and starting Quang on a cholinesterase inhibitor.

Extended family members live in the same street and visit socially but they do not provide care. Quang does not speak, read or write English. Quang's daughter is his primary carer and over past months she has become increasingly concerned about her father's obvious anxiety. Quang reportedly has strong spiritual beliefs but since he has become restless and disruptive during church services, he no longer attends with the family.

A culturally specific in-home community service has recently been cancelled and the family has largely become isolated from the Vietnamese community. The granddaughter reports that many of those in the community lack an understanding of dementia and/or BPSD and the family is embarrassed and concerned that others will think their father is 'crazy'.

Causes of anxiety in dementia

People living with and without dementia experience anxiety stemming from similar diverse causes. Personal and medical history, individual and shared trauma, and both ongoing and situational social,

relationship and environmental factors can all be antecedents to anxiety symptoms. People living with dementia can also experience anxiety associated with loss of independence, cognitive impairment, and the perceptions of others.

Higher rates of anxiety in those with dementia have been associated with unmet needs[16], pain[17-19], mental health[20], and social and psychological needs, including lack of company and daytime activities[21, 22] as well as staff competence to provide care[13, 23]. Anxiety can occur in response to the person with dementia's reduced capacity to make sense of and efficiently navigate their environment[24].

A person with dementia may experience an exaggerated anxiety response around changes to a familiar routine or environment, separation from their primary carer, being rushed, overstimulation and/or fatigue. People with lived experience of dementia report that concern around making errors, failing at simple tasks, forgetting information, not recognising others, having difficulty participating in conversation and/or being able to find the toilet can also trigger significant anxiety[25]. Anxiety in the early stages of dementia can arise from the diagnosis itself and subsequent fears for the future, particularly for people with younger-onset dementia[26].

Anxiety in AD has been associated with a history of chronic disease[27] and physical ill-health[28]. Thus, multiple factors influence whether, how and when a person experiences and expresses anxiety. The person's age at onset, level of awareness, disease progression, residential status, environmental context and social relationships or inability to navigate these, and the person's physical health may individually and/or collectively contribute to anxiety. People in the early stages of dementia may be better able to express their anxiety verbally[29] and/or those in later stages may be more likely to express their anxiety nonverbally[26].

Differential diagnosis

Presentation of anxiety is not always typical in those with dementia and medical comorbidities and presentation may vary according to dementia type. The presence of anxiety can also be difficult to establish when language is impaired[30]. Guidance for assessment and addressing anxiety in dementia is generally lacking[31]. Differential diagnosis can be confounded by overlapping symptoms of anxiety and depression, agitation, and reactive aggression. Symptoms of anxiety such as restlessness, fatigue and difficulty concentrating can also occur in dementia without anxiety, further confounding diagnosis. Validated diagnostic criteria for anxiety in Alzheimer's disease (AD) include restlessness, irritability, muscle tension, fears and respiratory symptoms in the context of excessive anxiety and worry[2, 32].

Measuring anxiety

There are few measures of anxiety developed specifically for older adults, and fewer for people with cognitive impairment and dementia[33-35]. Most anxiety scales were developed in English language. The RAID[36, 37], GAI[38], Geriatric Anxiety Scale (GAS)[33], Penn State Worry Questionnaire - abbreviated (PSWQ-A)[39], Hospital Anxiety and Depression Scale (HADS)[40], State-Trait Anxiety Inventory (STAI)[33, 41] and the Neuropsychiatric Inventory (NPI)[42] have been adapted and validated in multiple languages.

The Mayi Kuwayu modified Kessler scale (MK-K5) is reported as an acceptable and valid screening tool for depression and anxiety in Aboriginal and Torres Strait Islander populations[43]. There is no recommended scale for measuring anxiety in older Torres Strait Islander populations[44]. Anxiety scales tailored/validated for specific diseases including dementia with Lewy bodies (DLB) are also lacking[31]. The following scales have been widely used to assess anxiety in people living with dementia:

- The Rating Anxiety in Dementia scale (**RAID**) rates symptoms of anxiety according to information from all available sources. It includes four subscales: worry, apprehension and vigilance, motor tension and autonomic hyperactivity[11]. The RAID has been validated with people with dementia in acute, inpatient and community settings, and has sound psychometric properties with high sensitivity but variable specificity in different populations[34, 35, 45].

- The 20-item Geriatric Anxiety Inventory (**GAI**) and 5-item Geriatric Anxiety Inventory – short form (**GAI-SF**) are self-report or clinician-administered scales that measure dimensional anxiety in older people. They have sound psychometric properties in psychogeriatric populations[33-35, 46].
- The 16-item Penn State Worry Questionnaire (**PSWQ**)[47] and 8-item abbreviated version (**PSWQ-A**)[39] assess pathological worry, show adequate psychometric properties in older adults without a diagnosis of dementia[33] and may be useful for screening[48].
- The Mayi Kuwayu modified Kessler scale (**MK-K5**) is reported to have good psychometric properties and to be an acceptable and valid screening tool to identify risk for anxiety and depression in Aboriginal and Torres Strait Islander populations, with clinical utility for referral to assessment[43]. Psychometric properties and clinical utility for people experiencing cognitive impairment is unknown. The tool should be used only by health care professionals who have been trained to implement and deliver screening and care in culturally appropriate ways[43].
- The 7-item self-report Hospital Anxiety and Depression Scale (**HADS–A**) subscale[40] measures symptom presence/severity and is used commonly in residential aged care services (RACS) research[34]. People with cognitive impairment may require assistance to complete items and interpretation in people with dementia may be difficult. Psychometric properties are variable[49].
- The 40-item self-report State-Trait Anxiety Inventory (**STAI**) measures transient state anxiety and lasting trait anxiety. Psychometric properties decrease for older adults, its length may be burdensome and outcomes difficult to interpret in people with dementia[33].
- The 30-item Geriatric Anxiety Scale (**GAS**) and 10-item Geriatric Anxiety Scale (**GAS10**) are self-report screening and assessment tools designed with community-dwelling adults. Psychometric properties are good in nonclinical populations. Performance is decreased in people with cognitive impairment. Subscale scores should be interpreted with caution as a unidimensional structure has been reported[33].
- The anxiety subscale of the Neuropsychiatric Inventory (**NPI**) is completed during an interview with the carer[50]. The frequency and severity of the person with dementia's anxiety and the carer's subsequent distress are rated. The reliability and validity of the NPI, overall is well established however, individual NPI symptom domains can be more clinically relevant than the total NPI score[42].
- The revised NPI-Clinician (**NPI-C**) expands the anxiety subscale of the NPI to include additional items around feeling threatened, facial expression, worries over health, tearfulness, fear of abandonment, repeated questioning, distractibility and overconcern[51].
- The Behavioral Pathology in Alzheimer's Disease Scale (**BEHAVE-AD**) anxieties and phobias subscale includes four items: anxiety regarding upcoming events, other anxieties, fear of being left alone and other phobias[52, 53].
- The 14 item Hamilton Anxiety Rating Scale (**HAM-A** or **HARS**) classifies anxiety as mild, mild to moderate or moderate to severe[54]. The HARS does not capture worry and shows poor discriminant validity. The Hamilton Anxiety Rating Scale-revised (**HARS-R**) and **HARS-R-II** offer modestly better criterion-related validity[55].
- The 21-item short version of the Depression Anxiety Stress Scales (**DASS-21**) presents a three-factor structure[56]. The DASS-21 has been used in people with AD[57]. The scale is reported as reliable and valid in people over 60 without dementia[58].
- The 21-item self-report Beck Anxiety Inventory (**BAI**) assesses cognitive and somatic symptoms[59]. It has sound psychometric properties for use with older adults. The BAI may be unresponsive to change in clinical populations and shows limited discriminant validity[33].

Prevalence of anxiety

Anxiety disorders are the most common mental illness reported in the past year by the general population in Australia and globally, with higher prevalence in females[60-62]. Rates increased during the COVID-19 pandemic[63, 64]. The prevalence of anxiety amongst Indigenous peoples in Australia, aged older than 45, is reportedly 18.3-21%[65] with higher rates postulated[66].

It is unclear whether people living with dementia experience anxiety at higher rates than[34, 60, 61], or comparable to, people of similar age without dementia[67-71]. Anxiety is associated with greater cognitive impairment in older people living in the community and associated with better cognition in aged care settings[16]. In residential aged care, 35% of people without dementia, and 23% of people with dementia, are reported to have anxiety[67], with greater prevalence in females[72].

ASSESSMENT

To reduce Quang's presenting symptoms of anxiety, potentially contributing factors must be identified:

- *Illness/infection/discomfort/pain*
- *Treat or exclude underlying depression where indicated*
- *Medication review: interactions, dosage, adverse effects, recently prescribed*
- *Lack of attention to culturally-relevant needs*
- *Unfamiliar or altered physical environment*
- *Unrealistic expectations of others causing Quang to try to overextend his capabilities*

Assessing the situation:

- *Encourage Quang to report his concerns as far as he is able.*
- *Directly observe for any environmental aspects or triggers that may contribute to the symptoms.*
- *Ask Quang's family to keep a record of times (when? how long?) and events (if any) leading up to episodes of anxiety.*
- *With the family's consent, communicate with staff from the recently ceased community service as to any incidents that provoked Quang's anxiety.*
- *Consult Quang's family with regard to his life history and for further information around potentially relevant factors such as war experiences or historical trauma.*
- *Consult close family members to identify possible triggers for the symptoms.*
- *Is trialling pharmacological interventions indicated and/or appropriate for addressing Quang's anxiety?*

Anxiety is one of the most commonly reported psychological symptoms in dementia at 13% to 67% overall[4, 35, 73]. Anxiety is reported as a symptom in an average of 29% (95%CI 23% to 35%) of people with dementia living in the community[74] and as a disorder in 5% to 31%[16, 34]. Type of dementia may influence anxiety prevalence[74] although, consensus is lacking[73]. Higher risk of anxiety is reported in AD than vascular dementia (VaD)[74, 75], DLB[7], mixed dementia and unspecified dementia[75]. Anxiety is also common in frontotemporal dementia (FTD)[76].

Higher anxiety prevalence and severity is reported for people with younger-onset AD compared with later-onset AD[77-79]. Some studies report that such differences persist[78]; others that differences attenuate over time[77]. Awareness of one's declining cognition and functioning may lead to anxiety[25, 80, 81]. Anxiety prevalence reportedly increases as cognitive function decreases[74], and severity increases with increasing cognitive impairment in VaD[82]. Consensus is lacking however[8], as others report that anxiety tends to increase as cognition declines in the early stages of dementia and decreases in the later stages[26, 73].

Effects of anxiety

Anxiety in dementia is linked to higher carer burden due to increased dependence, earlier admission to residential care, other changed behaviours and psychological symptoms, overestimation of dementia severity, impaired social and activities of daily living (ADL) functioning, sleep disturbance as well as poorer quality of life[3, 12, 28, 83-85]. Excess anxiety may lead to the person with dementia 'shadowing' helpers, seeking reassurance as well as constantly searching for carers, companionship and/or assistance. This can result in wandering as well as increased carer stress and feelings of guilt[86]. According to the Progressively Lowered Stress Threshold (PLST) model of care[87],

symptoms of anxiety in dementia indicate escalation to more intense and troubling behaviours which further impede function and comfort.

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded 28 psychosocial or environmental intervention studies with outcomes relevant to anxiety in dementia. Of these, ten studies reported, at least, some evidence of benefit. No pharmacological/biological intervention studies met our rating criteria of moderate or strong quality for inclusion here, however only one negative study of modest quality was excluded. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained but not included in the summary numbers.

Addressing anxiety

First, individual triggers for anxiety, where identifiable, should be considered and remedied where possible. Anxiety in people with dementia can result from many antecedents. See *Module 1, Table 1.2* for a list of potential contributing factors. Left untreated, anxiety can escalate to the point of overwhelming the person with dementia until a catastrophic reaction ensues. Where the triggers and frustrations for the individual can be identified, minimised and/or avoided, anxiety may be reduced or prevented. *Appendix 4* provides suggested questions to facilitate comprehensive assessment.

Keeping the environment uncomplicated, maintaining structure and routine, reducing the need to make decisions, avoiding overstimulation, providing opportunities to succeed and reinforcing retained skills may help to support the individual with dementia to experience fewer and less severe anxiety symptoms. Likewise, redirection and reassurance may disrupt the emotional cycles perpetuating the anxiety. Strategies

STRATEGIES/OUTCOMES

- *It is important to establish who is able to provide information on Quang's behalf and who is the main decision maker within the family. Communication should be directed to the main person with the understanding that the family must be included in all discussions and/or decisions.*
- *The family was hesitant to provide information due to language barriers and fear of how the information will be used. They are concerned that they may be perceived as being unable to cope with their father's care needs which could result in him being removed from the family home. The family was assured that services are available to support them to continue to care for Quang at home.*
- *Quang's granddaughter is the only family member who speaks English. Access to an interpreter who speaks the same dialect as Quang and his family was offered, where appropriate.*
- *Quang's family has been reluctant to accept formal services due to community expectations that they should care for their aged relatives. They do not want to be judged by members of the local community as unable to care for Quang.*
- *Information about dementia and BPSD was provided in Vietnamese. The information in written or electronic format could be shared with the extended family and others.*
- *The family was linked with a Vietnamese agency that was able to provide further culturally-safe support and information.*
- *The family has been reluctant to continue with the cholinesterase inhibitor as they are unfamiliar with western medical practices. Quang's doctor was informed that he was no longer taking the prescribed medication.*
- *The family was provided with additional information in Vietnamese around the purpose and potential benefits of the cholinesterase inhibitor. Quang's daughter also discussed possible benefits of traditional Asian medicines with the GP.*
- *The family was able to suggest activities that could be encouraged, such as those that Quang previously found pleasurable, engaging, comforting and/or related to his spiritual beliefs and his favourite music.*
- *Quang's family was provided with strategies to avoid triggering his anxiety and to help ease his symptoms during acute periods of escalation.*
- *When extended family members were made aware of the situation, they were willing to assist. Quang was able to resume attendance for part of the church service with others helping to supervise him.*
- *The possibility of Quang attending a CALD day respite centre with others from a Vietnamese background was investigated.*

for repeated questioning include recognising the concerns that are prompting questions (for example, fear of abandonment), providing reassurance/comfort in a culturally appropriate manner such as hand holding or neck massage and initiating an enjoyable activity[10].

Psychosocial and environmental interventions

Trials of psychosocial and environmental interventions were mainly conducted in residential and community care settings with one study undertaken in a hospital.

Music
Quality of research studies: 1/5 strong, 4/5 moderate
Outcomes of studies: 1/5 positive, 3/5 mixed/limited/some evidence, 1/5 negative

All music studies were conducted in residential care. One trial showed moderate evidence for reduced anxiety after 6 weeks of twice-weekly small group, preferred music therapy. Music therapist-led sessions included games, moving to music and rhythmic accompaniment[88]. A study of male veterans reported decreased HAM-A anxiety scores for weekly music therapy with percussion instruments when compared with rest and reading over 12 weeks[89]. Another trial reported no benefit over usual care for music therapy with percussion instruments over 6 weeks[90]. These three medium studies included people with mild to moderate dementia.

A very large study of people with moderate dementia provided moderate evidence that both music with movement using props and instruments, and listening to preferred music, decreased RAID anxiety scores after 6 weeks and that chatting socially while listening to music had no significant effect. Differences between groups were no longer significant 6 weeks after interventions ceased[91]. A smaller study provided strong quality evidence of a positive outcome for an intervention delivered by psychologists. Small group receptive listening and drum playing to recorded music for residents with severe dementia improved STAI anxiety scores after 4 weeks, whereas group cake-making and tasting sessions showed no effect[92]. One study undertaken prior to 2012 provided moderate evidence of benefit for individual, receptive music therapy[93] however, a good quality study of live music programming found no benefit for anxiety[94].

Therapeutic recreation
Quality of research studies: 1/5 strong, 4/5 moderate
Outcomes of studies: 2/5 positive, 3/5 negative

A large hospital-based study provided moderate evidence of a small effect with decreased HADS anxiety scores after patients played a strategy board game GO (also known as Igo, Baduk, and Weiqi) for either 1 or 2 hours each day over 6 months. Anxiety scores decreased regardless of session length and decreases were significant compared with usual care[95].

Studies of creative and performing arts interventions showed mixed results. A very large RCT provided moderate evidence of significant decrease in anxious mood for humour therapy delivered by trained performers (ElderClowns) with trained residential care staff (LaughterBosses) in 9-12 sessions across 13 weeks however, the intervention provided no benefit over usual care[96]. A medium community-based study compared 2-hour choral singing sessions delivered by a professional conductor with 2-hour painting sessions. Decreased STAI anxiety scores were reported for both groups after 12 weeks[97].

A medium multicentre trial of tri-weekly individual, non-facilitated baby doll sessions compared with usual care over three weeks provided strong evidence of no change in anxiety scores and no benefit over usual care for female residential care residents[98]. Similarly, a large RACS trial that compared tri-weekly one-to-one individualised psychosocial sessions matched to residents' interests and ability, standardised one-to-one activities and usual care for people with mild to severe dementia provided moderate evidence of no difference in anxiety between groups after 3 weeks[99].

Cognitive rehabilitation/stimulation
Quality of research studies: 2/6 strong, 4/6 moderate
Outcomes of studies: 6/6 negative

A medium study reported moderate evidence of no effect on care home residents after five weeks of group mindfulness sessions[100]. Two medium community-based studies provided strong to moderate quality evidence of no benefit for Cognitive Behavioural Therapy (CBT). Weekly in-home sessions over 3 months[101] and manual-based CBT delivered by clinical psychologists over 15 weeks[102] were trialled.

A moderate quality community-based study comparing weekly goal-oriented cognitive rehabilitation and/or restorative approaches with therapist-led breathing/muscle relaxation sessions demonstrated no benefit over usual care[103]. Similarly, one very large strong quality community-based RCT of individual goal-oriented cognitive rehabilitation showed no benefit over usual care after 9 months[104] and one medium moderate quality study of occupational therapist (OT)-delivered cognitive stimulation reported no decrease in anxiety scores after 7 weeks[105].

Reminiscence
Quality of research studies: 2/2 moderate
Outcomes of studies: 2/2 positive

Reminiscence-based interventions in RACS provided moderate evidence of positive results with small effect sizes. Weekly individual, autobiographical reminiscence sessions, with a nurse-therapist, delivered over 5 weeks decreased GAI anxiety scores more effectively than usual care[106]. Greater benefit in decreased RAID anxiety scores was demonstrated after 4 weeks of using the *Memory Box* app, that incorporated easily accessible personalised multimedia content selected by family, when compared with social engagement to discuss current events[107].

Physical activity
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A medium study involving people with AD, DLB, Parkinson's disease dementia (PDD) or mixed dementia provided moderate quality evidence of no benefit for anxiety after 12 weeks of twice-weekly sessions of chair yoga, customised participatory music, or gentle chair-based exercise[108].

Animal-assisted
Quality of research studies: 1/2 strong, 1/2 moderate
Outcomes of studies: 2/2 negative

Two RACS trials of group and individual sessions with the companion robot *Paro* for people with moderate to severe dementia provided moderate and strong evidence, respectively, of no benefit for anxiety over interactive reading or usual care[109, 110].

Sensory therapy
Quality of research study: 1/1 moderate
Outcome of study: 1/1 some evidence

One small, RACS study of twice-weekly use of a *Snoezelen*® multisensory room in residents with severe dementia provided moderate evidence of decreased anxiety symptoms for *Snoezelen*® over individualised music after 8 weeks[111].

Touch therapy
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A large RACS study provided moderate evidence that 20-minute ear acupressure sessions 5 times weekly for residents with moderate to severe dementia provided no benefit for anxiety over relaxing lower limb and back massage delivered by a physiotherapist and no benefit over usual care after 3 months[112].

Education/training
Quality of research studies: 2/3 strong, 1/3 moderate
Outcomes of studies: 3/3 negative

A medium community-based trial provided strong evidence that weekly multicomponent training sessions for carers, led by psychologists and focused on sleep disturbances (*DREAMS-START*), provided no benefit for anxiety and did not outperform usual care[113]. A very large cluster RCT of OT-delivered training for residential care staff to improve activity provision provided strong evidence of no benefit compared with usual care at 4- and 12 weeks post-intervention[114]. A medium RACS study provided moderate evidence that manual-based dementia workshops and individual supervision sessions for direct care staff (*STAR*) over 8 weeks had no effect on anxiety symptoms in people with moderate to severe dementia[115].

Multicomponent
Quality of research studies: 1/2 strong, 1/2 moderate
Outcomes of studies: 1/2 some evidence, 1/2 negative

A small community-based study of clinical outpatients with mild to moderate AD provided strong evidence of a large effect for an intervention involving physical activity, cognitive stimulation and socialisation. Anxiety scores decreased after 3 months of 4-hour sessions 5 times weekly[116]. By contrast, a medium RACS trial with residents with moderate to severe dementia provided moderate evidence of no change in anxiety and no benefit over usual care for a multisensory stimulation, reminiscence and physical activity group intervention[117].

Summary

Cognitive rehabilitation/stimulation incorporated the greatest number of studies however, all reported negative outcomes. Small group psychosocial interventions based on passive and active music therapy, and individualised reminiscence-based activities and multisensory room sessions, helped to reduce anxiety symptoms in people living in residential care. Daily participation in therapeutic recreation in the form of GO-game with other patients and staff decreased anxiety in hospitalised people with dementia. People attending memory clinics benefitted from therapeutic recreation in weekly choral singing groups and painting groups, and a daily intensive, small group multicomponent intervention. All activities reporting beneficial outcomes were delivered by therapists or staff trained in the interventions. Nearly all were structured and had strong social components.

Although presented in separate categories, many interventions included shared components. For example, music interventions involved elements of reminiscence and vice versa, painting and board game interventions involved learning and executing new skills as well as expressing creativity. Several of the interventions involved synchronised activity, and all could be considered pleasant diversional therapies. See *Appendix 2* for interventions reported above.

Biological interventions

Research evidence for biological interventions to guide clinicians and care partners in supporting those who present with anxiety is lacking. Expert consensus guidelines recommend against the use of pharmacological agents[1, 118]. Where symptomatic pharmacological treatment is prescribed for anxiety, it's use should be time limited, closely monitored, reviewed, reduced and/or discontinued when indicated and prescribed in conjunction with appropriate psychosocial interventions. As always, the potential benefits to the person with dementia must be weighed against the side effects of pharmacological treatments. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[119, 120]. For example, where the person presents with anxiety and depression, treating the depression may also reduce anxiety.

Limitations

There is limited sound research to guide clinicians and carers in addressing anxiety in people with dementia. Rarely is anxiety in dementia trialled as a primary trial outcome. Problems occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. Few trials investigated the long-term effects of the interventions with only nine studies conducting post-intervention follow-up assessments ranging

from two weeks to four months. Of these, only three reported any benefit at follow-up[91, 92, 111]. Predictably, those experiencing the disabling features of anxiety and dementia may be unwilling to participate in research. Diagnosing anxiety in dementia can also be difficult due to underlying symptoms of depression and/or agitation.

Conclusions/Principles of care

In summary, anxiety is common in dementia with significant and disabling consequences. Recognised expert guidelines for addressing anxiety in dementia are limited. Music, therapeutic recreation, and reminiscence provide the best evidence for psychosocial approaches to anxiety symptoms. Environmental factors may also have a part in reducing anxiety symptoms. A multidisciplinary, individualised and multifaceted approach is recommended.

Recent research by Burley and colleagues[25, 81] considered perspectives on anxiety in dementia from the person with dementia and family carers. Engagement in meaningful activities, person-centred care and relationships were among the most significant areas of concern reported. The majority saw identifying and minimising precipitants to the anxiety as the first step in addressing symptoms and pharmacological options as a last resort.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

6. Apathy

MODULE 6: Apathy

Key messages

- Apathy is one of the most challenging and prevalent psychological symptoms of dementia.
- It is associated with increased disability and carer frustration as well as reduced quality of life.
- Apathy is significantly related to reduced independence in activities of daily living (ADL) beyond dementia severity, survival duration after admission to residential aged care services (RACS) and poor rehabilitation outcomes.
- Psychosocial interventions have the potential to reduce apathy.
- Tailored interventions, provided individually or in a group, have the best available evidence for effectiveness in dementia.
- Many interventions are heterogeneous; these include 'brain-activating rehabilitation', 'biographically oriented mobilisation', multisensory stimulation environments, one-to-one activities, reminiscence with videos, active traditional/nostalgic music sessions, and multicomponent interventions.
- The evidence for the efficacy of pharmacotherapy/biological interventions for apathy in dementia is limited.
- Single trials of donepezil plus choline alphoscerate and of donepezil plus memantine showed some benefit for apathy.
- Moderate evidence of efficacy was found for methylphenidate, and limited evidence for repetitive transcranial magnetic stimulation (rTMS).
- Acknowledging individuality, personal history and previous interests may guide individual strategies for people with apathy.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

Apathy summary

What is apathy and what does it look like in dementia?

- Apathy can describe lack of interest, behavioural inaction and lack of emotion or affect.
- The apathy spectrum includes reduced initiative, interest, motivation, spontaneity, affection, energy, enthusiasm, emotion and persistence as well as blunted affect.
- Synonyms for apathy include passivity, abulia and amotivation.
- Apathy in dementia is frequently accompanied by one or more other neuropsychiatric symptoms.

Lack of motivation is evidenced by diminished goal-directed behaviour, goal-directed cognition and emotion, relative to previous functioning levels and not attributable to intellectual impairment, emotional distress or diminished consciousness. Emotional distress is typically absent. Symptoms should cause clinically significant functional impairment not attributable to physical disabilities, motor disabilities or direct physiological effects of a substance.

Causes of apathy

Apathy is a major clinical feature of dementia with subcortical and frontal pathology such as dementia with Lewy bodies (DLB/LBD), Huntington's disease (HD), behavioural variant frontotemporal dementia (bvFTD) and vascular dementia (VaD). Apathy in Alzheimer's disease (AD) is also related to older age and depression. Apathy can occur in some psychiatric disorders and as a side effect of drugs.

Differential diagnosis

Apathy in dementia should be differentiated from depression and from medication effects. Symptoms of apathy should not be attributable to psychiatric illnesses, intellectual disability, physical disabilities, motor disabilities, change in level of consciousness or direct physiological effects of a substance.

Measuring apathy

Scales designed to specifically measure apathy include the Apathy Evaluation Scale (**AES**), the Apathy Inventory (IA/**AI**) and the Lille Apathy Rating Scale (**LARS**). The Neuropsychiatric Inventory (**NPI**) and the Neuropsychiatric Inventory-Clinician (**NPI-C**) include an apathy subscale.

Prevalence of apathy

- Apathy occurs in up to 89% of people with dementia.
- Prevalence rates are higher in progressive supranuclear palsy, FTD and AD.
- Apathy typically appears early in dementia and increases with dementia severity. It may fluctuate but typically persists.

Effects of apathy

- Apathy is associated with increased disability and frustration, worsening functional impairment and poorer quality of life for both those with dementia and carers, with family life and relationships often disrupted.
- Families not recognising an apathetic state may become increasingly resentful as they misperceive the person as lazy and unempathetic.
- Morbidity and mortality may be indirectly related to apathy as residents in long-term care tend to be less noticed by care staff and receive fewer direct care hours.

Addressing apathy

Acknowledging individuality, personal history and previous interests may guide individual

strategies for engaging those with apathy, as well as variations in response. The following indications should be considered:

- Excess disability of the person with dementia
- Potential for improvement in quality of life
- Burden to carers and/or family

Psychosocial and environmental interventions

- Individually-tailored interventions for apathy in dementia can be beneficial. These include 'brain-activating rehabilitation', 'biographically oriented mobilisation', one-to-one activities, reminiscence with videos, and active traditional/nostalgic music sessions.
- Some positive results were reported for music, physical activity, multi-sensory stimulation, reminiscence, cognitive rehabilitation/stimulation, and multicomponent interventions.
- Interventions with higher levels of support and elements of guided reminiscence and social interaction provide the best evidence of efficacy.

Biological interventions

- Cholinesterase inhibitors (ChEIs) have demonstrated small to moderate benefits for apathy; specifically, donepezil in combination with either memantine or choline alphoscerate. There is no clear indication that any one ChEI is superior.
- Good evidence indicates that antidepressants do *not* significantly improve apathy in people with dementia.
- Moderate evidence has demonstrated efficacy for methylphenidate although side effects may be a concern.
- Limited evidence has been demonstrated for repetitive transcranial magnetic stimulation (rTMS).
- No evidence was found for analgesics, traditional medicines, atypical antipsychotics, and other pharmacological/biological treatments.

Limitations

Few strong quality studies are available for addressing apathy in dementia. Research into the treatment of apathy is, particularly, hampered by the difficulties of recruiting and retaining study numbers to ensure sufficient power. Further, there is little to no evidence of sustainability of effects after interventions cease. Many studies treat participants as a homogenous group yet, dementia type and severity likely influence the effectiveness of interventions trialled.

Conclusions/Principles of care

- Psychosocial and environmental interventions are warranted in the treatment of apathy, particularly individually tailored activities.
- Of the pharmacological treatments reviewed, donepezil plus choline alphoscerate and donepezil plus memantine, and methylphenidate demonstrated evidence of maintaining or improving apathy levels.
- Limited evidence has been reported for repetitive transcranial magnetic stimulation (rTMS).
- Antidepressants and antipsychotic medications are not recommended for apathy in dementia.
- A lack of quality research is not necessarily indicative of a lack of efficacy. Stabilisation of apathy, as dementia progresses, may indicate that the intervention is beneficial even without evidence of improvement.

What is apathy and what does it look like in dementia?

Apathy represents a form of executive cognitive dysfunction which affects goal-directed behaviour and overlaps with other psychological and behavioural aspects such as mood, personality and cognitive functioning[1]. The nosology of apathy is blurred[2]. Apathy can describe an internal state of lack of interest or a state of behavioural inaction[3]. Marin[4] defined apathy as a syndrome of primary motivational loss or "loss of motivation not attributable to emotional distress, intellectual impairment or diminished level of consciousness". The apathy spectrum includes reduced initiative, interest, activity, spontaneity, affection, energy, enthusiasm, emotion, empathy, persistence and responsiveness[5]. Definitions for apathy overlap with those for passivity[6], abulia and amotivation.

Apathy is an independent syndrome[7, 8]. Definitions for this construct vary[9] and structured definitions are lacking in disease classification criteria. The International Classification of Diseases (ICD-11) describes apathy in dementia as "clinically significant indifference or lack of interest" in addition to cognitive disturbances[10]. The Diagnostic and Statistical Manual of Mental Diseases (DSM-5-TR) describes apathy as "typically characterised by diminished motivation and reduced goal-directed behaviour, accompanied by decreased emotional responsiveness", and lists apathy as an "other behavioral or psychological disturbance" accompanying neurocognitive disorders, and as a frontotemporal neurocognitive disorder symptom[11]. Apathy is widely recognised by clinicians and carers as a syndrome of decreased initiation and persistence as well as social disengagement and emotional indifference or absence[12].

Causes of apathy

Apathy is a major clinical feature of dementia with subcortical and frontal pathology such as dementia with Lewy bodies (DLB)[13], behavioural variant fronto-temporal dementia (bvFTD)[14], Huntington's disease[15], Binswanger's disease[16] and vascular dementia (VaD)[17-19]. Apathy in Alzheimer's disease (AD) has also been significantly related to older age and depression[3, 14]. However, comparatively higher levels of apathy are seen in people with younger onset dementia than in those with late onset[20, 21]. Impaired awareness is associated with more apathy as well as less anxiety and depressive symptoms in people with early AD[22].

Micro- and macro-structural white matter damage can disrupt structural connectivity, producing motivational deficits in dementia[23]. Neuroimaging studies indicate that several neuropathological foundations of apathy overlap in disorders such as FTD and Parkinson's disease (PD). Further, associated volumetric changes and dysfunction of brain connectivity in AD are unique from other types of dementia[1, 24-27].

Increased risks for developing apathy in dementia include older age[28] and greater severity of cognitive impairment[14, 29]. Apathy occurring in dementia is

PRESENTATION

Francis has always been active and busy. She enjoyed the demands of her extended family and work life. A diagnosis of younger onset dementia six months ago was devastating for Francis and her wife, Anne but they have been doing their best to meet the challenges as they arise. Recently Francis has had to stop work and Anne has reduced her work hours to spend more time at home. Their children are providing additional support when they can.

Although Francis remains physically well, family have noticed that she no longer starts her day with her usual walk. They often find Francis sitting unoccupied, staring into space. Anne is distressed and frustrated when she arrives home from work to find the lunch she prepared for Francis untouched. Small tasks she has left for Francis have also not been started.

Francis continues to respond positively to family visits and outings, although not with the same level of enthusiasm, and she will join one of them on a walk. She enjoys chatting with friends when they call but Francis is often unable to answer the calls in time, as she misplaces her phone. When this happens Francis doesn't return their calls. Friends are feeling uncomfortable and unsure of how best to respond to Francis' situation. Family members are concerned that Francis appears to 'do nothing' whenever she is home alone.

frequently accompanied by one or more neuropsychiatric symptoms[28], which may fluctuate during the course of the disease[1, 5, 30]. The often impoverished physical and social environment of residential care can further inhibit motivation, as can sensory impairments[1]. Helpful or unhelpful factors encountered in their home and community can also influence the person's subjective experience of apathy[31-33].

Differential diagnosis

Diagnostic criteria for apathy have been adapted over time[4, 34-36]. In 2021 the International Society for Central Nervous System (CNS) Clinical Trials Methodology Apathy Work Group proposed an updated multidimensional diagnostic criteria for apathy in the presence of a diagnosed syndrome of cognitive impairment or dementia as defined by ICD or DSM5 criteria[5]. Three apathy subtype domains previously labelled as behaviour, cognition and emotion have been relabelled as diminished initiative, diminished interest, and diminished emotional expression/responsiveness. A minimum of one symptom should be evident from two of the three dimensions most of the time for at least four weeks, and this must represent change relative to previous functioning and usual behaviour. Symptoms should not be attributable to psychiatric illnesses, intellectual disability, physical disabilities, motor disabilities, change in level of consciousness, or direct physiological effects of a substance. Additionally, symptoms must cause clinically significant functional impairment in personal, social, occupational, and/or other important areas[5].

Apathy is related to but distinct from depression and dysphoria[28, 37]. Although the overlap in symptoms can pose difficulties in differentiating the two and apathy is commonly misdiagnosed as depression, individuals with apathy tend to present as compliant or passive whereas those with depression are deliberately avoidant (Steffens et al 2022). Further, unlike depression, apathy is not typically associated with insomnia, impaired attention or feelings of hopelessness, anxiety and/or sadness[37, 38].

The use of antipsychotic/neuroleptic and antidepressant medications, which can induce side effects such as fatigue, lethargy, listlessness and reduced response to stimuli, can initiate, maintain or imitate apathetic behaviours[39]. SSRIs have also been reported to induce an amotivational or apathy syndrome which is reversible when the dose is ceased or reduced[40, 41].

Measuring apathy

The Apathy Evaluation Scale (AES)[42], the Apathy Inventory (IA/AI)[43] and the Lille Apathy Rating Scale (LARS)[44] are specifically designed to rate apathy[45]. Measures that rate apathy in specific neurodegenerative processes, such as the Frontal Systems Behaviour Scale (FrSBe)[46, 47] are also available. Rating scales frequently used in dementia include:

- The **AES** is recommended for the specialist assessment of general apathy symptoms in dementia based on the person's interests, activities and daily routine[30, 42, 45]. The 18-item scale has reasonable to good psychometric

ASSESSMENT

To address Francis' presenting apathy, potentially contributing factors must be identified:

- *Pain/discomfort/illness/infection*
- *Medication interactions, dosage, adverse effects, recently prescribed*
- *Impaired hearing and/or eyesight*
- *Exclude underlying depression, grief reaction to the diagnosis*
- *Lack of stimulation*
- *Unfamiliar/altered/deprived physical environment*
- *Reduced ability to initiate activities for herself*
- *Activities offered no longer of interest/unfamiliar/becoming too difficult*

Assessing the situation:

- *Encourage Francis to express her interests as far as she is able.*
- *Directly observe and share Francis' response to activities offered so successful events can be repeated.*
- *Consider Francis' previous interests for further potentially appropriate activities.*
- *Ask Anne for feedback re Francis' response to activities after the events.*

properties and can differentiate between apathy and depression[48]. Mast et al[49] note superior psychometric properties of the AES-Informant (AES-I) version and recommend the AES-I over clinician (AES-C) and self-report (AES-S) versions. A 10-item form (AES-10) has been validated in residential care[50].

- The **IA** is a very brief clinician-rated scale to assess global apathy in people with MCI[43] based on emotional blunting, lack of initiative, lack of interest and awareness of symptoms. Psychometric properties for these domains are not equally robust[49]. Self-report (IA-S), and informant-report (IA-I) versions are available.
- The **LARS** is a 33-item clinician-rated instrument designed to assess apathy associated with PD[30, 44]. It is valid and reliable for detecting apathy in mild to moderate dementia generally[51] through a structured interview with the person in the presence of the carer[49]. Subscales include emotional blunting and reduced productivity, interest, initiative, concern, social life and motivation as well as extinction of novelty seeking and self-awareness[44].
- The 16-item **DAIR** assesses apathy in dementia through a structured, clinician-rated interview with the person's carer[52]. Psychometric properties are rated as good[49] to excellent[53].
- The apathy subscale of the **NPI** is completed during an interview with the carer, in which they rate the frequency and severity of the person with dementia's symptoms as well as their own subsequent distress[54].
- The NPI-Clinician (**NPI-C**) expanded the apathy/indifference subscale of the NPI to include additional items around reduced participation, interest in new events and emotional expression[55].
- The apathy subscale of the 34-item Alzheimer's Disease and Related Dementia Mood Scale (**AMS**) is completed with the person's care partner or a formal carer[56].
- The **AS/SAS** is a 14-item tool adapted from the AES designed to assess global apathy in PD and has been validated in AD[57]. The scale is clinician-rated using person or informant interview. Reviews do not report favourably on the tool's psychometric properties[49, 58].
- The Frontal Systems Behaviour Scale (**FrSBe**)[47], previously known as the Frontal Lobe Personality Scale (FLOPS)[46] differentiates FTD from AD[59]. The 46-item clinician-rated interview with the person and their carer includes subscales for executive dysfunction, disinhibition, and apathy.
- The Apathy in Institutionalised Persons with Dementia (**APADEM-NH**)[9] is a 26-item carer-rated measure of cognitive inertia, emotional blunting and deficits of thinking and self-generated behaviours.

While specific scales differentiate components of apathy such as motivation, interest and behaviour, omnibus neurobehavioural scales do not. This may be important as different components of apathy appear to become more prominent over time in the older population generally[60, 61] and at different stages in those with dementia[14, 62]. Assessment of apathy in dementia is further complicated by the need to distinguish between diminished behaviour due to loss of motivation and loss of ability secondary to cognitive impairment[28, 37, 62, 63]. Person-centred assessment must further elicit the needs and perspectives of the person experiencing symptoms[49].

Prevalence

The frequency of apathy in neurological disorders ranges from 24% to 89% with a general prevalence of 54%[64]. High prevalence has been reported in progressive supranuclear palsy[65], FTD and AD[66]. Apathy tends to appear early in dementia, increases and persists over time with dementia severity and is associated with other neuropsychiatric symptoms [28, 62, 67]. Apathy may also present a fluctuating course[62] and those with less severe disease and symptoms are

more likely to improve or remit spontaneously[67]. The prevalence of apathy in mild cognitive impairment (MCI) is higher than in non-impaired older adult controls[68] with a range of 10.7% to 44.8%[69]. Apathy in MCI predicts a higher rate of conversion to AD[69-73].

Effects of apathy

Apathy is associated with increased disability and frustration as well as poorer quality of life in people with dementia and carers[31, 32, 74]. The degree of carer distress may be dependent on the premorbid personality of the person with dementia and expectations of the carers and the living situation[75, 76]. For example, family members are more likely to be distressed by seeing a premorbidly active person become apathetic whereas apathy in a residential aged care services (RACS) is easier for staff to tolerate as apathetic residents make few demands. Families not recognising an apathetic state may become resentful as they misperceive the person with dementia as lazy[75], increasing the risk of relationship breakdown[12]. Responses to issues of personal safety and risk may likewise be impaired.

The potential for apathy to prevent those affected from seeking assistance, to be misdiagnosed with depression or to be noncompliant with treatment further exacerbates the degree of dysfunction[49]. Apathy is significantly related to reduced independence in activities of daily living (ADLs) beyond dementia severity[77], survival duration after RACS admission[78] and poor rehabilitation outcomes. Those with dementia living alone may be at risk of self-neglect when unable to reliably initiate ADLs[38]. The person with dementia and apathy may retain capacity but not self-initiative and hence may undertake activities with or for others that they are unable to do alone[31, 79]. Morbidity and mortality may also be indirectly related to apathy as residents in long-term care tend to be less noticed by care staff and receive fewer direct care hours[78, 80, 81]. Deficits also influence staff-resident interactions and quality of care[81] as well as staff frustration and job satisfaction[82, 83].

Apathy is under-recognised and difficult to treat[25, 30], not least because people with dementia have poor insight into their condition[22]. It imposes high levels of economic, social and physical burden and distress on partners and carers[84], compounding disability in those with dementia[80] and frequently leading to earlier admission to RACS than for those who are similarly impaired without apathy[85].

Results

A literature search to set criteria (see *Appendix 5*) yielded 14 psychosocial/environmental and 16 pharmacological/biological intervention studies with relevant outcomes. The cognitive rehabilitation/stimulation and education/training groups incorporated the greatest number of psychosocial/environmental studies with 3 studies each. The cholinesterase inhibitors (ChEIs) and/or memantine category incorporated the greatest number of pharmacological/biological studies. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained but not included in the summary numbers.

Addressing apathy

Recognised guidelines for the treatment of apathy do not exist[30], yet response to treatment may have important implications in delaying residential care admission, alleviating carer burden and in turn improving quality of life for those with dementia. Indications for treatment of apathy are unclear. These differ from other psychiatric conditions such as depression because patient distress rarely applies to those with apathy, and in dementia apathy typically worsens with time rather than remitting. Additional confounds such as metabolic disorders, environmental differences as well as concurrent acute and/or chronic illnesses should be considered. *Appendix 4* provides suggested questions to facilitate comprehensive assessment.

Acknowledging individuality, personal history and previous interests may guide individual strategies for engaging those with apathy as well as variations in response[79]. Person-centred

care likewise seeks to view the person as a whole, incorporating the individual's personal and social psychology in developing a treatment plan[49, 86-88]. When treating apathy in dementia, the excess disability of the person with dementia, potential for improvement in quality of life and burden to carers and/or family should be considered.

Psychosocial and environmental interventions

Studies of psychosocial and environmental interventions were mainly conducted in residential settings with two studies set in the community, one in both RACS/community and one study undertaken in hospital.

Cognitive rehabilitation/stimulation
Quality of research studies: 1/3 strong, 2/3 moderate
Outcomes of studies: 2/3 positive, 1/3 negative

Two medium size studies provided moderate quality evidence of a small effect for brain-activating rehabilitation (BAR)[89] added to rehabilitation over 1 week in acute hospital[90] and added to usual care over 12 weeks in RACS[91]. BAR emphasises the importance of group membership, acceptance and supportive care through activities such as reminiscence, music and therapeutic activities. In both studies, MOSES withdrawal scores decreased for people receiving BAR and interventions outperformed standard rehabilitation/usual care. In contrast, a strong study comparing 90-minute sessions of small group cognitive training versus group reminiscence therapy versus individual cognitive rehabilitation versus usual care over 24 months reported no difference between groups for AI apathy scores[92].

Outcomes from a community-based RCT, undertaken prior to 2012, approached significance for reduced apathy in mild to moderate AD for a cognitive-communication stimulation program[93].

Music
Quality of research study: 1/1 moderate
Outcome of study: 1/1 positive

One moderate quality study reported positive results for group music sessions incorporating listening to music, singing and playing a xylophone three times weekly over 3 months[94]. Research undertaken prior to 2012 demonstrated moderate support for reduced apathy with rhythmical instruments[95] but no benefit for a small study of twice-weekly music sessions[96].

Reminiscence
Quality of research studies: 1/2 strong, 1/2 moderate
Outcomes of studies: 1/2 positive, 1/2 negative

One medium study of moderate quality demonstrated a large effect for ARS apathy scores for weekly structured object-/activity-based group reminiscence therapy[97] supported by culturally appropriate *YouTube* videos compared with small group unstructured conversation sessions[98]. Another medium study of strong quality compared 6 weeks of playing the Memory Matters (MM) iPad reminiscence game in therapist-guided, small group or individual sessions followed by 6 weeks of self-initiated/guided play with usual care. At 12 weeks there was a nonsignificant trend toward reduced apathy scores for small group MM play[99].

One reminiscence study undertaken prior to 2012 provided evidence for reduced apathy in day centre attendees with AD and VaD[100].

Animal-assisted interventions
Quality of research studies: 1/1 moderate
Outcomes of studies: 1/1 negative

Small group sessions with a companion robot *Paro* reported no change in apathy scores when compared with interactive reading[101]. Studies undertaken prior to 2012 provided moderate evidence of benefit for a therapeutic *robocat*[102], dog therapy[103, 104] and animal-assisted therapy with real cats compared with toy cats[105].

Multisensory interventions
Quality of research studies: 1/1 moderate
Outcomes of studies: 1/1 possibly positive (no control group)

A medium study indicated a medium effect for individualised multisensory stimulation sessions (MSS) and for one-to-one activities[106, 107]. In research conducted prior to 2012, evidence was provided for reduced apathy with MSS in severe dementia[108]. Further studies provided moderate support for inpatient multi-sensory behaviour therapy[109] and MSS integrated into 24-hour residential care[110].

Models of care
No recent study of models of care interventions met our criteria for inclusion and two studies conducted prior to 2012 found no benefit for an emotion-oriented model of care[111, 112].

Education/training
Quality of research studies: 3/3 moderate
Outcomes of studies: 3/3 negative

Studies of care staff education/training featuring Function-Focused Care[113, 114] and psychologist-delivered education sessions on BPSD, apathy and depression[115] provided moderate quality evidence of no effect for apathy.

Multicomponent interventions
Quality of research studies: 2/2 strong
Outcomes of studies: 1/2 positive, 1/2 negative

A very large RACS trial compared permutations of person-centred care (PCC) and antipsychotic review and/or physical activity and/or weekly social interaction with pleasant activities. NPI apathy subscale scores decreased for both PCC plus antipsychotic review plus exercise and PCC plus antipsychotic review plus social interaction at 9 months compared with baseline[116]. A small study showed no good evidence for an intensive 3-month community gymnasium-based program of socialisation, physical activity and cognitive stimulation[117].

Physical activity
Quality of research studies: 1/1 strong
Outcomes of studies:1/1 some evidence

A large study demonstrated a medium positive effect for biographically oriented mobilisation sessions provided by an occupational therapist and sport therapist weekly compared with usual care. Apathy scores were unchanged after 10 months however, scores increased significantly for usual care indicating a significant difference between groups and some benefit for the intervention[118]. A study conducted prior to 2012 demonstrated evidence of no benefit for *walk and talk* sessions[119].

Summary

Some benefit for apathy in dementia is demonstrated for cognitive rehabilitation, physical activity, multisensory, music, multicomponent, one-to-one activities and reminiscence-based interventions. Three strong to moderate quality studies demonstrated positive outcomes for tailored or individualised activities[106, 107, 116, 118]. A further four studies provided strong to moderate support for small group interventions[90, 91, 94, 98]. Across categories, there was strong to moderate support for guided interventions that incorporated elements of group reminiscence. Only three studies reported follow-up between eight weeks and one year after interventions ceased in RACS[106, 107, 115, 118], although none reported evidence that reductions in apathy were maintained.

Positive effects for guided group and one-to-one interventions suggest that delivery methods may be important, higher levels of guidance/supervision is necessary and social interaction is important. A biopsychosocial approach recognises that there are multiple contributors to apathy. To engage people with dementia and maintain interest and/or involvement, psychosocial activities should ideally be matched to the individual's interests and retained skills[79]. This is consistent with our findings. See *Appendix 2* for interventions reported above.

Pharmacological and biological interventions

Studies of pharmacological and biological interventions were conducted in community and clinic/hospital settings.

Cholinesterase inhibitors (ChEIs) and/or memantine
Quality of research studies: 7/7 moderate
Outcomes of studies: 1/7 positive, 1/7 possibly positive (no control group), 5/7 negative

A very large RCT demonstrated that adding choline alphascerate precursor cholinergic drug to donepezil resulted in a significantly greater decrease in NPI apathy subscale scores at 24 months compared with donepezil only, with small to moderate effect for people with mild to moderate AD[120].

Donepezil in combination with memantine was better at maintaining apathy scores at 12 months compared with galantamine combined with memantine[121].However, large and very large retrospective studies of moderate quality reported no decrease in AS apathy scores for donepezil in isolation or in combination with memantine[122, 123].

A very large open-label observational study of switching from oral donepezil or galantamine to rivastigmine patch compared with switching from rivastigmine patch to non-rivastigmine oral treatment, reported no change in NPI apathy subscale scores for people with mild to moderate AD[124]. Two retrospective studies reported above found no benefit for rivastigmine in isolation[123] or in combination with memantine[122].

A very large retrospective chart review of any switch between rivastigmine, galantamine, and/or donepezil provided evidence of no change in AS apathy scores at 6 months[125]. Other large retrospective studies showed no change in AS apathy scores for galantamine or switching from other ChEIs to galantamine[123, 126] nor for galantamine in combination with memantine[121] nor effiacy of memantine treatment in any manner, i.e. monotherapy or in combination with other ChEIs[122].

Research conducted prior to 2012 reported two of three donepezil studies provided evidence for reduced apathy[127-129] and a further two trials reported positive outcomes for galantamine[130, 131].

Psychostimulants
Quality of research studies: 1/3 strong, 2/3 moderate
Outcomes of studies: 1/3 positive, 1/3 some evidence, 1/3 negative

Psychostimulants are known to influence anatomic substrates that regulate wakefulness and executive function. Recent systematic reviews note that side effects such as tachycardia and psychosis may be a concern[132] however, the risk of adverse events compared with placebo may not be significant[133]. A strong, medium study of methylphenidate reported greater decrease in NPI apathy subscale scores compared with placebo for people with mild to moderate AD at 6 weeks[134, 135]. A moderate quality study in community-dwelling veterans with AD reported a large effect for methylphenidate in AES-C apathy scores at 12 weeks[136]. In contrast, supplementing ChEI treatment with modafinil did not outperform ChEI alone in lowering FrSBe apathy scores for psychiatric hospital outpatients with mild to moderate AD[137]. Research reported before 2012 indicates limited efficacy for reduced apathy with methylphenidate[138].

Other pharmacological/biological treatments
Quality of research studies: 1/2 strong, 1/2 moderate
Outcomes of studies: 2/2 negative

A multinational RCT provided strong evidence of no difference compared with placebo in total AES apathy scores after 1 year for sembragiline, a monoamine oxidase B inhibitor, add-on in people with moderate AD, already taking an AChEI or AChEI plus memantine[139]. Evidence from a medium study indicated no change in total AES scores for twice-daily sprays of intranasal oxytocin compared with saline spray at 1 week[140].

Brain stimulation therapies
Quality of research studies: 2/2 strong
Outcomes of studies: 1/2 limited evidence, 1/2 negative

Two trials of brain stimulation focused on the left dorsolateral prefrontal cortex (DLPFC) reported mixed results. A small trial of repetitive transcranial magnetic stimulation (rTMS) outperformed sham rTMS with a greater decrease in average AES-C apathy change scores following treatment 5 days per week over 4 weeks. Effects were not maintained at 4- or 8-week follow-up[40]. Transcranial direct current stimulation (tDCS) was no more effective for lowering SAS apathy scores than sham treatment after 2 weeks in a medium study of people with moderate AD[141].

Summary

Few pharmacological trials have examined apathy, considering its prevalence and burden on carers. Even fewer reported apathy as a primary outcome. The ChEIs and/or memantine category incorporated the greatest number of studies but included only one RCT[120]. Some evidence is reported for the efficacy of ChEIs although no clear indication that any one ChEI is superior. Trials provide mixed evidence of ChEI efficacy in isolation and in combination with other biological therapies.

No studies of analgesics, traditional medicines, antidepressants or atypical antipsychotics met our quality rating criteria for inclusion here. This aligns with current guidelines which do not support antidepressants or antipsychotics for mild to moderate symptoms associated with dementia generally. Psychostimulants may decrease apathy in some contexts or people with more severe symptoms. Specific brain stimulation therapy may be a viable short-term option for some people with AD. These findings align with those of a recent systematic review[142]. There is little to no sound evidence that pharmacological treatments work well for apathy in dementia[30, 133, 143]. See *Appendix 3* for interventions reported above.

Limitations

The potential confounds of studying apathy in dementia are numerous: a lack of standardised, assessment guidelines for diagnosing apathy[1, 49, 58, 144]; difficulties in differentiating it from, and overlap with, depression, fatigue syndromes and parkinsonism[3, 28, 37]; underreporting of apathy[30, 62]; and similar symptoms secondary to medications such as psychotropics and beta-blockers[39-41]. Further, dementia type and severity likely influence the success of interventions trialled, yet many studies treated participants as a homogenous group[8, 21, 22, 62, 143, 144]. The degree to which findings can be generalised across the stages of dementia

STRATEGIES/OUTCOMES

- *Francis' care partner, family members and friends are distressed as her quality of life appears to be diminishing quickly. They were provided with information about the changed behaviours and psychological symptoms that frequently occur in dementia, including apathy.*
- *It was explained that Francis' reduced ability to initiate activities for herself and apparent lack of interest is not necessarily an indication that she does not enjoy the activities offered. They were encouraged to focus on supporting Francis in activities that she was previously particularly invested in or that were very well practised as these were likely to be the most successful.*
- *The importance of establishing a routine was discussed as Francis is also adjusting to the change from her structured work life. A defined sequence of events reduced the need for Francis to make decisions or question the situation.*
- *Family members and friends worked with Anne to develop a roster to provide extra support and encouragement around Francis' routine.*
- *In-home and community aged care services were investigated for an option to accompany Francis during her walks and/or daytime activities, some days. Anne is aware that additional support from services will be needed in the future, and it could be beneficial to all for Francis to become familiar with the care workers during these pleasant and familiar experiences.*
- *Francis' friends and family have a better understanding of ways to engage with her and assist her to enjoy activities of interest. They now phone again if Francis doesn't answer their call the first time.*

is largely unknown.

Many studies comprise small sample sizes, brief follow-up horizons[87, 133, 144] and methodological shortcomings such as a wide range of definitions and terminologies for apathy[2, 145]. There is little evidence to guide clinicians as to how apathy at different phases of dementia responds to interventions[8]. Further, there is scant evidence of sustainability of effect once interventions cease. Distal outcomes of apathy such as time to residential care admission[85] and complications of immobility secondary to apathy are also neglected[146].

Conclusions/Principles of care

Apathy is a major source of carer distress and frustration for those living at home[31-33, 74]. As a negative symptom however, apathy tends to pose less overt disruption and economic consequences in residential care settings relative to more overt changed behaviours, expressions of unmet need or psychological distress[78, 80, 81]. Targeting apathy may alert carers at home or in RACS to an additional problem that would not normally demand their attention. The secondary benefits of successful interventions may be better evident in social gains or maintenance of functional abilities.

While a 'one size fits all' approach to treating apathy in dementia is inappropriate, individualised programs require additional resources. The benefits to care providers and organisations must ultimately outweigh implementation costs[79]. While psychosocial and environmental interventions are warranted[30], there is a paucity of sound research to guide clinicians and carers. Apathy is complex and multifaceted and further rigorous research is needed, including the investigation of integrative approaches[147]. The limited sound evidence for reducing apathy should not prevent clinicians from considering strategies on an individual basis[148]. Psychosocial approaches have the potential to reduce apathy in dementia. Of these, individually tailored interventions that incorporate elements of support, guided reminiscence and social interaction provide the best evidence.

Of the pharmacological treatments reviewed, there is evidence that ChEIs, methylphenidate and rTMS therapy provide some benefit for apathy, and these findings align with current literature on pharmacological/biological treatment of apathy in dementia[25, 30, 142, 149]. Published reviews report little evidence that pharmacological treatments are effective, and none are approved for apathy in dementia[30, 144]. Drug therapies may be beneficial for apathy within specific disease pathologies, and individual characteristics may influence efficacy[149, 150]. Clinical use may be limited by contraindications and adverse effects[133, 144]. Australian clinical practice guidelines report no benefit for antidepressants in apathy in dementia. Antidepressants with anticholinergic properties should be avoided due to negative impact on cognition and risk of adverse events[151].

The threshold for initiating ChEI pharmacotherapy in dementia varies with the setting and those providing care. It has been argued that early initiation of treatment may delay the emergence of changed behaviours. However, long-term use carries risk and there is limited consensus regarding dis-/continuation[152-154]. The effects of interventions should be evaluated with a valid and reliable scale. Decisions regarding prescribing and deprescribing should be made in consultation with the person and their carers as part of informed shared decision making, considering treatment goals, potential benefits, harms, and ongoing monitoring[152, 155].

A lack of positive research outcomes for apathy is not necessarily indicative of a lack of efficacy. Stabilisation of apathy may indicate that an intervention is beneficial even without evidence of improvement as the natural history of apathy is progression with dementia severity[29]. Apathy in dementia remains underdiagnosed and difficult to treat. Initial therapy should aim at alleviating intercurrent/underlying illnesses, addressing unsuitable physical/environmental factors and trialling appropriate psychosocial therapies. Only when these measures have been attempted should pharmacological interventions be considered/employed.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

7. Delusions and hallucinations

MODULE 7: Delusions and hallucinations

Key messages

- Psychotic symptoms in dementia are delusions and hallucinations which are indicative of a disturbance in the perception and/or recognition of objective reality.
- Delusions and hallucinations are some of the most commonly occurring BPSD, with prevalence rates ranging from 9% to 76%.
- Delusions and hallucinations occur in delirium, schizophrenia and other primary psychotic disorders which must be differentiated from psychosis secondary to dementia.
- Delirium and potentially reversible causes must be excluded.
- At times, the presence of delusions and hallucinations can be more distressing for carers than for the person with dementia.
- Although research evidence is lacking, individualised psychosocial interventions are recommended initially unless symptoms are causing significant distress or safety concerns.
- A lack of evidence should not prevent clinicians from considering culturally appropriate psychosocial approaches on an individual basis, where there is potential for benefit to people with dementia and/or their carers.
- Where pharmacological treatment is indicated, due to significant distress or safety concerns, atypical antipsychotics with appropriate psychosocial interventions may be of benefit.
- Some evidence indicates pain management with analgesic medications may reduce delusions.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

Delusions and hallucinations summary

What are delusions and hallucinations and what do they look like in dementia?

Psychotic symptoms are delusions, which are fixed, false unshakable beliefs not shared by others from that person's culture, and hallucinations which are disturbances in the perception and/or recognition of objective reality. People with dementia may develop psychotic symptoms secondary to brain changes or driven by cognitive decline compromising their ability to process reality logically. Prominent agitation or depressive symptoms can occur with delusions and hallucinations.

Criteria for psychosis of dementia include symptoms severe enough to disrupt the person's functioning and/or others' functioning or pose a threat to the safety of the person and/or others. Symptoms are not better accounted for by the effects of substances, a delirium, another medical condition, primary psychotic disorder or mood disorder and are not considered culturally appropriate.

Causes of delusions and hallucinations

- Interaction of potentially reversible causes include sensory deprivation/impairment, inappropriate sensory stimulation, depression, psychological distress, and iatrogenic causes.
- Delusions and hallucinations can also arise from delirium, substance use and other medical conditions such as infection and/or metabolic, blood, and endocrine disturbance.
- Individual characteristics including age, gender, type of dementia, psychiatric history, age at dementia onset and illness duration, family history of dementia/psychiatric illness, and genetic mutations can influence the risk of experiencing psychotic symptoms.

Differential diagnosis

Delusions and hallucinations in dementia should be differentiated from schizophrenia or other primary psychotic disorders based on past history of psychosis, content of delusion or hallucination, presence of misidentification phenomena, active suicidal ideation, family history and the dosage and duration of antipsychotic treatment. It is also important to rule out delirium.

Measuring delusions and hallucinations

Psychotic symptoms can be assessed using the delusions and hallucinations subscales of the Neuropsychiatric Inventory (**NPI**), the Behavioural Pathology in Alzheimer's Disease scale (**BEHAVE-AD**), the CERAD Behavior Rating Scale for Dementia (**BRSD**) or the Columbia University Scale for Psychopathology in Alzheimer's Disease (**CUSPAD)**. The clinician-rated Dementia-related Psychosis 3 (**DRP3**™) brief screening tool may be useful in busy clinical settings.

Prevalence of delusions and hallucinations

Psychotic symptoms occur in 9.2% to 76% of people with Alzheimer's disease (AD). Delusions are the most frequently reported psychotic symptom, followed by hallucinations. Hallucinations are more prevalent in dementia with Lewy bodies (DLB) and Parkinson's disease dementia (PDD) and are rarely reported in frontotemporal dementia (FTD) or vascular dementia (VaD).

Effects of delusions and hallucinations

Delusions and hallucinations have been associated with more rapid cognitive decline, impaired *real-world* functioning, lower quality of life, higher risk of comorbid BPSD, earlier admission to residential care, higher healthcare costs and increased carer burden.

Addressing delusions and hallucinations

It is important to rule out delirium or potentially reversible causes and to confirm that the claims of the person with dementia are not actually occurring. The presence of delusions and hallucinations may be more distressing for carers than for the person with dementia. Those who experience more distressing symptoms, particularly in DLB or PDD, may require more active treatment.

Psychosocial and environmental interventions

- One study of multidisciplinary care in a specialised hospital ward with homelike environmental features which met our quality criteria for review, reported a negative outcome.
- Research evidence for psychosocial and environmental approaches to guide clinicians and care partners in addressing delusions and hallucinations is lacking.

Biological and pharmacological interventions

- Where psychotic symptoms are a significant concern or safety risk, pharmacological intervention may be indicated as a first-line treatment in combination with psychosocial approaches.
- Intervention studies included atypical antipsychotics, analgesic medications and brain stimulation therapy.
- Findings for atypical antipsychotics were mixed.
- Some evidence was reported for a stepwise pain management protocol in people with dementia, agitation and delusions.
- Where distress or safety is an issue, risperidone is the only antipsychotic medication that is Pharmaceutical Benefits Scheme (PBS) approved for use in BPSD, where nonpharmacological methods have been unsuccessful, for people with dementia in Australia.
- Clinical guidelines recommend consideration of a cholinesterase inhibitor (ChEI) as an alternative treatment for people living with DLB who cannot tolerate antipsychotic medication.

Limitations

Moderate to high quality studies of interventions addressing delusions and hallucinations in dementia are lacking in the literature. Limited evidence is provided of the sustainability of effects after interventions cease. Further, delusions and hallucinations were typically not the primary outcome in the included studies.

Conclusions/Principles of care

- While delusions and hallucinations can have significant consequences for people with dementia and those providing care in all settings, they do not always need intervention.
- Expert consensus guidelines recommend the use of individualised, culturally appropriate psychosocial interventions to address delusions and hallucinations.
- Where distress or safety is an issue, short-term pharmacological treatment with antipsychotics may be indicated as a first line approach in combination with psychosocial interventions.
- No studies published since 2012 provided evidence for the efficacy of psychosocial interventions to reduce psychotic symptoms in dementia.
- Effective pain management, including analgesic medication may help to reduce symptoms.

What are delusions and hallucinations and what do they look like in dementia?

Psychosis in dementia is a disturbance in the perception and/or recognition of objective reality[1] which may develop secondary to brain changes or driven by cognitive decline, compromising their ability to process reality logically. Psychotic symptoms in people with dementia are delusions and hallucinations[2]. Patterns of psychotic symptoms associated with Alzheimer's disease (AD) are different to non-AD dementias however, there is also considerable symptom overlap[3-5]. People with psychosis in dementia may develop more than one symptom, and symptoms may occur concurrently or at different time points[6].

A recent consensus criteria definition proposed by international expert clinician and research groups, IPA.STAART, expand previously published criteria for psychosis. The revised definition recognises that dementia-associated psychosis may feature prominent agitation or depressive symptoms[4]. These criteria apply to psychosis in all major and mild neurocognitive disorders[4, 7]:

- the presence of one or more characteristic symptoms i.e. visual or auditory hallucinations and/or delusions
- a primary diagnosis that meets all criteria for major or mild neurocognitive disorder
- symptoms not present continuously since before the onset of dementia symptoms
- symptoms present, at least occasionally, for at least 1 month or more
- symptoms severe enough to disrupt functioning of the person or others and/or pose a threat to the safety of self or others
- symptoms not better accounted for by effects of substances (e.g. medication, drugs of abuse, toxins), another general medical condition or other condition known to cause hallucinations (e.g. hypothyroidism, epilepsy, migraine, synaesthesia) not occurring exclusively during the course of delirium
- the criteria for a primary psychotic disorder or mood disorder are not met and such disorders do not account for the symptoms
- symptoms cannot be considered culturally appropriate (e.g. visions or spiritual visitations in some cultures/religious practices, hallucinations of a dead person during bereavement).

The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11) includes *Psychotic symptoms in dementia* under a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" under Neurocognitive disorders, Dementia. Psychotic symptoms are described as "clinically significant delusions or hallucinations"[8]. The DSM-5-TR does not provide a clear definition of psychosis in dementia[9].

A delusion is a fixed, false unshakable belief not shared by others from that person's culture. Delusions in dementia can be paranoid, simple, non-bizarre[2] and bizarre[4]. Subtypes of delusions common in AD-related dementias and dementia with Lewy bodies (DLB) include paranoid and misidentification, with themes of persecution. Jealousy, misinterpretations, including delusions of theft, suspicion, abandonment, danger, infidelity, and the delusion that one's house is not one's home also occur[3, 10]. Because delusions in dementia can sometimes represent reality (e.g. a nursing home is not actually a person's home, other residents may be taking items from their room)[11] and tend not to be fixed or indisputable, it has been suggested that they may not fit the DSM-5-TR definition[4, 12, 13].

Hallucinations are perceived sensory experiences such as an object or event that occurs in the absence of actual external sensory stimulation e.g., people see, hear, smell, feel, or taste things that do not appear to exist[4, 14]. Hallucinations in AD are more commonly visual than auditory. Recurrent visual hallucinations are a core feature in the diagnosis of DLB and Parkinson's

disease dementia (PDD). These can be grouped into hallucinations of passage (i.e. perceiving fast moving shadows moving past) and presence (i.e. sensing that 'someone' is nearby) and illusions (e.g. seeing a bear's face in a cloud). Hallucinations can also be classified as minor (e.g. seeing an object hovering in the corner) or complex (e.g. seeing a floating basket of bears in the corner)[3]. In those with DLB or PDD, visual hallucinations may occur daily, last for minutes, usually involve people or animals and are often experienced as unpleasant[3]. Hallucinations in PDD are associated with deficits in attention, language, verbal memory, visuospatial tasks and frontal executive dysfunction[15].

Misidentifications may be differentiated from hallucinations by the presence of external stimuli. Examples include the belief that a familiar person is an imposter, that images on television are real, that the image in the mirror is a stranger or that there are ghost-like intruders in the house[3, 5, 10]. Hallucinations and misidentification increase with worsening cognitive impairment in DLB[4, 14]. Differentiating between delusional misidentification and hallucinations can be challenging due to comorbidities and overlap in syndromes[3, 5].

PRESENTATION

Maria is an 85-year old widow who immigrated from Greece to marry at age 18. She has memories of leaving her family to take the long trip to Australia aboard a converted post-war 'troop ship'. She slept in triple tiered bunks in the large empty hold converted to the women's quarters. The plain and inadequate food made her feel homesick. Meeting her intended husband for the first time when she landed in Melbourne, learning English and living in a migrant hostel were major changes for Maria.

Maria's married life presented many challenges as she supported her husband who was ultimately diagnosed with PTSD. Her two daughters remember their father's explosive outbursts and the times when Maria took them to a neighbour's home for protection. When Maria developed dementia, her daughters supported her at home until her admission to residential care became necessary as her dementia progressed.

Maria has become increasingly anxious following the death of a resident who previously shared her room. She described visual hallucinations of the resident in her room and delusions of staff attempting to cause her injury. Maria is eating poorly and losing weight due to suspicions that her food is poisoned. Staff members report that she can be uncooperative and resistant, particularly during medication administration and personal care. Maria is becoming increasingly isolated as other residents are frightened by her response to her symptoms and they avoid her. At times she has tried to run away from the facility, which places her in considerable danger.

Causes of delusions and hallucinations

Delusions and hallucinations can arise from non-neurodegenerative causes such as delirium and drugs as well as medical conditions such as infections or endocrine, hematologic and/or metabolic disturbances[2, 5]. Hallucinations can arise from the interaction of potentially treatable factors such as misinterpretation of reality, sensory deprivation, vision loss, inappropriate sensory stimulation, depression, psychological distress and iatrogenic causes[5, 13, 16].

Age, gender, psychiatric history, age at onset and duration of dementia, dementia type, family history of dementia or psychiatric illness as well as genetic mutations contribute variably to risk of psychotic symptoms[5, 10, 17, 18]. In AD, older age, depression, and aggression are related to delusions; greater cognitive deficits, and dementia severity are related to hallucinations[5]. In frontotemporal dementia (FTD), psychotic symptoms are associated with a strong family history of psychosis and genetic mutations[10]. Female APOE4 carriers diagnosed with AD and LBD have a higher risk of psychosis[19].

Differential diagnosis

Delusions in dementia may resemble other phenomena such as confabulation and disorientation[3, 5]. Hallucinations and/or delusions may be mistakenly attributed to the person's religious/spiritual beliefs and/or cultural background. Cultural background and beliefs should be considered in assessment and treatment[20-24]. Sensory impairment should also be considered in assessment as lost or ineffective hearing aids and/or glasses can contribute to misinterpretation of the environment. Poor sleep, social isolation, loneliness, adversity and bereavement may also influence hallucinations[20]. It is important to differentiate delusions and hallucinations due to schizophrenia,

other primary psychotic disorders or a secondary psychotic syndrome[25]. Past history of psychosis and family history can help differentiate schizophrenia from psychosis of dementia[26].

Measuring delusions and hallucinations

While no standardised measure of psychosis in dementia currently exists, the following scales are widely used and assessment can be accomplished with good reliability and validity[3, 27]. Scales assess different aspects of psychosis[3, 28, 29] although substantial overlap exists. The Neuropsychiatric Inventory (NPI) and Behavioural Pathology in Alzheimer's Disease scale (BEHAVE-AD) are reportedly equivalent in detecting clinical improvement in response to treatment[29].

- The delusions and hallucinations subscales of the **NPI** are completed during an interview with the carer, in which they rate the frequency and severity of the delusions and hallucinations, as well as their own subsequent distress[30, 31]. The NPI-Clinician (**NPI-C**) is a revised version of the original NPI[31]. Other versions include the NPI-Questionnaire (**NPI-Q**), a brief self-administered questionnaire developed to be completed by informants[32] and the NPI-Nursing home (**NPI-NH**)[33]. All versions include items for delusions and hallucinations. The reliability and validity of the NPI overall is well established and it is available in multiple languages[3, 34].
- The 25-item **BEHAVE-AD** includes a psychosis subscale of seven delusions and five hallucinations items through informant interview, based on the preceding two weeks[35]. It is appropriate for use in acute, community and residential care settings and is available in multiple languages[3].
- The Consortium to Establish a Registry for Alzheimer's Disease Behavior Rating Scale for Dementia (**CERAD-BRSD**) contains two items relevant to delusions and one for hallucinations[36].
- The Columbia University Scale for Psychopathology in Alzheimer's Disease (**CUSPAD**) includes 11 items relevant to delusions and five for hallucinations. Psychotic features are identified with a simple decision tree, making it suitable for use by trained and lay interviewers[37]. The CUSPAD distinguishes between types of delusions[3].
- The Dementia-related Psychosis 3 (**DRP3™**) Screen is a brief clinician-rated tool to detect delusions and hallucinations in people with dementia in clinical settings. It is reportedly sensitive, has content validity and may be useful in medical practices[38].

Prevalence of delusions and hallucinations

In Australia, people living with dementia who are aged 65 years or less are more likely to report delusions and hallucinations compared with those in older age groups[39]. Delusions and hallucinations occur commonly in people with AD with reported prevalence between 9.2% and 76%[3, 10, 39]. The prevalence of delusions in AD ranges from 7.1% to 35% while hallucinations are reported less frequently at 5.6% to 18%[3, 40, 41]. Symptoms tend to increase in the mild to moderate stages of dementia and decrease in later stages[5, 10, 39] although some reports deviate from this[42].

Variation in prevalence rates is due to differences in populations studied (e.g. inpatients, outpatients, residential care or community), definition and assessment procedures, treatment with neuroleptic medications and diagnostic criteria for, dementia[3, 4, 18, 42]. The higher prevalence of hallucinations in people with DLB (22.3 % to 73%) and PDD (19.1% to 39.5%), along with the reported lower yet variable prevalence of delusions and hallucinations across different FTD variants (5.6% to 18.4%)[3, 40, 43] suggests these symptoms are related to disease-specific neuropathology[44-46]. Overall, psychotic symptoms in dementia appear to fluctuate with dementia type and over time, present episodically and frequently recur once present[3-5].

Effects of delusions and hallucinations

Delusions and hallucinations contribute substantially to poorer outcomes for people with dementia and their carers[6, 47]. Carers' reactions and responses to dealing with delusions or

ASSESSMENT

Potentially contributing factors to delusions and hallucinations should be identified:

- *Exclude potentially reversible causes*
- *Misinterpretation of reality and/or others' intentions*
- *Sensory deprivation/impairment or inappropriate sensory stimulation*
- *Illness/infection/delirium/depression*
- *Pain/discomfort not well managed*
- *Medication review: interactions, dosage, recently prescribed, adverse effects, compliance*
- *Lack of attention to culturally relevant needs*
- *Altered routine, unfamiliar people, reduced time spent with family*
- *Unfamiliar/altered physical environment*
- *Reduced threshold for coping with stress*

Assessing the situation:

- *Consult family members to identify possible reversible causes for delusions and hallucinations and/or underlying reasons for Maria's distress.*
- *Has a recent medical review been conducted?*
- *Encourage Maria to express her concerns as far as she is able.*
- *Directly observe what may speciffcally trigger the symptoms.*
- *Assess the immediate environment for possible triggers.*
- *Consider Maria's life history and previous experiences of trauma for further information with regard to triggers for the symptoms.*
- *Are the delusions and/or hallucinations troublesome to Maria?If not, do they need treatment?*
- *If so, is pharmacological intervention indicated? Is it possible and/or practical?*
- *What are her family's wishes?*

hallucinations impact on the person with dementia[48]. Education and support for family and/or paid carers may be indicated[49]. Supportive responses from family, friends and others in the community may also be helpful[50, 51]. Delusions and hallucinations are associated with increased carer burden[52-54], earlier residential care placement[55], poorer quality of life[56, 57] and physical health[58] as well as higher healthcare costs[59, 60]. Delusions and hallucinations, together or separately, have been associated with a more rapid cognitive decline in dementia[61] as well as other BPSD, including aggression[62] and more severe depression[4].

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded one psychosocial/ environmental intervention study and three biological/pharmacological studies published since 2012. Our quality rating criteria for inclusion, of moderate or strong studies, excluded one study of modest quality only. Where evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained but not included in the summary numbers.

Addressing delusions and hallucinations

Initially, it is important to rule out a delirium as the cause of delusions and hallucinations and to confirm that the claims made by the person are not actually occurring e.g., their valuables may have been stolen, visitors may be staying in their home. Delusions and hallucinations are not always distressing to the person with dementia. It is important for the clinician to determine what the symptoms mean for the individual and if the situation warrants treatment beyond supporting others in the care environment[63, 64]. While evidence of delusions and hallucinations should always be investigated for potentially treatable causes, the presence of a *benign* hallucination is typically more distressing for care partners, family and/or staff than for the person with dementia. *Appendix 4* provides suggested questions to facilitate comprehensive assessment.

By contrast, those with dementia, particularly DLB or PDD, experiencing complex, unpleasant visual hallucinations may benefit from active treatment. Even when the person retains insight into the fact that the hallucinations are not part of reality, their distress can be significant. Professional consensus recommends trialling psychosocial interventions, or where indicated in combination with medication, as part of an individualised care plan[65-67].

Psychosocial and environmental interventions

While research evidence for psychosocial/environmental interventions is lacking, clinical guidelines recommend that psychosocial interventions are considered on an individual basis, where they are beneficial to the person with dementia and culturally appropriate [68-70]. See Principles of care for guidance and *Appendix 2* for intervention reported below.

Models of care
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A large retrospective chart review of the effects of multidisciplinary inpatient care reported a decrease in the number of people presenting with NPI delusions. Care was provided in a specialised hospital ward with environmental features to provide a homelike atmosphere. No significant reductions in NPI delusions or hallucinations scores were found[71].

Biological Interventions

Expert consensus guidelines suggest that where psychotic symptoms are very distressing and/or dangerous to the person with dementia or others, pharmacological interventions should be used concurrently with psychosocial interventions as first-line treatment[68-70]. Pharmacological treatment may also be indicated when symptoms appear to have a physical or iatrogenic aetiology i.e. stemming from biological causes or are unresponsive to psychosocial interventions[69].

Atypical antipsychotics
Quality of research study: 1/1 strong
Outcome of study: 1/1 negative

In a very large study of pimavanserin, which was stopped because of efficacy, 61.8% of participants who had responded, were randomised to continuation with pimavanserin or placebo. Relapse rates, while generally low, were lower in those continued on pimavanserin (28%) versus those on placebo (13%)[72, 73]. Another very large study reported that pimavanserin did not decrease NPI-NH psychosis scores significantly at 12 weeks and was no more effective than placebo in people experiencing psychotic symptoms associated with moderate to severe AD[74].

Two very large RCTs published before 2012 provided evidence for the efficacy of risperidone in reducing psychotic

STRATEGIES/OUTCOMES

- *Medical review was arranged, including bloods and urine. An interpreter was engaged to assist with a psychogeriatric consult. Underlying depression was excluded.*
- *Maria suffers from visual impairment due to bilateral cataracts which could potentially contribute to her misinterpretation of items in the environment. The feasibility of cataract surgery was raised with her doctor.*
- *Family reported distressing situations that Maria experienced in communal living when migrating to Australia by ship and living in a migrant hostel. Maria's initial move to residential care and any subsequent changes to her environment provoke anxiety around her fear of communal living.*
- *A small night light was provided by the family to assist Maria's orientation if she wakes during the night.*
- *Some staff members had little knowledge of dementia and they became fearful of Maria when she tried to describe her symptoms. Staff training sessions in dementia, BPSD and trauma-informed care were provided.*
- *As Maria is reverting to her first language and her opportunities for formal education as a child were minimal, her ability to communicate her concerns and care needs to staff are increasingly limited. Visual resources and pictorial language aids were developed with the assistance of family. A language-appropriate telephone interpreter service was trialled but attempts were largely unsuccessful.*
- *Maria's new roommate is a friendly and sociable lady who is settling in well. Maria has responded positively to her company, despite communication limitations.*
- *Maria's family continue to visit frequently but they feel she lacks companionship between their visits. A referral was made to Greek-specific services including a request for a Greek-speaking visitor to spend time with Maria each week.*
- *The frequency of Maria's delusions and hallucinations decreased over time. With reduced isolation and increased support Maria is less distressed by the symptoms when they do occur. Staff indicate that they feel more skilled in dealing with these situations as they arise. A follow-up psychogeriatric review is scheduled in 2 months.*

symptoms after 12 weeks, one of which is also mentioned under antidepressants above[75, 76]. One study of aripiprazole[77] and two studies of quetiapine found no effect[76, 78-80]. Use of any antipsychotic medication must be time-limited and closely monitored for adverse effects.

Analgesic medications
Quality of research study: 1/1 moderate
Outcome of study: 1/1 positive

A very large residential aged care services (RACS) study that included a large subgroup of people with psychotic symptoms provided evidence of a small effect for a stepwise pain management protocol. Paracetamol then buprenorphine, pregabalin and/or morphine daily according to assessed pain needs was compared with usual care. NPI delusions, but not hallucinations, subscale scores decreased at 8 weeks in people with one or more symptom of psychosis[81].

Brain stimulation therapies
Quality of research study: 1/1 strong
Outcome of study: 1/1 negative

A medium study that compared four days of twice daily active transcranial direct current stimulation (tDCS) with sham tDCS placebo reported no change in NPI hallucinations subscale change scores, at any time point, in people experiencing visual hallucinations associated with moderate to severe LBD or PDD[82].

Antidepressants
No studies of antidepressants that were published since 2012 met our quality criteria for inclusion. A large study published prior to 2012 provided evidence of decreased psychotic symptoms for both, citalopram and risperidone, with fewer adverse effects for citalopram after 12 weeks[75].

Cholinesterase inhibitors (ChEIs) and/or memantine
No studies published after 2012 met our quality criteria for inclusion. A post hoc analysis of 12 trials published prior to 2012 reported benefit for memantine in people with the combination of agitation/aggression and delusions[83]. No further studies replicating these findings were found in our review. Pooled analysis of three RCTs provided evidence of no benefit for memantine over placebo for reducing psychotic symptoms in AD[84].

Summary

The adverse effects of pharmacological interventions raise concerns however, situations can arise which place the person with dementia and/or others around them at risk, requiring an urgent response. In these cases, expert consensus guidelines recommend risperidone for psychotic symptoms, with individualised treatment decisions based on thorough medication risk-benefit assessment[67-70]. Risperidone is the only Pharmaceutical Benefits Scheme (PBS) approved antipsychotic for treatment of dementia-related psychotic symptoms, where nonpharmacological methods have been unsuccessful, in Australia[69].

Current guidelines do not recommend the routine use of atypical antipsychotics as the risk of harms outweigh potential benefits[69, 70]. Therapeutic use must be approved by the person's attending physician or specialist and family carers/guardians must be informed prior to initiation. No antipsychotic medication is both safe and effective for the treatment of psychosis in dementia[67]. Antipsychotic use has been associated with further cognitive decline and greater risk of somnolence, extrapyramidal symptoms, abnormal gait, oedema, urinary tract infections, incontinence, falls, cerebrovascular adverse events and mortality[69].

Although classified as an atypical antipsychotic, pimavanserin is a selective 5-HT receptor subtype 2A inverse agonist and antagonist and, as such, does not induce clinically significant antagonism of adrenergic, dopaminergic, histaminergic, or muscarinic receptors[73, 85]. This mechanism of action is unique and pimavanserin has been associated with lower mortality than atypical antipsychotic use, in community-dwelling people with Parkinson's disease (PD), during the first 180 days of treatment[86]. Pimavanserin is approved for use internationally for psychosis

in PD[73, 87] however, it is not PBS listed for BPSD[69] and not routinely available in Australia[88]. As always, treatment decisions should be guided by all available evidence of efficacy balanced with potential adverse effects[72, 87]. See *Module 2, Table 2.3* for side effects associated with antipsychotics. See *Appendix 3* for interventions reported above.

Limitations

While current guidelines recommend psychosocial interventions as a first-line approach to address delusions and hallucinations in dementia, good quality research in this area is lacking. Treatment outcomes reported are generally dependent on the severity of psychotic symptoms at baseline, which may vary across dementia subtypes. Delusions and hallucinations were also typically not the primary outcome in the included studies. Problems occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. Further, limited evidence is provided of sustainability of effects after interventions cease with only one biological study reporting short-term, post-intervention follow-up[82].

Conclusions/Principles of care

In summary, delusions and hallucinations have significant consequences for people with dementia and those providing their care. Current expert consensus guidelines recommend the use of multidisciplinary, individualised and multifaceted care including psychosocial interventions as a first-line approach and short-term pharmacological intervention only where indicated. While quality evidence for psychosocial interventions is lacking, clinicians should consider culturally appropriate psychosocial interventions on an individual basis where there is potential for benefit to the individual with dementia and/or their carers[68-70]. Stepwise pain management may also be beneficial in decreasing the severity of delusions.

Where symptoms are severe, causing distress and/or a safety risk to the person and/or others, short term pharmacological treatment with risperidone may be indicated. In some cases, ChEIs, memantine and citalopram may serve as alternatives to atypical antipsychotics. Individuals with different types of dementia may respond to medications differently. Antipsychotic medications should be avoided where possible in people with DLB as they are at particular risk of extrapyramidal side effects and other severe negative reactions. Where they are deemed necessary, only second generation/atypical antipsychotics should be prescribed. Published evidence and clinical guidelines recommend consideration of a ChEI as a safer alternative treatment in people with DLB[69, 89].

Delusions and hallucinations in people with dementia can occur as a consequence of many potential antecedents. See *Module 1, Table 1.2* for a list of potential contributory factors. The identification of triggers or underlying causes will assist in addressing the symptoms and associated behaviour. Where pharmacological intervention is indicated for distressing delusions or hallucinations, these should be prescribed with appropriate psychosocial interventions. Additionally, individualised medication risk-benefit assessment should be undertaken, informed consent must be obtained, potential side effects discussed and a protocol put in place for monitoring, review, reduction and/or discontinuation when indicated[69, 70]. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[69, 90]. For example, where the person presents with psychotic symptoms and agitation, treating the psychotic symptoms may also reduce agitation.

Pain management is important in the treatment of BPSD and should always be considered, although only one trial of analgesic medication indicating benefit for delusions is included here[81]. While evidence for interventions to guide clinicians and care partners in supporting those who present with delusions and hallucinations is limited, research and drug trials for psychosis in dementia are ongoing[66, 89, 91].

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

8. Depression

MODULE 8: Depression

Key messages

- Symptoms of depression in dementia include unhappiness, irritability, social withdrawal, inactivity, fatigue, tearfulness and loss of interest.
- Depression may be a presenting feature of dementia and it is one of the most challenging psychological symptoms to diagnose and treat.
- Cultural differences and experiences of trauma may influence symptom presentation and manifestation.
- Depression in dementia should be differentiated from apathy, anhedonia, sleep disturbance, 'quiet' delirium or the underlying dementia.
- The prevalence of depressive symptoms in dementia ranges from 10% to 78% and reportedly clusters around 39%.
- Expert consensus and guidelines recommend individualised psychosocial interventions as a first-line approach.
- Support was demonstrated for selected psychosocial and environmental interventions, with music and reminiscence approaches providing the best evidence.
- Few studies indicate effective pharmacological treatment and strong evidence demonstrated limited efficacy for antidepressants.
- Studies indicated that light therapy techniques outperformed placebo and usual lighting.
- Appropriate psychosocial intervention should accompany any indicated pharmacological or biological intervention.
- Psychotic depression and suicidal ideation require urgent psychogeriatric review.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

Depression summary

What is depression and what does it look like in dementia?

Symptoms of depression in dementia include:

- unhappiness
- inactivity
- tearfulness
- sleep disturbance
- low self-esteem
- hopelessness
- irritability
- social withdrawal
- fatigue
- loss of interest
- appetite disturbance
- negativity
- suicidal ideation.

Cultural differences as well as past and ongoing experiences of trauma can influence symptom presentation and manifestation.

Causes of depression

Degenerative changes in the brain associated with dementia can lead to depression. Previous history or family history of depression as well as personal history of trauma, abuse or adversity may be contributing factors. Depression in the early stages of dementia may be related to an awareness of the losses and prognosis associated with diagnosis.

Differential diagnosis

It is essential to differentiate depression in dementia from anhedonia, sleep disturbance and impaired concentration. Apathy and 'quiet' delirium should also be excluded, bearing in mind that apathy can be a symptom of depression. Apathy can also occur independently and be mistaken for depression.

Measuring depression

Expert consensus recommends the use of the Cornell Scale for Depression in Dementia (**CSDD**) and, unless cognitive impairment is too severe, the Geriatric Depression Scale (**GDS**) to assess depression in dementia for non-indigenous populations. Other rating scales used to assess depression include the Hamilton Depression Rating Scale (**HAM-D**), the depression/dysphoria subscale of the Neuropsychiatric Inventory (**NPI**) and the dysphoria subscale of the NPI-Clinician (**NPI-C**). When administered by trained and culturally competent clinicians, the adapted nine-item Patient Health Questionnaire (**aPHQ-9**) may be an appropriate screening tool for depressive symptoms in Indigenous Australian adults.

Prevalence of depression

- Depression is one of the most commonly occurring psychological symptoms in dementia, with prevalence rates clustering around 39% and a range of 10% to 78%.
- Variance in prevalence arises from differing definitions, diagnostic tools used and populations assessed.
- People with vascular dementia (VaD) and Parkinson's disease dementia (PDD) have higher rates of comorbid depression compared to other dementia subtypes.

Effects of depression

Depression in dementia is frequently underdiagnosed and undertreated, impacting on quality of life. Apart from the distress to the person affected, depression is associated with increased carer burden, earlier admission to residential aged care, increased mortality, medical comorbidity, social withdrawal and reduced quality of life.

Addressing depression

It is important for the clinician to identify potentially reversible factors that may be contributing to the depressive symptoms in the person with dementia. Untreated physical symptoms such as those related to infection, constipation and/or pain may be exacerbating the low mood. Expert consensus guidelines recommend psychosocial interventions as a first-line approach for non-psychotic depression in dementia.

Psychosocial and environmental interventions

- Psychosocial and environmental intervention studies for addressing depression in dementia have increased in number, quality and complexity over the past decade.
- Psychosocial/environmental interventions were primarily conducted in residential aged care and community settings. Few studies were conducted in acute hospitals.
- Music and reminiscence approaches, both individual and group, provided the best evidence for addressing depression in dementia.
- The cognitive rehabilitation/stimulation and multicomponent categories included the most intervention studies and also provided evidence of benefit.
- Multicomponent interventions provided the best strength evidence with the highest number of strong quality studies.
- Therapeutic recreation approaches also provided evidence of efficacy in these settings.

Biological interventions

- Pharmacological and biological intervention studies were primarily conducted in the community.
- The evidence for the efficacy of antidepressants is limited and expert guidelines note that these are primarily ineffective for people with depression in dementia.
- If indicated, there is some evidence that escitalopram or sertraline may provide short-term benefit.
- Findings reported are based on comparisons of group data of antidepressants versus placebo and it is possible that individual patients may respond.
- Light exposure therapy outperformed usual or placebo lighting, although the degree of benefit may be dependent on climate and the person's usual exposure to sunlight.
- Very limited evidence for the efficacy of Cholinesterase inhibitors (ChEIs) and memantine, alone or in combination with other therapies is presented.
- Single studies indicated limited benefit for other pharmacological and biological treatments such as brain stimulation therapies, methylphenidate, traditional medicines and citicoline, a cholinergic supplement.

Limitations

Many intervention studies have limitations around methodology and sampling which impact on outcomes and/or the generalisability of results. Few studies conducted long-term follow-ups to determine the sustainability of intervention effects.

Conclusions/Principles of care

- In line with expert consensus recommendations, an increasing number of psychosocial interventions were reported as effective in addressing depression in dementia.
- Music and reminiscence approaches provide the most promising evidence.
- Few studies indicate effective pharmacological treatment and the evidence for antidepressants is limited, although individual responses can vary.
- If pharmacological or biological interventions are indicated, these should be provided with psychosocial and/or environmental approaches to addressing depression.

What is depression and what does it look like in dementia?

Depression in dementia can present as unhappiness, irritability, social withdrawal, inactivity, fatigue, tearfulness and loss of interest contributing to excess disability[1]. Other symptoms that can also manifest are disturbed sleep, poor appetite, low self-esteem and energy, negativity, hopelessness and to a lesser extent, suicidal ideation or thoughts. Cultural differences as well as past and ongoing experiences of trauma may influence symptom presentation and manifestation[2-4]. Severity of depression can vary from mild through moderate to severe. There is support for a relapsing-remitting course in depression of dementia[5, 6].

The DSM-5-TR does not provide a clear definition for BPSD[7]. The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11) includes *Mood symptoms in dementia* under a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" under Neurocognitive disorders, Dementia[8]. The National Institute of Mental Health (NIMH) provides provisional criteria for depression of Alzheimer's disease (AD) that specifies that depressive symptoms be present for at least 2 weeks, represent a change from previous functioning and not occur as a result of a medical condition other than AD or non-mood related dementia symptoms.

Causes of depression

Dementia is associated with brain changes that can lead to depression, for example AD and Lewy body pathology, monoamine neurotransmitter alterations, cerebrovascular disease and/or neuroinflammation. An overlap in the underlying neural substrate of AD and depression is also reported[11, 12]. Genetic factors may contribute to depression in dementia and depression may have a causal role in AD[13]. Dopaminergic and serotonergic gene pathways have been associated with the manifestation of mood syndromes[14]. Depression, particularly late-onset, frequently presents prior to a diagnosis or in the early stages of dementia[15, 16] and may be a prodrome for AD[17] providing further support for this association.

Depression in the early stages of dementia may be related to an awareness of the losses and prognosis associated with the diagnosis, however evidence suggests this accounts for only a small percentage of cases[1, 18]. A family history of major depressive disorder, younger age, neuroticism[19] and pain in people with concomitant communication impairment[20] may contribute to depression in dementia.

Differential diagnosis

A diagnosis of depression in dementia requires differentiating symptoms from apathy, hypoactive or quiet delirium and the underlying dementia[5, 21, 22]. Apathy can also cause symptoms which overlap with depression such as loss of interest, low energy,

PRESENTATION

Cathy is a transgender woman. She had wanted to affirm her gender for many years but Cathy waited until her children were adults before she transitioned. Cathy was diagnosed with dementia 5 years ago and moved into residential care 3 months ago. Staff report that they are having increasing difficulty encouraging Cathy to attend activities that she previously enjoyed. They often have difficulty getting her up in the mornings with Cathy complaining that she is too tired to come to breakfast. Her family are concerned that Cathy doesn't appear pleased to see them or enjoy their visits, even when they bring her adored grandchildren. Cathy is sad and tearful much of the time. Her lack of appetite has led to recent weight loss. Cathy's notes confirm that she has lost 4kg since admission.

Since her admission Cathy has found most of the care staff to be supportive in helping with her grooming. They know that like many of the other ladies, Cathy feels better about herself when her hair is done nicely, and she is wearing some make up. A few staff members have made a special effort to lift Cathy's mood recently with additional attention to her grooming. Some of the other residents have commented negatively on staff assisting Cathy in this way and expressed their transphobia in front of her. On occasions like this Cathy has been heard to say that she would be "better off not living". Cathy is further distressed when she is aware that some staff members are not comfortable with providing her care.

withdrawal and lack of motivation. Apathy can be distinguished by a lack of emotion rather than typical depressive cognitions, emotions and behaviours such as feelings of sadness, tearfulness or complaints about futility of life[23]. Apathy can also be a symptom, and an early marker, of depression in dementia which further compounds diagnosis[24].

Many symptoms of dementia overlap with those of depression. For example, disturbed sleep, anxiety, agitation, appetite changes, impaired concentration or decreased engagement may be mistakenly attributed to depression[25, 26]. Pathological crying (i.e. sudden onset of crying episodes in the absence of underlying emotional change) can be misdiagnosed as depression[27]. Differential diagnosis is often difficult. The overlap of symptoms with possible underlying medical conditions and the dementia itself requires a sound knowledge of medical and psychiatric causal factors[28]. A therapeutic trial may aid the decision[29].

Measuring depression

The assessment of depressive symptoms in dementia must be comprehensive and include a risk assessment for the possibility of self-harm[30, 31]. Culturally appropriate screening for people of culturally and linguistically diverse backgrounds is essential[32, 33]. The following scales are widely used to assess depression in dementia:

- The Cornell Scale for Depression in Dementia (**CSDD**) is a 19-item clinician-rated tool based on assessment of the person and a semi-structured interview with an informant[34]. Expert consensus recommends the CSDD for assessing depression in dementia[35, 36].
- The Geriatric Depression Scale (**GDS**) is a short self-report scale designed for rating depression in the elderly. The 30-, 15-, 10- or 4-item versions can be read aloud. Yes/no responses only are required regarding the previous week[37]. The GDS is recommended for assessment of less severe symptoms and for use in community[38-41].
- The Hamilton Depression Rating Scale (**HAM-D**)[42] is a clinician-rated tool consisting of 17 or 21 items requiring graded responses according to the severity of symptoms over the past week [43].
- The adapted nine-item Patient Health Questionnaire (**aPhQ-9**)[2] is the only formally validated screening tool which has been culturally adapted for depression in Indigenous Australian adults. It is not specific to depression in dementia. The questionnaire is designed to be completed with the person by trained, culturally competent clinicians with local knowledge in a semi-structured interview. It is a tool for initiating discussions with the person about their mood rather than determining the need for treatment and does not replace careful assessment.
- The depression/dysphoria subscale of the **NPI** is completed during an interview with the carer, in which they rate the frequency and severity of the person with dementia's depressive symptoms over the past four weeks as well as their own subsequent distress[44, 45].
- The NPI-Clinician (**NPI-C**) has expanded the dysphoria subscale of the NPI to include six additional questions around facial expression, pessimism, irritability, eating habits, feelings of guilt and loss of enjoyment[46].

Prevalence of depression

Depression is one of the most commonly occurring psychological symptoms of dementia[11]. Although prevalence ranges from 10% to 78%, it reportedly clusters around 39%[4]. Major depressive disorder in dementia rates vary between 13% and 35%[47]. Variance in prevalence arises from the differing definitions, difficulty making an accurate diagnosis in dementia, populations assessed, variability in carer stress influencing reporting and different assessment tools, some of which are not designed for assessment of those with comorbid dementia[4, 47].

Depressive symptoms in dementia tend to increase in prevalence as cognition declines and

then decrease when cognition is severely impaired[1]. Prevalence possibly decreases over time, in part, as the person becomes progressively less able to interpret and/or report their mood[48]. With the progression of dementia, assessment of depressive symptoms increasingly relies on observation[11]. Previous history of depression, family history of mood disorders in first-degree relatives, personal history of emotional problems, female gender, poor self-reported health, pain, a recent major loss and younger age have been associated with a higher risk for depression in dementia[10, 19, 20].

Depression is more prevalent in vascular dementia (VaD), dementia with Lewy bodies (DLB), and Parkinson's disease dementia (PDD) than in AD, and is reportedly more persistent in the multi-infarct than subcortical subtype of VaD[4, 49]. A higher frequency of neurovegetative symptoms (e.g. changes in eating habits or weight, fatigue) are reported in VaD than in AD[50]. Concomitant BPSD, particularly irritability, aggression, anxiety, agitation and psychosis are reportedly high among people with dementia and depressive symptoms and it is important to consider all presenting symptoms when developing a behaviour support plan. Treating depression in dementia may aid in preventing and addressing physical aggression[51].

Effects of depression

Depression may be a presenting feature of dementia and it is one of the most challenging psychological symptoms to diagnose and treat. Despite the high prevalence of depression in people with dementia living in residential care, it is frequently underdiagnosed and undertreated[52, 53]. Depression in dementia is associated with increased carer depression and burden, earlier admission to residential aged care services (RACS), higher likelihood of suicide, increased mortality, greater disability in activities of daily living (ADLs), medical comorbidity, social withdrawal and reduced quality of life[52, 54]. Multiple unmet needs have been reported in people with dementia and depression living in residential care, which can lead to an increase in other BPSD[55, 56].

Results

Studies of interventions for addressing depression in dementia have increased in number, quality and complexity since 2012. A systematic literature review to set criteria (see *Appendix 5*) yielded 127 psychosocial and environmental as well as 30 biological and pharmacological intervention studies with outcomes relevant to depression. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained but not included in the summary numbers.

ASSESSMENT

To reduce Cathy's presenting symptoms, potentially contributing factors must be identified:

- *Chronic or acute pain/discomfort/ illness/infection*
- *Medication review: interactions, dosage, adverse effects, recently prescribed*
- *Overstimulation (noise, people, activities) or impoverished environment/boredom*
- *Lack of attention to Cathy's specific needs and historical trauma*
- *Others expecting too much of her and Cathy trying to overextend her capabilities*
- *Altered routines, new care staff, particular staff, other residents and/or family members*
- *Unfamiliar/altered/threatening physical environment*
- *Reduced threshold for coping with stress*

Assessing the situation:

- *Encourage Cathy to express her needs as far as she is able.*
- *Directly observe any situations that appear to exacerbate her depressive symptoms.*
- *Consult close family members to identify potentially contributing factors.*
- *Consult Cathy's life history for further information with regard to her symptoms.*
- *Ask staff who know Cathy quite well if they can assist in identifying any unmet needs or possible reasons for her increasingly low mood.*
- *Assess the immediate environment for possible factors contributing to Cathy's symptoms.*
- *Is a trial of pharmacological interventions indicated and/or appropriate for treatment of Cathy's depression?*

Addressing depression

While depression in dementia is largely attributed to brain changes and genetic factors, it is nonetheless important to identify potentially reversible factors that may be contributing to the low mood. Untreated physical symptoms such as those related to infection, constipation and/or chronic pain may exacerbate depressive symptoms. *Appendix 4* provides suggested questions to facilitate comprehensive assessment. Expert consensus and guidelines recommend psychosocial interventions as first line treatment[57-59]. As the course of depressive symptoms in dementia can be relatively transient[6], evaluation and follow-up of initial symptoms is indicated prior to considering pharmacological treatment[5, 60].

Psychosocial and environmental interventions

Trials of psychosocial and environmental interventions were primarily conducted in residential aged care and community settings. Few studies were conducted in hospital settings. Cognitive rehabilitation/stimulation and multicomponent intervention studies included the highest number of studies. One study in each of these categories provided strong evidence of positive outcomes[61, 62].

Music
Quality of research studies: 3/14 strong, 11/14 moderate
Outcomes of studies: 5/14 positive, 5/14 mixed/limited/some evidence, 4/14 negative

Personalised music showed benefit in people hospitalised for acute BPSD including people with severe AD[63, 64]. A medium study of daily 30-minute individualised music sessions via cordless headphones reduced negative mood when compared with usual care, over an average 23-day hospital stay[64]. Day centres and hospital inpatient centres offered group coaching for incorporating singing or music listening in everyday care for people with dementia and their family carers or nurses. This large study provided strong evidence of a small positive effect when compared with usual care plus group physical or social activities[65].

Ten weeks of passive listening to preferred music and one-to-one music listening and interacting with a music facilitator (e.g. singing and/or rhythmic movement) improved mood scores in a medium study of group home residents and hospital in-patients with severe AD[63]. A medium study of assisted living facility residents with moderate to severe dementia provided equivocal evidence for the benefit of streamed customised music to individuals' rooms. Depression scores decreased for music delivered in late afternoon and evening compared with the morning[66].

A large study provided strong evidence that supplementing occupational therapy (OT) sessions with 60-minute interactive group music stimulation (e.g. singing, rhythmic movement) 5 times weekly over 8 weeks decreased CSDD depression scores and provided benefit over usual OT[67]. A very large study provided moderate evidence that music with movement using props and instruments decreased depression scores after 6 weeks and was better at decreasing depression than chatting socially while listening to music[68]. Two large studies offered moderate quality evidence that small group tailored sessions delivered by music therapists with musical instruments and/or singing decreased depression after two weeks[69] and decreased scores at six weeks compared to usual care[70]. These four studies were undertaken in RACS.

Two small RACS studies provided moderate quality evidence of positive outcomes for sessions delivered by professionals. Intensive 70-minute group rhythmic percussion and singing sessions decreased depression scores after 12 weeks in people with cognitive impairment[71]. Small group receptive listening and drum playing to recorded music for people with severe dementia improved verbalisation mood scores and facial expression mood scores after 4 weeks[72].

Two RACS studies of people with mild to moderate AD[73, 74] and one study of people with moderate to severe dementia[75] reported no benefit for active music therapy, and one study of male veterans with mild to moderate AD reported no benefit for music therapy with percussion instruments when compared with rest and reading[76].

Reminiscence-based interventions
Quality of research studies: 3/10 strong, 7/10 moderate
Outcomes of studies: 3/10 positive, 6/10 mixed/limited/some evidence, 1/10 negative

Weekly group reminiscence sessions delivered over 8 weeks and 5 weeks of individual reminiscence sessions provided strong and moderate evidence of decreased depression scores, respectively. Both were more effective compared to usual care in these medium studies[77, 78]. Two large studies of the *SolCos* model of tailored reminiscence[79] provided moderate quality evidence of decreased depression after 4 and 8 weeks of individual sessions, respectively[80, 81]. A large RACS study provided moderate evidence of significantly decreased depression scores after 3 months of small group reminiscence sessions compared with social conversation[82]. A medium RCT provided moderate evidence of a small effect for productive activities with reminiscence in OT sessions compared to sharing a meal in a small group[83]. A small RCT provided moderate evidence of a small effect on depressive symptoms for the *Memory Box* computer app incorporating personalised multimedia content[84]. All these studies were undertaken in RACS.

A large hospital-based RCT provided strong evidence of a small effect for weekly small-group clinician-led reminiscence sessions tailored for people from Eastern cultural backgrounds, with a greater decrease in depression scores after 12 weeks when compared to usual care[85]. A medium study showed RN-guided one-to-one tablet-based app reminiscence therapy outperformed one-to-one personal storytelling sessions for women attending day centres, with moderate evidence of a medium effect for a greater decrease in depression scores[86].

In contrast, a medium RACS study provided strong evidence of no difference in depression scores between groups who co-created life story books (LSB) in weekly individual life review sessions with a psychologist compared with those who passively received a life story book made by family[87]. Further, group reminiscence sessions for people attending a community-based mental health centre showed moderate evidence of no effect in a small pre-post study[88].

Therapeutic recreation
Quality of research studies: 2/8 strong, 6/8 moderate
Outcomes of studies: 3/8 positive, 2/8 mixed/limited/some evidence, 3/8 negative

A large hospital-based study provided moderate evidence of a small effect for significantly decreased depression scores after patients played a two-player strategy board game GO (aka Igo, Baduk, Weiqi) for either 1 or 2 hours each day, when compared with usual care over 6 months[89]. Daily small group mah-jong in a medium multicentre RACS study of people with very mild to mild dementia and moderate depressive symptoms showed moderate evidence of significantly decreased depression scores after 12 weeks. Changes were not maintained at 3-month follow-up and no effect was found for tai chi or handicraft sessions[90].

A large RCT provided moderate to strong evidence of increased duration of observed happy mood, but no significant effect on depression scores, for humour therapy delivered by trained performers (ElderClowns) with trained RACS staff (LaughterBosses) in 9-12 sessions over 13 weeks (SMILE)[91-93]. Two drama sessions (Scripted-IMPROV) set in community day centres and RACS in which participants played main character roles demonstrated limited evidence of decreased depression scores for a subset of people with baseline scores indicating probable depression in a large study of moderate quality[94]. A medium community-based study compared 2-hour sessions delivered by professionals of either choral singing or painting. Moderate evidence of a small effect was found for painting sessions after 12 weeks, but no effect for choral singing and no significant difference between groups 4 weeks after interventions ceased[95].

In contrast, a medium community-based study provided moderate evidence that neither participating in guided art tours, nor making art in a studio, nor visiting a museum independently weekly over 6 weeks lowered depression scores in people with mild to moderate dementia[96]. Similarly, 3 weeks of tri-weekly one-to-one sessions of preferred leisure activities in a large RACS study of people with mild to severe dementia provided moderate evidence of no observed changes in sadness[97]. A medium RACS study also provided strong evidence of no

effect for various one-hour small group 'play activities' requiring physical and cognitive effort as well as cooperation or for book and magazine reading sessions after 8 weeks[98]. A study conducted before 2012 showed no significant benefit for activities tailored to match the person with dementia's capabilities, in a study of strong quality[99].

Cognitive rehabilitation/stimulation
Quality of research studies: 7/23 strong, 16/23 moderate
Outcomes of studies: 5/23 positive, 3/23 mixed/limited/some evidence, 15/23 negative

Two medium community-based studies provided mixed results for cognitive behavioural therapy (CBT)[100, 101]. Moderate evidence was demonstrated for decreased depression scores in people with mild to moderate dementia and anxiety after 10 sessions of manual-based CBT delivered by clinical psychologists over 15 weeks[100]. Strong evidence indicated that weekly in-home CBT sessions over 3 months followed by weekly phone sessions over a further 3 months did not significantly decrease depressive symptoms and were no more effective than usual care[101].

Three medium cognitive rehabilitation studies of moderate quality reported some positive outcomes for people living in the community. An RCT reported decreased depression scores after 6 weeks and a small positive effect for paper and pencil memory training, and a large positive effect for a wearable camera memory aid, but no significant effect for a written journal[102]. Some evidence supported the benefit of weekly goal-oriented cognitive rehabilitation and/or restorative approaches[103]. A 12-week study provided evidence of decreased depressive symptoms but no benefit over usual care for multidisciplinary, twice weekly 5-hour sessions of cognitive and physical training/stimulation plus twice-weekly carer psychoeducational workshops for psychogeriatric clinic outpatients with mild AD[104].

Two medium RACS studies provided moderate evidence of a small effect for small group cognitive stimulation sessions of games and creative activities[105] and sessions conducted by trained facilitators[106] compared with usual activities. Strong evidence was reported for reduced depression scores after six weeks of cognitive training and creative expression in a large RACS trial of people with mild-severe dementia, but the effect was not maintained at 1-month follow-up[62]. A medium community-based study of people with mild-moderate dementia and major depression provided moderate evidence of a medium effect for weekly home-based Problem Adaptation Therapy (PATH) compared with supportive therapy after 3 months[107].

Two medium studies of moderate quality conducted in RACS and hospital settings showed no evidence of positive outcomes for brain activating rehabilitation[108, 109]. Another medium study provided moderate evidence of no benefit over usual care after 12 weeks of twice-weekly individual or small group cognitive rehabilitation sessions involving reminiscence, reality orientation and physical exercise[110]. Two very large community-based RCTs provided strong evidence of no significant effects for individual goal-oriented cognitive rehabilitation of everyday function nor for combined cognitive behavioural/cognitive rehabilitation sessions[111-113].

Five studies ranging in size from small to large provided moderate evidence of no benefit. Small group cognitive stimulation sessions in RACS[114, 115], individual cognitive stimulation sessions in RACS[116], group sessions in the community[117] and group sessions plus usual pharmacotherapy in the community[118]. One very large community-based study provided strong evidence of no effect for in-home individual cognitive stimulation sessions delivered by carers[119].

Likewise, one very large and one medium community-based study provided strong evidence of no significant effect for cognitive training and cognitive training with cholinesterase inhibitors (ChEI) for people with AD, respectively[120, 121]. Twice-weekly group mindfulness sessions indicated moderate evidence of no effect on RACS residents' depression scores after five weeks in medium study[122]. A study undertaken prior to 2012 demonstrated evidence for the effectiveness of a cognitive-behavioural intervention of pleasurable events as well as carers' problem solving and environmental adaptation strategies. Beneficial effects were reported after 9 weeks and maintained at six-month follow-up[123]. A further study found no benefit for a cognitive-behavioural family intervention[124].

Physical Activity
Quality of research studies: 5/12 strong, 7/12 moderate
Outcomes of studies: 1/12 positive, 2/12 mixed/some evidence, 9/12 negative

A large Tai Chi study indicated strong evidence of a small effect after 10 months of 20-minute Tai Chai sessions three times weekly for RACS residents with mild dementia[125]. Similarly, strong evidence indicated a small effect for decreased depression and negative mood scores after 3 months of easy dance movement sessions modified for standing or sitting community-based people with mild dementia. However, this very large study showed no effect for instructor-led, non-dance exercise sessions and no sustained benefit from dance sessions at 3- and 9- months post-intervention[126].

A very large RACS study of moderate quality compared 45-minute tri-weekly sessions of high-intensity group functional exercise and OT-led group recreational activities without exercise. Only a subgroup of residents with high levels of depressive symptoms at baseline showed significantly decreased depression scores for both interventions after 4 months and effects were still present at 12 weeks post-intervention[127].

Most studies of physical activities reported no effect. A very large study provided strong evidence of no effect for moderate to high-intensity small group aerobic exercise sessions after 4 months for people with mild AD[128]. Similarly, two medium community-based studies reported moderate evidence of no effect on depression scores. The interventions were aerobic exercise or non-aerobic stretching and toning sessions for people with early AD over 24 weeks[129] and daily at-home carer-supervised exercise and walking in people with mild-moderate AD[130].

Twice-weekly physiotherapist-led exercise delivered in a group at a day centre or individually in-home provided moderate quality evidence of no effect on depression scores for people with mild to moderate dementia after 12 months in a very large study[131]. A medium study of people with AD, DLB, PDD or mixed dementia provided moderate evidence that depression scores did not improve, but significantly increased, for all intervention groups after 12 weeks of twice-weekly 45-minute sessions of either chair yoga, customised participatory music or gentle chair-based exercise[132].

Two very large RACS studies provided strong evidence of no effect on depression scores when compared with recreational activities. Interventions were 15-minute daily recumbent cycling sessions over 15 months[133] and high-intensity small group functional strengthening and balance exercise sessions over 12 weeks[134, 135]. Six months of multicomponent physical activity training sessions with or without ADL training demonstrated moderate evidence of no change in depression in a large study of RACS residents with mild to severe dementia[136]. A small study provided moderate evidence of no effect for residents with mild to moderate dementia and depression after walking and talking with a volunteer one-to-one weekly over 8 weeks, and no difference when compared with individually tailored sessions of pleasant activities[137]. A very large RCT conducted before 2012 reported significantly reduced depression scores on the CSDD, but not the HAM-D, for exercise plus behavioural management training for carers (Reducing Disability in AD) after 3 months[138].

Animal-assisted interventions
Quality of research studies: 1/8 strong, 7/8 moderate
Outcomes of studies: 2/8 mixed/limited/some evidence, 6/8 negative

Two medium RACS studies reported moderate evidence of decreased depression scores for interactions with a therapy dog and handler. Interventions provided weekly visits to people with moderate to very severe dementia over 10 weeks[139] and twice weekly small group sessions for people with severe dementia over 12 weeks[140].

By contrast, two medium studies of more structured therapeutic sessions in people with mild to moderate dementia reported moderate evidence of no decrease in depressive symptoms. Biweekly 1-hour animal-assisted therapy dog sessions for memory clinic attendees did not outperform usual home support over 8 months[141] and small group sessions incorporating

individual therapy dog interaction demonstrated no benefit over similar sessions with a human therapist only in RACS residents[142].

No study of the companion robot seal *Paro* provided evidence that the intervention significantly lowered depressive symptoms, or provided greater benefit than usual care or activities, for people with all stages of dementia living either in the community or in RACS[143-146].

Sensory interventions
Quality of research studies: 5/5 moderate
Outcomes of studies: 1/5 positive, 1/5 some evidence, 3/5 negative

Five medium studies provided limited or mixed outcomes from moderate quality evidence. One RACS study of aromatherapy massage delivered weekly over 8 weeks to people with moderate to severe dementia and agitation or depression reported a significant decrease in depression scores for aromatherapy massage compared with usual activities[147]. A study of twice-weekly *Snoezelen*® multisensory room sessions compared with individualised music over 8 weeks reported decreased depression scores for both interventions at 16 weeks with no difference between groups[148]. Lavender oil aromatherapy massage showed no improvement in observed negative affect scores in RACS residents with moderate-severe dementia over 2 weeks[149]. Studies of *Snoezelen*® multisensory rooms in RACS provided no evidence of positive outcomes or additional benefit when compared with one-to-one activity sessions with a therapist[150-152].

Touch therapies
Quality of research studies: 1/2 strong, 1/2 moderate
Outcomes of studies: 1/2 positive, 1/2 limited evidence

Ear acupressure sessions 5 times weekly provided moderate evidence of significantly greater benefit for depressive symptoms than lower limb and back massage delivered by a physiotherapist in a large study of RACS residents with moderate to severe dementia, after 3 months[153]. A small study of community-dwelling people with mild to moderate dementia and depression provided limited evidence of a significant decrease in depression scores after 10 months of tri-weekly therapist-delivered Shiatsu treatment with mild-intensity physical exercise when compared with the exercise sessions only[154].

Education/training
Quality of research studies: 7/12 strong, 5/12 moderate
Outcomes of studies: 3/12 positive, 9/12 negative

A very large RCT compared usual care and dementia/BPSD education for staff with and without supervised education: Targeted Interdisciplinary Model for Evaluation and Treatment of Neuropsychiatric Symptoms (TIME)[155]. Strong evidence indicated a small effect for the TIME staff education and implementation in RACS residents with mild to severe dementia after 12 weeks with a significantly greater decrease compared to usual care and education[155].

A medium RACS study provided moderate evidence of a positive effect for manual-based dementia workshops with individual supervision for direct care staff: Staff Training in Assisted Living Residences (STAR)[156] with a medium effect on depression scores after 8 weeks in people with moderate to severe dementia[156]. Compared with usual care, strong evidence supported staff/carer sleep education sessions, conducted by an experienced trainer in RACS, to develop and implement individualised sleep plans in a medium RCT for people with mild to severe dementia and disturbed sleep[157].

Three community-based interventions for carers reported no benefit. A medium study of moderate quality compared DVD-based training in memory and communication for care partners of people with moderate to severe dementia to usual care (RECAPS & MESSAGE)[158]. A large study of strong quality compared weekly psychologist-led multicomponent training sessions on sleep disturbances to usual care in people with mild to severe dementia and sleep disturbances (DREAMS-START)[159]. A very large study of strong quality compared strategies provided to carers of veterans with mild to moderate dementia and pain/aggression: Preventing aggression

in veterans with dementia (PAVeD)[160] to enhanced usual primary care[161].

Similarly, a medium study comparing usual activities with twice-weekly training in Tai Chi, traditional Chinese calligraphy and drawing reported moderate evidence of no change in GDS scores in community-dwelling people with AD and no between-group difference after 6 weeks[162]. A practice-based educational intervention for GPs led by a peer educator including patient audits with feedback did not change GDS scores in primary care patients and did not perform better than patient audits without feedback and RACGP guidelines provided by mail, in a very large, one-year cluster RCT of strong quality[163].

Two large trials of an educational intervention encouraging dementia-specific facility care workers to engage residents with moderate to severe dementia in activities to optimise function reported moderate evidence of no benefit for depressive symptoms after 6 months (Function-Focussed Care)[164, 165]. Two very large RACS cluster RCTs provided strong evidence of no benefit when compared with usual care. Interventions were OT-delivered staff training, including environmental assessment at 4- and 12 weeks post-intervention[166] and nurse-educator facilitated education on incorporating reminiscence into care plans: Dementia Education Programme Incorporating Reminiscence for Staff (DARES)[167] which *increase*d depression scores at 12-16 weeks post-intervention[168].

Models of care
Quality of research studies: 3/14 strong, 11/14 moderate
Outcomes of studies: 2/14 positive, 3/14 mixed/limited/some evidence, 9/14 negative

A medium RACS study provided moderate evidence of a greater decrease in CSDD scores for people with moderate to severe dementia and major depression after 15 weeks of multidisciplinary specialist mental health consultation, including psychosocial and medical recommendations, compared with usual care[169].

Mixed outcomes were reported in a very large study of moderate quality. A medium positive effect was found for comprehensive stepwise training for multidisciplinary teams working on RACS units and implementation of the STA OP! protocol[170, 171] after 3 months compared with dementia/pain management training[172]. However, moderate evidence of no change in QUALIDEM negative affect scores post-intervention and at follow-up was also reported[170]. Staff dementia training supplemented with the VIPS practice model of person-centred care (VPM)[173] was more effective at decreasing depression scores than dementia training alone and outperformed training supplemented with Dementia Care Mapping (DCM)[174] in another very large study of moderate quality after 10 months[175].

Two community studies provided moderate evidence for significant decreases in depression scores in people with mild dementia. Interventions were individualised care consultations delivered over two years via local Alzheimer Association chapters in a very large study, Project Learn MORE[176]; and small weekly social worker-led, structured support group sessions over 10 weeks in a medium study[177].

In contrast, three very large community-based studies also demonstrated no significant decrease in depression scores. Strong evidence of no benefit was provided in people with MCI or mild to moderate dementia after 18 months of individualised dementia care delivered by an interdisciplinary team, Maximising Independence at Home (MIND)[178]. The evidence reported was moderate after 12 months of in-home OT case management and support for people with mild dementia[179] and a tailored, guided shared house intervention after 12 months[180].

A very large RACS study provided strong evidence of no effect on depression scores from staff training and application of either person-centred care (PCC), person-centred environment (PCE) improvements, or a combination of PCC and PCE[181]. Small to very large, non-randomised studies of moderate quality for visual and auditory environmental changes in RACS[182] and the use of small-scale care settings versus traditional larger-scale units[183, 184] also reported no effect on depression scores. A multidisciplinary/multicomponent approach, Act in Case of Depression (AiD)[185] in a very large RACS study showed moderate evidence of no change in depression

scores. A medium community-based trial provided strong quality evidence of no positive effects on depression for psychiatric assessment and therapy after six months[186]. RACS research from before 2012 reported no significant change in depression for psychiatric management compared with psychogeriatric consultation and standard care[187] or collaborative care[188].

Multicomponent interventions
Quality of research studies: 9/19 strong, 10/19 moderate
Outcomes of studies: 2/19 positive, 4/19 mixed/limited/some evidence, 13/19 negative

A medium study of in-home multicomponent OT, including problem-solving tasks and recreational/ADL activities, provided strong evidence for decreased CSDD scores after 16 weeks[61]. Two RACS studies reported moderate evidence of large positive effects. A medium study compared usual care with twice-weekly 30-minute group activity sessions incorporating question-asking reading (QAR), reminiscence, cognitive behavioural therapy, environmental supports and behaviour programs for people with mild to moderate dementia and depressive symptoms over 6 weeks[189]. A large study compared the efficacy of three different weekly interventions: cognitive stimulation, reminiscence and aromatherapy massage for people with moderate to severe dementia and depressive symptoms. Aromatherapy massage only showed a significant decrease in CSSD scores after 10 weeks[190].

A medium hospital-based trial compared twice-weekly sessions of neuropsychologist-delivered cognitive training, music therapist-delivered music therapy and a neurologist-coordinated neuroeducation program for people with mild to moderate dementia prior to their discharge home. Moderate evidence indicated reduced depression scores for all interventions after 12 weeks, but benefits were not maintained for any group after 3 months[191].

Limited, moderate quality evidence of a significantly greater decrease in depression scores was demonstrated in a small RCT of in-home sessions incorporating future planning and reminiscence to maximise coping, Preserving Identity and Planning for Advance Care (PIPAC)[192] compared with empathic listening and support administered over the phone. Moderate quality evidence also indicated a small-to-medium effect of significantly greater decrease in NPI depression scores in people with moderate dementia, but no additional benefit in subjective mood scores, after 8 weeks of twice-weekly 30-minute interactive multicomponent therapy compared with usual care in a large RACS study. Sessions included breathing exercises, music/massage, singing, dancing and/or playing instruments to stimulate senses and encourage social interaction[193].

A very large RACS study provided strong evidence of no significant change in depression scores of veterans with mild-severe dementia after 9 weeks of learning eating techniques using Montesorri-based activities aimed at increasing nutritional status and BMI[194]. Another very large trial of a multidisciplinary, structured multicomponent tailored intervention including individual counselling for people with mild to moderate dementia and their carers, education and group meetings provided strong evidence of no significant effect on depressive symptoms compared with usual care at 12 months[195].

Studies of community-dwelling people recently diagnosed with dementia reported no significant decrease in depressive symptoms. Weekly 75-minute small group sessions of dementia education, psychotherapy and handouts delivered by memory clinic nurses over 10 weeks for people who had received a diagnosis of AD, VaD or LBD showed no benefit in a large study of moderate quality (Living Well with Dementia)[196]. A very large trial of the multifaceted Danish Alzheimer Study intervention (DAISY) provided strong evidence of no effect after 12 months of psychosocial counselling, education and support compared with usual structured follow-up support[197, 198].

Three community RCTs also provided strong evidence of no significant change in depressive symptoms for multicomponent interventions featuring physical activity when compared to usual support. A culturally and linguistically adapted home-based exercise, psychoeducation and support program based on the Reducing Disability in AD (RDAD)[138] intervention for people with moderate dementia showed no benefit in a very large study after 3 months[199]. A large trial

showed no benefit in people with MCI or mild-to-moderate AD for a twice-weekly 3.5 hour cognitive and motor stimulation program delivered over 3 years[200] and a small trial of people with mild-to-moderate AD found that 4-hour physical activity, cognitive stimulation and socialisation sessions 5 times weekly over 3 months did not change CSDD scores however, scores increased in people receiving usual care[201].

Two very large trials of moderate quality reported no beneficial outcomes. Individualised, multicomponent intensive rehabilitation for people with mild to moderate dementia recently discharged from hospital to RACS did not outperform usual therapy after 3 months[202]. Inpatient group therapy for care dyads including cognitive and social rehabilitation for people with mild to severe dementia and psychoeducation for their care partners for 3 to 4 weeks did not significantly change depression scores at 3 and 6 month follow-up compared with baseline[203].

Two RACS studies provided moderate evidence of no change in depression scores. A medium study of a multisensory stimulation, reminiscence and physical activity group intervention (Sonas)[204] did not outperform usual care in people with moderate to severe dementia over 7 weeks[204b]. A facility-level quality improvement program Music and Memory involving staff training, support resources and individualised music playlists delivered via iPods provided no benefit over 6 months in a very large study of 196 RACS[205].

Two very large, RACS cluster RCTs provided strong evidence of no benefit after 9 months.

STRATEGIES/OUTCOMES

- *Cathy's medical history indicates that she has previously been diagnosed with depression in her 20s. Her family reports that, in general, she has been happier and more emotionally stable since she transitioned.*
- *With the progression of dementia, Cathy has become more inclined to focus on unpleasant memories and express her distress around previous traumatic and discriminatory experiences.*
- *Staff report a recent incident with a female resident which was particularly distressing for Cathy when she used the communal female toilet near the dining room at lunchtime. The other resident loudly expressed her displeasure at sharing the female toilets with Cathy, complaining to others nearby. One resident responded with "People like that shouldn't be allowed to live here". Cathy didn't return to her table to finish her lunch but headed back to her room in tears.*
- *Cathy's GP undertook a medical assessment and medication review. A trial of antidepressant medication was considered. At a family conference, it was explained that a trial of psychosocial interventions are recommended initially. Family were also made aware that it may be some weeks before the potential benefits of an antidepressant are evident and Cathy may experience side effects but hopefully, these will resolve.*
- *Some care staff have little knowledge of depression that can occur with dementia, trauma-informed care or the special needs of transgender people. Training was provided to enable staff to develop strategies that may assist in providing respectful care and support to Cathy and other residents as needed. It was highlighted that reminiscence activities may not be appropriate for people with trauma in their past.*
- *During the training sessions it became evident that some staff felt that caring for Cathy conflicted with their cultural and/or religious beliefs. One staff member was distressed by pressure from her family to avoid Cathy. Additional individual sessions were arranged to consider appropriate strategies and support for affected staff members.*
- *The recreational officer arranged for Cathy to have additional individual time in a quiet area for activities she previously enjoyed such as listening to her favourite music, nail care, hand massage or spending time with the facility's cat. Some of Cathy's favourite meals were incorporated into her diet to encourage her appetite.*
- *Staff were alerted by the family that LGBTIQA+ Pride month was approaching and that this was a special time for Cathy. Her family planned outings to enable Cathy to be part of some of the Pride events and the facility arranged some inclusive activities relevant to Pride month. Many of the other residents responded positively and Cathy enjoyed the additional, culturally relevant attention.*
- *Cathy's family and care staff indicated that her mood was lifting somewhat and her appetite had improved, particularly when family members brought Cathy some favourite treats. Cathy's depressive symptoms will continue to be monitored with a view to reassessing her potential need for medication.*

Various combinations of staff training and implementation of person-centred care (PCC), group activities, social interactions, exercise, and antipsychotic review based on NICE Dementia Guidelines, Well-being and Health for People with Dementia (WHELD)[206] demonstrated no change or significantly *increased* depression scores in residents with mild to severe dementia[207]. The WHELD intervention plus staff coaching, supervision and review reported no significant benefit compared with usual care in residents with moderate to severe dementia[208]. A significant decrease was reported on one measure of depression, but not another, in a medium study of an insomnia intervention including carer education, daily walking and increased light exposure reported before 2012 [209].

Summary

Moderate to strong support is demonstrated for selected psychosocial and environmental interventions. An increasing number of psychosocial interventions have been reported as effective in addressing depression in dementia and these positive findings are in line with expert consensus recommendations for psychosocial interventions tailored to the needs and life contexts of people with depression in dementia. Music and reminiscence interventions, respectively, provided the highest number of studies with positive outcomes or evidence of at least some benefit. Successful group and individual approaches were balanced in both categories. Efficacy may be context dependent but trials of small group music therapy with instruments and/or singing, individualised/personalised music, incorporating music in everyday care and music with movement indicated beneficial effects. The cognitive rehabilitation/ stimulation category included the next highest number of studies indicating benefit. Effective approaches were undertaken in all care settings and included CBT, memory support, games, creative expression and Problem Adaptation Therapy (PATH).

While multicomponent interventions provided the best strength evidence with nine of 19 strong quality studies, 13 studies in this category overall reported negative outcomes. Trials were heterogeneous, comparing multiple different approaches and components within each study were often varied. Studies providing positive outcomes or evidence of some benefit included formal and informal carers and were undertaken in RACS, hospital and community settings.

Studies of models of care assessed multiple diverse care models. Most studies did not show benefit, including three RCTs which provided strong evidence of negative outcomes. Effective RACS and community studies included aspects of specialist assessment/consultation, multidisciplinary care, structured support and PCC. Only three of 12 studies of education and training indicated significantly positive outcomes, but more than half of studies overall provided strong quality evidence. Trials indicating benefit included supervised, manual-based education with implementation in RACS.

Nine of the 12 physical activity studies reported no effect on depressive symptoms regardless of exercise type, intensity or location and nearly half of the studies were of strong quality. People with mild dementia may benefit from synchronised or meditative movement and dance as part of group exercise.

Studies of therapeutic recreational interventions offered mixed approaches and outcomes. Small to medium positive effects indicate that playing strategy games regularly as well as interactive, creative and performing art interventions may be beneficial. Animal-assisted interventions were largely not beneficial. Two studies of dog-assisted therapy reported some or limited evidence of a positive effect and no trials of a companion robot demonstrated benefit. Sensory intervention studies focused on aromatherapy massage or multisensory rooms. Single studies provided limited evidence for each. Only two studies of touch therapies were of sufficient quality for inclusion, providing limited evidence for ear acupressure and Shiatsu treatment. See *Appendix 2* for interventions reported above.

Biological and pharmacological interventions

Most biological/pharmacological studies were undertaken with community-dwelling people.

Analgesic medications
Quality of research studies: 1/2 strong, 1/2 moderate
Outcomes of studies: 1/2 some evidence, 1/2 negative

Pain management is always a first line consideration in addressing any BPSD. A stepwise pain management protocol found moderate evidence of a significantly greater decrease in NPI depression scores, with a small effect, for paracetamol as a first step before pregabalin, buprenorphine and/or morphine compared with usual care, in a very large cluster RCT of RACS residents with moderate-severe dementia[210, 211]. A very large RACS study of strong quality found no significant change in depression scores for paracetamol or buprenorphine compared to placebo and no difference between groups. The risk of adverse effects for buprenorphine was noted[212].

Antidepressants
Quality of research studies: 4/6 strong, 2/6 moderate
Outcomes of studies: 1/6 some evidence for sertraline, 1/6 some evidence for escitalopram 4/6 negative

High quality clinical practice guidelines and meta-analyses of treatment efficacy do not support the use of antidepressants for mild to moderate depression in dementia unless indicated for pre-existing severe depression. Psychosocial interventions are recommended as first line treatment and antidepressants should be considered where symptoms are unresponsive[58, 60, 213-215]. Findings reported here are based on comparisons of group data of antidepressants versus placebo. Where a person's quality of life is impacted, it is important to be aware that individual responses may vary.

One medium study of moderate quality reported a significant decrease in HRSD depression scores for people with AD and major depressive disorder with 25-50 mg/day of sertraline, but no change in scores for desipramine or venlafaxine at 12 weeks[216]. While strong evidence from a large study indicated a significantly greater decrease in CSDD scores for escitalopram compared to placebo at 28 weeks for dementia clinic patients with mild to moderate AD, no effect was found at study completion of one year[217].

Strong evidence from two very large studies of people with probable AD and depression indicated no significant difference between placebo and either sertraline or mirtazapine at 39 weeks[218, 219]. A large study of people with moderate AD reported no significant change in depression scores nor difference from placebo at 12 weeks after up-titrating escitalopram from 5mg/day to a maximum of 15mg/day[220]. Strong evidence of *increased* depressive symptoms was found with placebo replacement of SSRIs: escitalopram, citalopram, sertraline or paroxetine compared to continued treatment at 25 weeks in a very large multicentre withdrawal RCT of RACS residents with mild to severe dementia, without depressive disorder[221].

Overall, no good evidence was found for antidepressants in research undertaken prior to 2012. Although one small study indicated a significant reduction in severity of CSDD scores compared to placebo at 3 and 12 weeks[222], a very large study found no benefit for sertraline or mirtazapine[223] and a medium study reported no difference for sertraline compared to placebo[224]. Further, two very large studies found no significant difference between placebo and fluoxetine[225], or between placebo and venlafaxine[226].

Combination therapy of an antidepressant with an antipsychotic has been recommended for psychotic depression in dementia[227] however, no studies for combined therapy were found for this review. Sertraline or mirtazapine did not outperform placebo in a very large RCT conducted before 2012[223]. Likewise, no significant difference from placebo was found for sertraline[224], venlafaxine[226] or fluoxetine[225] in three medium trials or a very large sertraline RCT[228]. One small RCT of sertraline did show significantly decreased depression scores[222].

Psychostimulants
Quality of research study: 1/1 moderate
Outcome of study: 1/1 positive

A medium RCT provided moderate quality evidence that methylphenidate outperformed placebo in improving CSDD scores at 12 weeks for community-dwelling male military veterans with AD; there were more side effects with methylphenidate[229].

Brain stimulation
Quality of research studies: 2/6 strong, 4/6 moderate
Outcomes of studies: 2/6 positive, 3/6 some evidence, 1/6 negative

A small, community RCT provided moderate evidence of significantly decreased CSDD depression scores for in-home blue-white, fluorescent light when compared with usual light, but not with yellow-white control light, during daytime waking hours after 6 weeks in people with mild to severe dementia and disturbed sleep[230]. Consistent daytime light therapy tailored to maximally affect the circadian system provided moderate evidence of a significant decrease in depressive symptoms with a small to medium effect at 4 weeks compared with baseline and compared to lower level placebo lighting in a medium trial of RACS residents with dementia and sleep problems[231]. Similarly, a medium prospective study of time-limited exposure to therapeutic bright light units reported moderate evidence for a significantly greater decrease in depression scores with a large effect for bright light exposure at 8 weeks compared with placebo low-level light exposure in RACS residents with mild to severe dementia[232, 233].

Bilateral 20 Hz high frequency repetitive transcranial magnetic stimulation (rTMS) provided strong evidence for a significantly greater decrease, with small to medium effect, in GDS scores compared with low 1 Hz frequency and sham treatments at 3 months post-intervention in a medium community RCT of people with mild to severe AD[234].

Two medium RCTs compared repeated transcranial direct current stimulation (tDCS) with sham tDCS. Ten sessions of active bilateral 2mA anodal tDCS compared with sham tDCS for 20 minutes per side, in people with probable AD, demonstrated moderate evidence for decreased CSDD scores for both groups at 2 weeks but a significantly greater decrease for active tDCS was reported[235]. A second trial of 6 sessions of 2mA anodal tDCS over the left dorsolateral prefrontal cortex only in people with moderate AD reported strong evidence of no difference in depression scores between active tDCS and sham treatment[236].

Literature on ECT for those with dementia and depression is limited. However, it has been reported as effective and well tolerated[237, 238]. While ECT is recommended as a second line treatment for people without dementia and it has been used in people with dementia, the evidence for this is sparse. ECT is typically reserved for severe or urgent (e.g. danger from malnutrition, suicidality), treatment resistant or psychotic depression[239]. No ECT trials met our criteria for inclusion in this review. An RCT undertaken before 2012 demonstrated a small but significant reduction in depressive symptoms for whole-day bright light therapy[240].

Cholinesterase inhibitors (ChEIs) and/or memantine
Quality of research studies: 1/7 strong, 6/7 moderate
Outcomes of studies: 1/7 positive, 1/7 some evidence, 5/7 negative

Overall evidence for ChEIs and memantine is largely negative and in all studies, depression was reported as a secondary outcome. Rivastigmine patch, alone and combined with weekly cognitive stimulation significantly decreased HAM-D and GDS scores with large effect compared to baseline and the combination treatment outperformed rivastigmine only in people with moderate AD at 6 months in a large RCT of strong quality[241]. A very large open-label observational study compared ChEI switch from oral donepezil or galantamine to rivastigmine patch with a switch from rivastigmine patch to non-rivastigmine oral. Moderate quality evidence demonstrated a significant improvement in NPI depression subscale scores for the switch from oral ChEI to rivastigmine patch at 6 months, in people with mild to moderate AD[242].

A large retrospective chart review 6 months before and 6 months after any switch between rivastigmine, galantamine, and/or donepezil provided moderate evidence of no change in GDS scores for any ChEI switch in people with mild to moderate AD[243]. Outcomes of other large to very large, moderate quality retrospective studies showed no benefit for people with all levels

of AD from introducing galantamine or switching from other ChEIs to galantamine[244], nor for galantamine in combination with memantine[245, 246], nor evidence for the efficacy of donepezil or memantine monotherapy treatment or in combination with other ChEIs[245, 246]. While adding a ChEI to antidepressant treatment has been recommended as a second-line option for depression in dementia[227], no studies for combined therapy were found for this review.

Traditional medicines
Quality of research studies: 2/2 moderate
Outcomes of studies: 1/2 positive, 1/2 some evidence

Polyherbal treatment *Bacopa monnieri*, *Hippophae rhamnoides* and *Dioscorea Bulbifera* extracts outperformed donepezil monotherapy in significantly decreasing GDS scores, with a large effect, in community-dwelling people with severe AD at 12 months in a very large RCT of moderate quality[247]. A medium, open-label study of Ninjin'yoeito (NYT) extract traditional Japanese medicine add-on to donepezil compared with donepezil alone provided moderate evidence of a significant decrease in depression scores with a small effect for combined treatment when compared with donepezil monotherapy, but not baseline, in clinic outpatients with mild to moderate AD at 24 months[248].

Other pharmacological/biological
Quality of research studies: 1/4 strong, 3/4 moderate
Outcomes of studies: 1/4 positive, 3/4 negative

Two very large studies of moderate quality assessed citicoline, a cholinergic supplement, in combination with ChEIs in people with moderate dementia over nine months. Citicoline added to participants' highest tolerated dose of rivastigmine patch compared with rivastigmine only, demonstrated a significant decrease in GDS scores for both groups[249]. In contrast, no significant change in depression scores was reported for citicoline in combination with any ChEI when compared with baseline and no difference between groups when compared with ChEI alone[250].

A medium RCT provided strong evidence of no effect on depression for subcutaneous etanercept, an autoimmune drug, in people with mild to moderate AD compared to placebo at 24 weeks[251]. Melatonin 5mg/night did not outperform placebo in depressive symptoms at 8 weeks for outpatients with mild to moderate dementia and sleep alteration, in a large RCT of moderate quality[252]. A study conducted prior to 2012 also showed no benefit for melatonin[240].

Summary

Few studies indicate effective pharmacological/biological treatments. Limited evidence was provided that stepwise pain management was effective. Antidepressants are generally not recommended for depression in dementia. If indicated, there is some evidence that escitalopram or sertraline may provide short-term benefit. Blue-light, tailored daytime light and bright light exposure outperformed usual or placebo lighting. The degree of benefit found for light therapy may be dependent on climate and exposure to sunlight which is possibly less relevant in the Australian setting. Single studies of brain stimulation therapies found higher frequency, bilateral rTMS and direct current stimulation offered greater benefit than lower frequency and unilateral applications.

Cholinesterase inhibitors (ChEIs) and memantine provide very limited evidence of efficacy although this category reported the highest number of studies. Two single studies indicated benefit from switching to rivastigmine patches from an oral ChEI and rivastigmine patches with and without cognitive stimulation. However, other studies based on retrospective chart reviews reported no benefit for combinations of, or switches between, different ChEIs or memantine, including rivastigmine. Outcomes from retrospective chart reviews should be viewed with caution as these lacked randomisation.

Single studies reported limited evidence for other pharmacological agents including a study which indicated reduced depression with methylphenidate. Two single studies reported evidence that traditional medicines added to donepezil outperformed donepezil monotherapy. One study of citicoline, a cholinergic supplement, reported positive outcomes with and without

rivastigmine patches whereas, a second study found no benefit with or without any ChEI. Other pharmacological treatments such as melatonin and etanercept, an autoimmune drug, provided no evidence of decreased depression.

Wherever possible, the use of symptomatic, pharmacological agents, when required for treatment of depression should be time limited, closely monitored, reviewed, reduced and/or discontinued when indicated. Where antidepressant medication is indicated, the person with dementia and their family may need an explanation as to the time required before potential benefits are evident[213]. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[57, 59]. For example, where the person presents with depression and agitation, treating the depression may also reduce agitation. See *Appendix 3* for interventions reported above.

Limitations

While intervention studies for addressing depression in people with dementia are plentiful in the literature, many have limitations regarding methodology and sampling. These impact outcomes and/or potentially limit the generalisability of the results. Some studies provided insufficient data to calculate effect sizes, limiting interpretation of the degree of benefit provided by interventions. Problems also occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. Long-term follow-up to determine the sustainability of the benefits after interventions ceased was also limited. Of the 44 psychosocial intervention trials that reported follow-ups conducted between one month and 24 months post-intervention, only 10 indicated that benefits were maintained. Only two biological/pharmacological studies conducted any follow-up.

Conclusions/Principles of care

In summary, depression in dementia has significant consequences. Best evidence suggests that music and reminiscence interventions can help to reduce depression. Synchronised movement to music and meditative movement such as Tai Chi may also be helpful. Cognitive behavioural therapy showed mixed results. The evidence presented indicates manualised education and care programs need a minimum threshold of input and supervision to be effective. Creative and performing art as well as multicomponent interventions may provide benefit. Specific applications of cognitive rehabilitation, cognitive stimulation, memory aids and psychological therapies demonstrated some benefit, which is further supported by a Cochrane review[253]. The evidence for dog-assisted therapy, strategy games, group exercise, multisensory rooms, aromatherapy massage and acupressure is largely based on single studies and hence, cannot be recommended.

Many effective interventions were also social activities, and this aspect may have a role in reducing depressive symptoms. The National Institute for Clinical Excellence Guidelines[58] indicate that interpersonal therapy may be helpful in the treatment of those with depression in dementia. A multidisciplinary, individualised and multifaceted approach, tailored to the needs and the life contexts of people with dementia is recommended.

Strong evidence indicated limited efficacy for antidepressants. While comparing outcomes of controlled trials for people with depression and dementia on an antidepressant or placebo have largely found no significant difference, some individuals may have positive outcomes. Studies provide evidence that light therapy techniques may decrease depressive symptoms. Other approaches such as stepwise pain management, brain stimulation therapies, methylphenidate, and ChEIs alone or in combination with other therapies, and traditional medicines have occasional reports of benefit and cannot be recommended on the basis of these alone. If pharmacological or biological treatment is indicated, this should be accompanied by psychosocial and/or environmental interventions. As always, the potential benefits to the person with dementia must be weighed against the potential side effects of pharmacological treatments.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

9. Disinhibition

MODULE 9: Disinhibition

Key messages

- Disinhibition in dementia presents as behaviours associated with a reduced capacity to edit immediate impulsive responses.
- Disinhibition of a sexual nature is particularly challenging.
- Causes of disinhibition include frontal lobe pathology, substance use or other medical/psychiatric conditions e.g., delirium, mania, cerebral event etc.
- In the case of sexual disinhibition, differential diagnosis requires establishing whether the manifested behaviours are 'normal' sexual behaviour for that person or inappropriate behaviours due to impairments in impulse control and moral judgement.
- Disinhibition is common in people with dementia, but rates vary widely, and sexual disinhibition is reportedly less common.
- Addressing disinhibition requires the identification of potentially modifiable factors.
- Strategies to discourage inappropriate behaviours associated with disinhibition and psychoeducation/psychotherapy to support family carers and aged care staff may be helpful.
- Expert clinical guidelines indicate that atypical antipsychotics provide no benefit for disinhibition.
- Sound evidence for pharmacological interventions is lacking; drug toxicity profiles, adverse effects and potential harms must be considered carefully.
- Given the limited evidence available in the literature, strategies for addressing disinhibition must be considered on an individual basis.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

Disinhibition summary

What is disinhibition and what does it look like in dementia?

- Disinhibition in dementia typically occurs with reduced capacity to edit immediate impulsive responses.
- Behaviours include those associated with impaired judgement and reduced awareness of the environment, impaired ability to process emotions and to understand and respond to the thoughts and feelings of others as well as reduced awareness of the impact on others.
- As sexual disinhibition in dementia is particularly problematic, the literature tends to focus on this area.
- Attempts to classify sexually inappropriate behaviours typically differentiate between those that are misplaced in social context and those that would be considered inappropriate in most contexts.

Causes of disinhibition

- Frontal lobe pathology
- Drugs, alcohol
- Social factors and environmental stressors
- Secondary to delirium
- Secondary to a cerebral event
- Secondary to psychiatric syndromes such as mania or psychosis

Differential diagnosis

Consultation with the person with dementia, their family, particularly partners/spouses and formal carers is essential when determining if the presenting disinhibition is indicative of a change, an exacerbation of their pre-morbid behaviour or 'normal' for the person. Where disinhibition presents as a sudden change in the person living with dementia, comorbid psychosis, delirium and/or a cerebral event should be excluded.

Where disinhibition of a sexual nature occurs, it is important to determine if the presenting behaviour is actually 'normal' sexual behaviour for the person presenting inappropriately as impulse control, judgement and/or moral values become increasingly impaired.

Measuring disinhibition

Standardised measures of disinhibition in dementia include the **Disinhibition Scale** and Middelheim Frontality Score (**MFS**). The disinhibition subscale of the Neuropsychiatric Inventory (**NPI**), the NPI-Clinician (**NPI-C**), the Behavioral Syndromes Scale for Dementia (**BSSD**), the Challenging Behaviour Scale (**CBS**) and the CERAD Behaviour Rating Scale for Dementia (**BRSD**) also include relevant items.

Prevalence of disinhibition

- Symptoms reportedly occur in 1% to 61% of people with dementia.
- Rates of sexually disinhibited behaviour are lower, but these behaviours may be underreported.
- The prevalence of disinhibition tends to rise as dementia increases in severity.
- Reports are inconsistent with regard to prevalence ratios in males and females, although more reports indicate a higher prevalence in males.

Effects of disinhibition

Disinhibition in dementia can be associated with negative symptoms, hallucinations or delusions, frustration and subsequent agitation and/or aggression. Disinhibition may also provoke an aggressive response from others. Urinary tract infections, physical trauma and/or sexually transmitted infections are potential consequences of sexual disinhibition. Disinhibition can be stigmatising, is one of the most difficult BPSD for carers, leads to earlier admission to residential aged care, and in more extreme cases anti-social/criminal behaviour (for example, causing injury, sex offending) and/or legal action.

Addressing disinhibition

Developing a behaviour support plan for disinhibition begins with a thorough assessment. One challenge for the clinician is to determine which of the underlying, individual factors driving the disinhibition are potentially modifiable. Possible precipitating factors including medications, comorbid physical conditions, stroke, seizure disorder, comorbid psychiatric condition or environmental triggers should be considered.

Psychosocial and environmental interventions

Suggested strategies are provided in the following areas:

- supportive psychotherapy or education of family carers and/or aged care staff;
- increased, positive contact with family and pets;
- identifying potential triggers, social cues and early indicators;
- modifying environmental aspects, clothing and aged care staff roles;
- providing distraction, redirection and modified learning techniques;
- activities to occupy the person's hands;
- avoiding overreaction or knee-jerk responses that induce shame or humiliation.

Biological and pharmacological interventions

- No randomised controlled trials (RCTs) are currently available.
- Many case studies describe a trial-and-error approach including details of previous unsuccessful attempts.
- Hormonal therapy is controversial as it can be viewed as feminisation or chemical castration of males.
- Expert consensus guidelines note no benefit for antipsychotic medication and recommend against their long-term use.
- The potential benefits to the person with dementia and the safety of others must be weighed against the potential side effects of pharmacological treatments.
- It is crucial to obtain informed consent from the person or from a proxy and in some cases, from official bodies such as the Guardianship Tribunal for the use of hormonal agents.

Limitations

Despite the significant challenges inherent in addressing disinhibition in dementia, sound research to guide clinicians and carers is extremely lacking. Evidence for interventions based on case studies, subscale scores only or clustered BPSD domains which include disinhibition cannot be considered robust.

Conclusions/Principles of care

The limited evidence available suggests that disinhibition must be addressed on an individual basis. Where sexual disinhibition occurs, a dilemma arises in attempts to allow the person with dementia's sexual expression while protecting the safety, rights and dignity of all. The need for a multidisciplinary, individualised and multifaceted approach is stressed.

What is disinhibition and what does it look like in dementia?

Disinhibition is an umbrella term that describes diverse clinical presentations that involve lack of self-control or self-regulation[1]. Disinhibition is described as responsive, hedonic (pleasure-seeking)[2], compulsive, impulsive, self- or other-directed actions. Disinhibition can transgress social and cultural rules and norms, appear inappropriate for the context and/or show disregard for others' needs, feelings, safety and consent[3]. Social inappropriateness may be one of the first recognisable signs of neurodegeneration in dementia[4].

Disinhibition may be objectively observable e.g., a person grabs and eats a whole packet of biscuits in a supermarket, or they may be more subjective and context-specific such as swearing in public. Those presenting with disinhibition typically lack insight regarding their actions and may or may not recognise the consequences for themselves and others. People with lived experience of dementia have described disinhibition in dementia as having a "loss of filter" and speaking or thinking out loud, losing insight and being/feeling "impulsive", "inappropriate", "provocative" and "frivolous". They also describe being/feeling intolerant, easily irritated and even outraged, and recognise that these behaviours can "upset people" and can lead to "embarrassment" and confrontations with others [1].

The DSM-5-TR does not provide a clear definition for BPSD[5]. The World Health Organisation International Classification of Diseases, Eleventh Revision (ICD-11) includes "clinically significant lack of restraint manifested in disregard for social conventions, impulsivity, and poor risk assessment" under a classification for 'behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention' under Neurocognitive disorders, Dementia[6; ICD11, 6D86.5, Parent: 6D86].

Changed behaviours often co-occur in dementia and clusters of specific BPSD can be grouped into domains. Disinhibition is commonly grouped with agitation and aggression[7, 8]. The presence of this BPSD cluster reportedly increases the probability of other BPSD[9]. Interventions targeting BPSD clusters rarely provide insight into strategies that may specifically help to decrease disinhibition. Disinhibition is an early and common presenting feature of younger-onset dementias[10], fronto-temporal dementia (FTD), behavioural variants and other less common neuropathologies including semantic dementia (SD) and primary progressive aphasia (PPA) and is present to lesser degrees across all dementia syndromes[11].

Consensus definitions of disinhibition in dementia are lacking. Disinhibition has been reported as consisting of four independent subsyndromes: abnormal motor behaviour, hypomania, egocentrism/loss of insight and poor self-care[12]. Disinhibition can occur face-to-face and/or in virtual environments e.g., digital, online, using personal electronic devices. Disinhibition involves impulsivity (for example, reckless driving, vulnerability to exploitation), compulsivity, and social disinhibition[13, 14]. Social disinhibition in dementia can include[13, 15]:

- urinating or undressing in public
- finding humour where others don't
- uncontrolled eating
- excessively demanding attention
- low frustration tolerance and impatience
- anti-social behaviour
- emotional inappropriateness
- pathological gambling
- unwanted staring
- taking things from others.
- exhibiting vulgar, impulsive or deviant behaviour
- loud, insulting or hurtful comments about others, including strangers
- use of language considered inappropriate to the context
- discussing personal or private matters in a public space or forum

When disinhibition presents with a sexual resonance, the challenges for those providing care increase[16]. Sexual disinhibition in dementia has been defined as "sexual behaviours that are inappropriate, disruptive, and distressing and that impair the care of the person in a given

environment"[17, 18]. Criteria for inappropriate sexual behaviour in dementia have been defined as[19]:

- diagnosed dementia or major neurocognitive disorder
- the behaviour did not present before the person developed cognitive impairment/dementia
- verbal or physical behaviour that is explicitly sexual, or perceived as such based on the contex
- the behaviour is considered inappropriate because
  - it occurs in a public space and is actually or potentially offensive to others
  - it is directed at, and unwanted by, the recipient
  - it is directed at a vulnerable person who is unwilling or unable to consent
  - it is excessive in context i.e. sought at inconvenient times, interferes with normal daily activities, and/or poses are risk to self or other and the person is incapable of controlling the behaviour.

Sexual disinhibition may take place in face-to-face and/or in virtual environments i.e., online and in inappropriate spaces using personal electronic devices. These can include:

- touching genitals in public, exhibitionism
- sexual remarks, propositioning others
- grabbing, groping, reaching inside the underwear of vulnerable others
- masturbation in the presence of others
- simulating sexual acts
- requesting unnecessary genital care
- unwelcome cuddling, fondling, frotteurism or manipulating others' clothing
- chasing others for sexual purposes
- attempts at intercourse or other sexual acts, rape
- aberrant sexual behaviour such as sexual aggression
- photographing genitals/sex organs of others and/or recording sex acts without consent
- publicly reading, viewing, posting pornographic material.

Sexual expression is a human right and a normal and basic need in older adults including those with dementia[19-21]. Sexual expression can be complex and diverse. Appropriate sexual expression in people with dementia is not pathological. Understanding and support in this area may be required for those living in residential care and their staff[22]. Tools are available to assess how supportive of residents' sexual expression an aged care home is[23, 24]. It is important to differentiate behaviour that may be 'normal'

PRESENTATION

Alex has a three-year history of vascular dementia. His wife sought residential care placement for Alex when she could no longer cope with his increasing sexual demands and he accused her of becoming pregnant through an extra-marital affair with one of their neighbours. On admission to residential care Alex presented as generally quite pleasant and friendly. Staff reported some "inappropriate joking" and taking items from other residents as a prank. Staff are aware that these instances could put Alex at risk of harm if other residents react angrily to his intrusions.

Not long after his admission, Alex discovered that a small group of female residents regularly enjoyed watching an evening 'reality show' on television together in one of the communal sitting areas. The show includes near nudity and sexual content. Alex is keen to watch the show with the residents but they find his response to the content inappropriate and they feel uncomfortable when he joins them.

On one occasion Alex approached several female staff members and a female resident in a sexually suggestive manner. Transferring him to another section of the facility was trialled but Alex's disinhibition soon resumed, escalating to the point of inappropriately touching others and publicly masturbating in the dining room during meals. When staff intervened or asked him to return to the privacy of his room he could become verbally aggressive and threatening.

sexual expression for the person from behavioural changes that are likely due to dementia. The implications of these changes for the person and their family as well as informal and formal carers can be significant.

Social, moral, cultural, ethical and medico-legal factors inevitably influence proposed classifications of sexual disinhibition[25, 26]. A systematic review identified four main content domains: hypersexual/obsessive-type behaviour, lewd/inappropriate behaviour, inappropriate interactions with others, inappropriate comments[27]. Formal and informal carer report suggests a five-domain structure: inability to inhibit, oversharing, inappropriate comments, inappropriate exposure and overly flirtatious[27, 28].

Causes of disinhibition

Disinhibition in dementia has been associated with frontotemporal ventral-predominant [29] and parietal lobe atrophy[30], white matter hyperintensities[31], focal brain lesions[32] and rare syndromes such as Kluver-Bucy syndrome[33, 34]. Atrophy in frontal brain areas and subcortical circuits can cause impaired social cognition and contribute to social disinhibition[14, 35]. Impaired social cognition impacts on capacity to recognise others' facial expressions/emotions, processing one's own emotions, ability to self-monitor and understanding the intentions and feelings of others i.e., theory of mind and empathy[19, 36, 37]. Impaired executive function, and memory and sensory deficits, can further contribute to lack of empathy, confusion and misunderstanding of social context, cues, and expected behaviour.

Disinhibition, and impaired social cognition, are core symptoms of behavioural-variant frontotemporal dementia (bvFTD)[38-40] and common in the spectrum of FTD and Lewy body dementia (LBD)[37, 41] including Parkinson's disease dementia (PDD)[36]. Disinhibition features in rarer diagnoses such as Semantic dementia (SD), Primary Progressive Aphasia (PPA), Huntington's disease[42] and younger-onset variants[43]. It is also associated with vascular dementia (VaD)[11, 44] and Alzheimer's disease (AD)[45, 46].

Acute expressions of disinhibition in people with dementia may result from many potential antecedents. See *Module 1, Table 1.2* for a list of potential contributory factors. Social factors, physical or sensory impairments, environmental stressors, fatigue, ill-health and lack of opportunity to engage in meaningful activities, companionship and/or sexual expression can contribute to disinhibition. For example, sexual disinhibition may present due to the lack of a usual sexual partner, lack of privacy[47], lack of opportunity to develop/sustain significant relationships, restrictive policies and/or care-rationing in residential care settings[48, 49].

Sexually ambiguous behaviour such as undressing in front of others because they are feeling too warm and/or in an inappropriate place or attempting to remove irritating clothing may be mistakenly deemed sexually inappropriate. Urinating in inappropriate places can occur when the person with dementia is unable to remember where the toilet is or find their way to the bathroom. Additionally, misinterpretation of the intentions of carers and/or staff, visual triggers in the environment, loneliness, unmet need for attention or affection in combination with diminished insight, judgement and/or awareness of their surroundings and others, may trigger disinhibition in a person with dementia[20]. Misidentification of others, often a daughter, as their long-term spouse[18] can be particularly problematic if the person with dementia is recalling their spouse when they were much younger.

Differential diagnosis

The crucial task for the clinician is to attempt to understand what the disinhibition means for the individual person living with dementia. Consultation with the person with dementia, their family, particularly partners/spouses and formal carers is essential when determining if the presenting disinhibition is indicative of a change, an exaggeration of their premorbid behaviour or normal for the person.

Disinhibition can occur with an exacerbation of the person's premorbid personality due to dementia. A history of disorders such as obsessive-compulsive disorder (OCD), attention deficit/

hyperactivity disorder (ADHD), bipolar disorders, substance use disorders, and personality disorders should be considered and/or excluded as the primary cause of disinhibition. Disinhibition in FTD can overlap with symptoms of primary psychiatric disorders[50].

Where disinhibition presents as a sudden change in the person with dementia, comorbid psychosis, delirium and/or a cerebral event should be excluded[51, 52]. Those with dementia may also be more vulnerable to the disinhibiting effects of alcohol[18]. Adverse effects of medications have been linked to sexual disinhibition and hypersexuality with the use of benzodiazepines [18, 53], donepezil[54], fluvoxamine[55] and levadopa in PD[56, 57].

In the case of sexual disinhibition, it is important to determine if the presenting behaviour is normal sexual behaviour for the person in an abnormal context, rather than inappropriate sexual behaviour. This may include diverse perspectives including consultation with the person[58], partners/spouses as well as consideration of sexual diversity [59-61] and cultural perspectives[62].

Measuring disinhibition

Standardised measures of disinhibition in dementia and validated scales for measuring sexual behaviour in dementia are available[13, 27]. Scales validated in people with dementia are listed below. Scales designed to assess disinhibition and sexual behaviour in specific populations such as PD[63-68] and Traumatic Brain Injury (TBI)[69, 70] are not listed here as they have not been validated in people with dementia[27].

- The **Disinhibition Scale** consists of 26 questions each for the person and the carer rating behaviours over the preceding four weeks[12]. Items are grouped into four domains: abnormal motor behaviours (e.g. hyperactivity), stereotyped routines (e.g., obsessive ideas/rituals), psychosis, hypomanic behaviour (e.g. hypersexuality) and poor self-care (e.g. poor insight into deficits).
- The 10-item carer-rated Middelheim Frontality Score (**MFS**) reliably differentiates FTD from AD, and measures the presence/absence of disinhibition, impaired emotional control, and stereotyped behaviour[13, 71]. Sexual disinhibition is assessed through hypersexuality, aberrant change in sexual behaviour, inappropriate sexual advances and comments[28].
- A tool to assess sexual disinhibition (**SD**) in dementia measures frequency, severity and impact on the carer in five domains: inability to inhibit, oversharing, inappropriate comments, inappropriate exposure, and overly flirtatious[28].

The following scales include items relevant to disinhibition:

- The disinhibition subscale of the Neuropsychiatric Inventory (**NPI**) is completed during an interview with the carer, in which they rate the frequency and severity of the person with dementia's disinhibition, as well as their own subsequent distress[72].
- The NPI-Clinician (**NPI-C**) expands the original disinhibition subscale by a further eleven items including disrobing, social judgement, demanding attention, insulting others and eating behaviours. An item relevant to sexual aggression has also been added to the aggression subscale[15].
- The 25-item carer-rated Behaviour Symptoms in Alzheimer's Disease Scale (**BEHAVE-AD**) includes one sexual disinhibition item[73].
- The Behavioral Syndromes Scale for Dementia (**BSSD**) includes a global disinhibition syndrome assessing a broad range of behaviours associated with impulsivity and sexual disinhibition based on the previous week[74].
- The Cohen-Mansfield Agitation Inventory (**CMAI**) provides items that measure inappropriate dress or disrobing, inappropriate physical and inappropriate verbal sexual advances[75].
- The Challenging Behaviour Scale (**CBS**) measures the frequency and severity of swearing, urinating in public, stripping, inappropriate sexual behaviour and deviant behaviour based on the past eight weeks[76]. There is insufficient evidence to recommend this scale[77].

- The CERAD Behaviour Rating Scale for Dementia (**BRSD**) includes an item for socially inappropriate behaviour[78].
- The Dementia Behavior Disturbance Scale (**DBDS**) includes impulsive, compulsive and inappropriate behaviour items as well as two sexual disinhibition items[79].
- The 24-item carer-rated Frontal Behavioural Inventory (**FBI**) discriminates FTD from AD and captures behavioural and personality changes, including disinhibition and one hypersexuality item[80].
- Frontal Lobe Personality Scale (**FLOPS**) has one item that measures sexual disinhibition and mentions inappropriate sexual comments, advances and being overly flirtatious[81].
- The 46-item carer-rated Frontal Systems Behavior Scale (**FrSBe**) includes disinhibition and executive dysfunction subscales, capturing inappropriate sexual comments/advances and being overly flirtatious before and after diagnosis[82].
- Iowa Scale of Personality Change (**ISPC**) includes one item that measures inappropriate sexual comments and advances[63].

Scales relevant to social cognition may be helpful in assessing deficits in self-monitoring, empathy, theory of mind, social communication and other aspects of socioemotional functioning. Assessment of social cognition and social behaviour can support accurate diagnosis of bvFTD[39]. Measurement type is important, and informant report may be more sensitive in identifying disinhibition related to social functioning[35, 83]. Relevant scales include:

- the Revised Self-Monitoring Scale (**RSMS**)[84]
- the Edinburgh Social Cognition Test (**ESCoT**)[85]
- La Trobe Communication Questionnaire (**LCQ**) Disinhibition/Impulsivity subscale[86].

Prevalence of disinhibition

Rates of disinhibition vary widely, with symptoms reportedly occurring in 1% to 61% of people with dementia[87-89], and a pooled prevalence of 17% across dementia diagnoses[11, 89]. Variance in prevalence arises from the different definitions of disinhibition, settings, measurement tools and the duration of assessment. Reported rates are typically lower in community samples[87] than in acute[88] and residential aged care settings. The prevalence of disinhibition tends to rise as cognition and functioning in activities of daily living diminish, and dementia increases in severity[90].

With prevalence rates ranging from 2% to 25%, sexual disinhibition in dementia is not reported as frequently as general disinhibition in dementia[27, 56]. Decreased libido and sexual activity are reportedly more characteristic[91]. Underreporting of problematic

ASSESSMENT

To reduce Alex's disinhibition that is potentially putting him and others at risk, contributing factors must be identified:

- *Sexual history and premorbid patterns of sexual interest*
- *Pain/discomfort/illness/infection*
- *Medication reactions, interactions, dosage, recently prescribed, adverse effects*
- *Lack of usual sexual partner/privacy*
- *Altered routines, new staff, particular staff and/or family members*
- *Unfamiliar/altered/deprived physical environment*
- *Psychotic symptoms/misidentification*
- *Loss of premorbid social controls*
- *Misinterpretation of environmental cues*
- *Sensory impairments*

Assessing the situation:

- *Directly observe for specific triggers for Alex's disinhibition.*
- *Ask staff who have come to know Alex if they can assist in identifying possible triggers that provoke his disinhibition.*
- *Consult Alex's life history as well as behaviour support and clinical charts for further information with regard to triggers.*
- *Assess the immediate environment for possible triggers.*
- *Consult family members to identify additional triggers which may be unknown to staff and not previously documented.*
- *Is a trial of pharmacological interventions indicated and/or appropriate for treatment of Alex's disinhibition?*

sexual behaviours, by formal care staff[92] and family carers, is likely and endorsement differs according to carer relationship i.e., spouse carers endorse certain domains more often than do adult children[28, 93]. Underreporting may be due to embarrassment, shame, social taboos[62], a sense of spousal duty and/or loyalty to the person with dementia. In a residential setting a 'victim' may also be impaired to the extent that they are unable to report the incidents. The cultural and religious background of carers may further influence perceptions of disinhibition and willingness to report.

Reports are inconsistent with regard to prevalence ratios in males and females with some indicating that sexual disinhibition occurs equally in both sexes, but more reports indicate a higher prevalence in males[16, 28, 94, 95]. This may be a reflection of various factors, including societal values. Male expression of sexuality is typically more active and may be perceived as more aggressive. Further, informal and formal carers of people with dementia are predominantly female which means they are the more often in a position to be subject to potentially inappropriate male sexual behaviour[19].

Effects of disinhibition

Disinhibition can be one of the most distressing and challenging BPSD for those providing care in all settings[88, 91, 96, 97]. Ethical/moral norms, the person with dementia's rights and their need for appropriate care are important considerations for carers[18]. Disinhibition can be stigmatising[1, 91, 98], and in more extreme cases anti-social or criminal e.g., causing injury or sex offending which can lead to legal action[42, 99]. Disinhibition can provoke an angry or aggressive response from other aged care home residents, day centre attendees and/or families as well as members of the public, depending on the context[47, 98, 100]. Disinhibition is associated with higher fall rates in long-term care residents with dementia[101]. Urinary tract infections, physical trauma and/or sexually transmitted infections are potential consequences of sexual disinhibition[102].

Sexual disinhibition in dementia is linked to earlier admission to aged care homes[103, 104], other BPSD, poor quality of life[56] and increased carer burden[105]. Residential aged care staff members have described sexual disinhibition as the most challenging BPSD[106]. Within residential care, the vulnerability of other residents can raise major concerns. The need may arise for staff to determine the capacity of residents with dementia to pursue a sexual relationship[60, 61, 107].

General principles and considerations to assist in the assessment of a person with dementia's competency to participate in a sexual relationship[12, 52, 60, 61, 107-110].

- *Embed the evaluation in an interdisciplinary team process.*
- *Determine the specific question and gather information from multiple perspectives.*
- *Conduct a medical records review for relevant information e.g., conditions affecting sexual functioning, medications that affect cognition and physical safety, pertinent elements of functional status.*
- *Conduct a clinical interview where possible (for example, behavioural observation, medical/psychological/social history, relationship history).*
- *Try to establish knowledge/understanding, reasoning/capacity and voluntariness/ choice as well as sexual wishes or values:*
  - *Is the person with dementia aware of the relationship?*
  - *Is he/she aware of who is initiating sexual contact?*
  - *Does he/she believe that the other person is his/her spouse/partner and thus acquiesces out of a delusional belief?*
  - *Is he/she aware of the other person's intent?*

- *Can he/she state what level of sexual intimacy they would be comfortable with?*
- *Does the person with dementia have the ability to avoid exploitation?*
- *Is the behaviour consistent with formerly held beliefs and values?*
- *Does he/she have the capacity to say no to any uninvited sexual contact?*

Written policies regarding sexuality and sexual health in residential aged care are often lacking, as is staff education and training regarding this aspect of care[111-113]. Routine assessment of the sexual health and sexual needs of residents may be indicated[47, 114, 115]. Clinicians should refer to local policy documents where available. Ageism, ableism, stigma, culture and/or religion may influence others' perceptions of the appropriateness of sexual expression in those with dementia including other aged care home residents[116], direct care staff in the community and residential care as well as health care professionals[48, 60, 62, 111, 117, 118].

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded no psychosocial and environmental, or biological and pharmacological intervention trials, that met our quality criteria for inclusion. As the evidence from studies assessing disinhibition published before 2012, previously included in the *BPSD Guide* (2012), did not meet our updated quality criteria these studies have been retained.

Addressing disinhibition

Developing a behaviour support plan for disinhibition begins with a thorough physical examination, medication review, medical history, sexual history, assessment of the current social and living environment as well as specifics of the presenting disinhibition, potential antecedents, precipitants and consequences[12, 20, 52]. Possible precipitating factors including medications, comorbid physical conditions, stroke, seizure disorder, comorbid psychiatric condition or environmental triggers should be considered and/or addressed. See *Appendix 4* for suggested questions to facilitate comprehensive assessment.

Where sexual disinhibition occurs, a problem-solving approach which addresses the presenting behaviours rather than clouding the issue with moral judgement is recommended. Education of family carers and/or aged care staff around sexuality issues in dementia should reinforce that disinhibition occurs secondary to cognitive impairment[19]. Consensus guidance on optimal approaches to addressing disinhibition and sexual disinhibition is lacking. Comparative evidence suggest that psychosocial/environmental interventions may outperform drug treatments[1, 20]. Refer to Principles of care for guidance.

Psychosocial and environmental interventions

Psychosocial/environmental interventions research to guide clinicians and carers in supporting those who present with disinhibition is lacking. Reviews of the literature provide the following guidance regarding person-centred care principles and non-pharmacological interventions[1, 8, 20]:

- Develop a consistent, neutral and simple response when disinhibition occurs. Avoid knee-jerk overreactions which prompt shame and leave the person with dementia feeling 'bad'.
- Avoid confrontation as this may provoke a catastrophic reaction.
- Observe for early indicators of sexual disinhibition, such as humour with sexual undertones or flirting. This may allow unwelcome behaviours to be diverted prior to them escalating.
- Encourage increased positive contact with friends, family and pets to help compensate for loss of companionship. Be aware of the potential for an increased need for close supervision of young children during visits.

- Remove precipitating aspects of the person's environment or triggers, such as overstimulating television shows, where possible.
- Engage the person in positive interactions and provide enjoyable experiences when disinhibition is NOT occurring; do not reinforce negative behaviours by paying these undue attention.
- Modify aspects of the person's environment which can be misinterpreted (i.e. visual and social cues) and reallocate seating in communal areas if indicated.
- Consider providing hands-on activities (for example, sport, painting, sewing, building, folding). Adjust activities to increase or decrease stimulation as needed and tailor activities to the individual's interests and retained abilities to encourage success.
- Where behaviours are inappropriate because they are occurring in a public space, discreetly redirect the person to their room or a private space and/or provide clothing and aids to support privacy e.g., activity/modesty apron, modified clothing with fastenings at the back.
- Be aware that restricting the person with dementia's use of their hands is physical restraint and is not recommended.
- Psychosocial interventions reported elsewhere such as physical activity, music approaches, education and models of care may warrant a trial[1].

Biological and pharmacological interventions

Research into biological/pharmacological interventions is also lacking. Case studies are reported in the literature but RCTs and comparative pharmacological studies specifically for treating sexual disinhibition are lacking[1, 20, 119]. Short-term pharmacological treatment should be reserved for use when nonpharmacological strategies have failed and the disinhibition represents a risk to the health, safety and/or well-being of the person and/or others. Clinicians need to consider drug toxicity profiles, adverse effects and potential harms. See *Module 2, Table 2.3* for side effects associated with antipsychotics.

Reduced symptoms for NPI agitation cluster scores, which included agitation/aggression, disinhibition, irritability and aberrant motor behaviour subscales have been cited elsewhere for a stepwise pain management protocol when compared with usual care[120, 1]. No significant decrease in disinhibition subscale scores was found and this study did not meet our inclusion criteria. Citalopram decreased disinhibition subscale scores in people with FTD but likewise, this study did not meet our inclusion criteria [1, 20, 121]. Others report various pharmacological interventions trialled for sexual disinhibition, including hormonal therapies, but good evidence is lacking. The authors conclude that nonpharmacological interventions are preferred although not always effective[20].

Expert guidelines do not provide recommendations on the use of antidepressants for disinhibition and recommend against the routine use of antidepressants in dementia[57]. Clinical practice guidelines indicate that antipsychotics, including risperidone, are not effective for treating disinhibition in dementia[57]. Accordingly, a decrease in psychotropic prescribing, particularly antipsychotics, in Australian primary care patients with dementia was reported between 2011 and 2020[122]. Where these are prescribed, only second generation/atypical antipsychotics should be considered. Antipsychotic medications should be avoided in people with DLB and PDD as they are at particular risk of extrapyramidal side effects and other severe negative reactions.

Good quality evidence for treating sexual disinhibition with hormonal agents is lacking[123] and it can be controversial when viewed as feminisation or chemical castration of males[124]. Depending on the jurisdiction, it may be necessary to obtain consent from a guardianship tribunal or board, not just from a family member or person responsible before administering hormonal therapy. For example, under the NSW Guardianship Act, use of oestrogens or antiandrogens in men with impaired decision-making capacity who exhibit sexual disinhibition is regarded as 'Special

Treatment' and approval by Guardianship Tribunal is required[125].

When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[57, 126]. For example, where the person presents with agitation and disinhibition, treating the agitation may also help to reduce disinhibition.

Limitations

Sound research into addressing disinhibition, particularly sexual disinhibition, in people with dementia is lacking[16]. Our review of the literature and other systematic reviews indicate that disinhibition is typically not the primary outcome of intervention studies and many studies do not specifically address disinhibition. Problems occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. Where outcomes are reported for disinhibition, these are typically based on case studies, subscale scores only or clustered BPSD domains which include disinhibition. Evidence for interventions based on these reports alone cannot be considered robust.

Conclusions/Principles of care

In summary, disinhibition in dementia can have significant and disabling consequences. A dilemma can arise in attempts to allow the person with dementia sexual

STRATEGIES/OUTCOMES

- *Direct observation suggested possible triggers for the disinhibition related to the presence of specific female staff members and a very sociable female resident who was seated at Alex's table in the dining room. The female resident was happy to change her seating to join a friend at another table.*
- *Care staff reported that Alex was possibly misidentifying their intentions during personal hygiene tasks as these also provoked a sexual response at times. Change in rosters enabled two male staff members to cover Alex's personal care needs between them, on most occasions.*
- *A review of Alex's current medication did not suggest any association with his disinhibition.*
- *When asked, Alex's wife reported that they had been a close couple until recent years. Alex had always been openly affectionate but since his diagnosis he has become increasingly less aware of social boundaries. Information and support were provided to Alex's wife.*
- *Alex's wife and family members provided additional items of special interest from his home that helped to keep him occupied during periods of reduced environmental stimulation. They also increased their visits and included Alex's beloved dog in their visits to provide Alex with additional positive contact.*
- *Given the small number of residents involved, the ladies were happy to relocate their 'reality show' television viewing to a rotating roster of their rooms.*
- *Consultation with staff members indicated that some were shocked and repulsed by Alex's disinhibition. They were unaware that these behaviours can occur during the course of dementia.*
- *Staff education was arranged with a community Clinical Nurse Specialist around factors contributing to Alex's disinhibition, 'normal' sexual expression in older people with dementia, diminished privacy issues, strategies to avoid provoking and/or address unwanted sexual behaviours as well as potential consequences of overreaction and shaming the resident.*
- *Opportunities for staff to debrief and validate their personal reaction to Alex's sexual disinhibition in a safe and enabling environment were provided. While female staff members were encouraged to avoid placing themselves at risk of Alex's sexual advances, it was emphasised that they were in no way responsible for Alex's sexually inappropriate behaviours when these did occur.*
- *In consultation with Alex's family, strategies to enable him to meet his sexual needs such as additional privacy, magazines, private time with his wife, professional sex therapy were considered.*
- *Additional behavioural and environmental measures reduced the frequency and intrusiveness of Alex's disinhibition.*
- *Increased staff awareness of the underlying issues increased staff confidence and ability to implement successful strategies for dealing with the disinhibition while allowing Alex to maintain dignity and stay safe.*
- *Close monitoring of Alex's disinhibition continued with a view to considering a trial of pharmacological interventions if the safety of others was jeopardised.*

expression while protecting the safety, rights and dignity of all. Recognised expert guidelines for addressing disinhibition are limited, particularly where sexual behaviours are involved. This should not prevent clinicians from considering strategies to reduce disinhibition on an individual basis[127]. Good clinical practice suggests tailoring psychosocial interventions to the person with dementia. For example, a person-centred approach[128] may indicate that for one person reducing the frequency of disinhibited incidents may result from regularly providing a distracting activity that provides pleasure at high-risk times of the day, for another it may be reviewing their personal care routine and for a third, it may be arranging outdoor walks with a carer or family member to reminisce about aspects of the local area.

Interventions for addressing disinhibition and sexual disinhibition should respect the rights of the person with dementia, the views of family and care partners as well as policy and regulations regarding informed consent. Cultural and community views as well as legal issues must also be considered[1]. Where pharmacological treatment is in the best interest of the person with dementia or necessary for the safety of others, medications may have a role to play in addressing disinhibition although sound evidence is lacking. Pharmalogical treatments have limited efficacy, potentially serious adverse effects and the evidence is largely based on case studies. As always, the need for a multidisciplinary, individualised and multifaceted approach is stressed.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

10. Nocturnal disruption

MODULE 10: Nocturnal disruption

Key messages

- The presentation of nocturnal disruption varies with dementia subtypes; the range of sleep-related symptoms is associated with both the person's night and daytime behaviours.
- Nocturnal disruption occurs with the degenerative brain changes associated with dementia; multiple physical, medication, environmental and/or social factors can also contribute.
- Differential diagnosis requires eliminating delirium, comorbid medical and/or psychiatric conditions, substance abuse, medication effects, parasomnias and other primary sleep disorders.
- Nocturnal disruption occurs in 20% to 100% of people living with dementia; prevalence can be inherent to the type of dementia.
- The impact on the health and well-being of the person with dementia, their informal and formal carers as well as others in their environment can be significant.
- Addressing nocturnal disruption requires identifying potential contributing factors such as pain, hunger, thirst, infection and/or socio-emotional needs.
- Interventions using the companion robot *Paro*, overnight acupressure and training for residential aged care (RAC) staff and carers provide the best psychosocial evidence.
- Light therapy is the only biological therapy recommended by clinical guidelines for irregular sleep-wake rhythm disruption, although the evidence is inconsistent.
- Limited evidence is reported for cholinesterase inhibitors (ChEIs) and short-term benefit for analgesic medications.
- Sleep disturbances can occur secondary to depression, anxiety and/or agitation and treating the underlying condition may be helpful.
- The potential risk/benefit ratio for the individual with dementia must always be considered before prescribing medication.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
   - the person living with dementia
   - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
   - the care partner/care staff
   - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

   (See *Module 1* for further details)

Nocturnal disruption summary

What is nocturnal disruption and what does it look like in dementia?

Nocturnal disruption refers to disturbances of the person with dementia's circadian rhythm and sleep. Symptoms vary with dementia subtypes but may present as:

- increased early-morning awakenings
- nocturnal sleep fragmentation
- decreased total sleep time
- decreased sleep efficiency
- reverse day-night patterns
- decreased slow wave sleep
- excessive daytime sleepiness
- decreased rapid eye movement sleep
- nocturnal confusion
- increased daytime napping
- loss of normal sleep architecture
- increased sleep onset latency.

Causes of nocturnal disruption

Degenerative changes in people with dementia contribute to nocturnal disruption. Physical factors such as pain, adverse effects of medication, and/or social and environmental factors also play a role. Other BPSD may also become exacerbated at night and disrupt sleep due to reduced environmental cues.

Differential diagnosis

Delirium, comorbid medical and/or psychiatric conditions, substance abuse, physiological effects of medications, parasomnias and other primary sleep disorders can be misdiagnosed as nocturnal disruption in dementia. Comprehensive clinical assessment is essential to exclude these potential causes of sleep disturbance.

Measuring nocturnal disruption

Current guidelines recommend the use of actigraphy whenever possible, for at least seven days and keeping a sleep log in the assessment of nocturnal disruption. Questionnaires such as the Sleep Disorders Inventory (**SDI**), the sleep disorders subscale of the 12-item Neuropsychiatric Inventory (**NPI**) or the NPI-Clinician (**NPI-C**), the Pittsburgh Sleep Quality Index (**PSQI**) and the Epworth Sleepiness Scale (**ESS**) may be useful.

Prevalence of nocturnal disruption

Nocturnal disruption occurs in 20% to 100% of people with dementia. It can also be inherent to the type of dementia and tends to occur more frequently in dementia with Lewy bodies (DLB), Parkinson's disease dementia (PDD), Huntington's disease (HD) and frontotemporal dementia (FTD) than Alzheimer's Disease (AD) and vascular dementia (VaD).

The occurrence of nocturnal disruption in dementia increases with dementia severity, the presence of APOE ε4 allele, anxiety, depression, loneliness, reduced exposure to environmental cues and poor sleep hygiene.

Effects of nocturnal disruption

Nocturnal disruption in dementia is associated with poor sleep quality, depressive symptoms, greater carer burden, earlier admission to residential aged care, social disruption, loneliness, poorer cognitive functioning, more rapid cognitive decline, greater functional impairment, poor quality of life and/or wandering in people with dementia as well as higher risk of falls and mortality.

Addressing nocturnal disruption

It is important to determine potentially treatable factors contributing to the nocturnal disruption. Addressing underlying causes may require effective pain management, relieving hunger or thirst, treating infection or adverse drug reactions and/or improving social health. Carer education around sleep hygiene and assessing night-time, environmental disturbance occurring close to the person with dementia may be helpful.

Psychosocial and environmental interventions

- Psychosocial intervention studies were primarily conducted in residential settings.
- Most studies fell under the animal-assisted interventions, touch therapies or education and training categories.
- Interventions using the companion robot *Paro*, overnight acupressure, and those focused on training for carers and staff in aged care homes provide the best psychosocial evidence for improved sleep-wake parameters.
- Studies of physical activity and models of care interventions found no benefit for nocturnal disruption.
- Although scientific evidence is lacking, traditional measures may contribute to addressing nocturnal disruption and should not be overlooked.

Biological and pharmacological interventions

- Light therapy interventions provide some evidence and are the only treatment recommended by clinical guidelines, although they are not recommended in combination with melatonin.
- Limited evidence is reported for ChEIs and the short-term benefit for analgesic medications.
- Evidence for melatonin therapies is mixed and current guidelines do not recommend their use in older adults living with dementia.
- Although some evidence of a very small-small effect is reported for suvorexant compared with placebo, review studies indicate equivocal evidence regarding efficacy and risks for people with dementia.
- No evidence of benefit was demonstrated for psychostimulants or antidepressants.
- Sleep disturbance and nightmares are well known potential adverse effects of ChEIs and some antidepressants can worsen rapid-eye-movement (REM) Sleep Behaviour Disorder.

Limitations

There are limited intervention studies in the literature and many studies report no effects or mixed results. Few studies investigated the long-term effects of the interventions, limiting their clinical utility in nocturnal disruption.

Conclusions/Principles of care

- Nocturnal disruption can cause significant distress for people with dementia living at home and their carers.
- Likewise, the challenges for residential care staff and distress for other residents in aged care homes are significant.
- Understanding the potential causes underlying nocturnal disruption will assist e.g., depression, anxiety, agitation and/or pain.
- The potential risk/benefit ratio for the individual with dementia must always be considered before prescribing pharmacological agents for nocturnal disruption.
- A lack of sound evidence should not prevent clinicians from considering person-centred strategies to reduce nocturnal disruption on an individual basis.

What is nocturnal disruption and what does it look like in dementia?

Nocturnal disruption refers to circadian rhythm and sleep disturbances, which may arise from alterations of the circadian system or a misalignment between the circadian rhythm of the person and the activity schedule in their social and/or physical environment. The symptoms of nocturnal disruption vary according to dementia type and may present with the following features[1-3]:

- increased sleep latency
- nocturnal sleep fragmentation
- increased early-morning awakenings
- decreased total sleep time
- decreased sleep efficiency
- nonrestorative sleep
- decreased slow-wave and rapid-eye-movement (REM) sleep
- altered thermoregulation
- nocturnal confusion
- increased daytime napping and excessive daytime sleepiness
- other behaviours and symptoms such as agitation, verbal disruption, hallucinations and wandering may also be exacerbated nocturnally.

The *International Classification of Sleep Disorders, 3rd Edition, text revision* (*ICSD-3-TR)*[4, 5] diagnostic criteria for Irregular sleep-wake rhythm disorder (ISWRD) are:

- The person or carer reports a chronic or recurrent pattern of irregular sleep and wake episodes throughout the 24-hour period, characterised by symptoms of insomnia during the scheduled sleep period (usually at night), excessive sleepiness (napping) during the day, or both.
- Symptoms are present for at least three months.
- Sleep logs are required, accompanied by actigraphy monitoring, whenever possible, for at least seven days, preferably 14 days. These show no major sleep period and multiple irregular sleep bouts (at least three) during a 24-hour period.
- The sleep disturbance is not better explained by another current sleep disorder, medical disorder, mental disorder, or medication/substance use.

Sundowning may be a clinical subtype of irregular sleep-wake rhythm disorder that features more severe sleep fragmentation and lower circadian rhythm amplitude for some people living with Alzheimer's Disease (AD)[4].

People with lived experience of dementia describe changes in their sleep patterns that reflect these features e.g., earlier or later waking, inability to get to sleep, sleeplessness and waking in the middle of the night[6, 7]. Some people with dementia describe "bad" or "crazy" dreams and experience sleepwalking and sundowning[6]. For people living with dementia and sleep disturbance, waking at night in dim light and darkness can be "scary", confusing, isolating, and lonely when other people may not be around to provide comfort[6-8].

Causes of nocturnal disruption

The causes of nocturnal disruption are multifactorial and the association between sleep, circadian systems and neurodegeneration can be bi-directional[1, 2, 9, 10]. The 24-hour sleep-wake cycle is regulated internally by the suprachiasmatic nucleus (SCN) and the nocturnal secretion of melatonin, and externally by environmental time cues (aka zeitgebers), including daylight

and physical activity[1, 9]. In people with dementia, degenerative changes often occur in the SCN. Impaired light perception due to cataracts/macular degeneration, altered melatonin and cortisol secretions, dysregulated core body temperature, changes in feeding/eating and limited exposure to environmental time cues, particularly for those living in residential aged care can also contribute to sleep pattern changes[1, 9, 11, 12].

Nocturnal disruption can also be caused by physical factors such as pain, discomfort, infection e.g., COVID-19[13] and iatrogenic causes as well as hospitalisation and communal living environments[6]. The presence and absence of external environmental stimuli including light exposure as well as room temperature, mealtimes, noise levels and social interactions or isolation can also contribute to nocturnal disruption[8, 12]. Other behaviours and symptoms associated with dementia such as agitation, verbal disruption, hallucinations and wandering may become exacerbated at night due to reduced environmental cues. Adverse effects of medication including cholinesterase inhibitors (ChEIs) and antidepressants[14, 15] or of their withdrawal can affect sleep.

PRESENTATION

Elaine is a recent admission to residential aged care from hospital, following a medical crisis. She was diagnosed with Alzheimer's disease 5 years ago and depression has been excluded during her hospital admission. Elaine previously lived a chaotic lifestyle at home with no routine and presented to hospital as dishevelled and underweight. She reportedly had no regular sleeping pattern at home so was frequently awake at night. Staff reported that her sleep/ wake cycle has been irregular since admission to the facility.

When she is often awake late into the night, Elaine wanders into other resident's rooms while they are sleeping and turns on the lights and/or television. In the mornings, she does not wish to be disturbed by staff and Elaine has become increasingly resistant to their encouragement to eat breakfast or have a shower. She can ill afford to lose weight at this time. Because staff are busy, Elaine frequently falls back to sleep until 11am or later.

Differential diagnosis

It is important to differentiate nocturnal disruption in dementia from parasomnias and other primary sleep disorders[3, 16]. For example, obstructive sleep apnoea is a common primary sleep disorder that both contributes to, and may be exacerbated by, cognitive dysfunction and dementia[2, 17]. Up to 60% of people living with Parkinson's disease dementia (PDD) and up to 70% of people with dementia with Lewy bodies (DLB) experience REM Sleep Behaviour Disorder (RBD), and RBD is also reported in Huntington's disease (HD)[2, 15]. Genetic factors may also play a role in familial circadian rhythm sleep disorders[18].

Comprehensive clinical assessment is essential to rule out sleep disturbance due to delirium, comorbid medical and/or psychiatric conditions, substance abuse or physiological effects of medications such as ChEIs, SRIs, venlafaxine, mirtazapine, bisoprolol, and tramadol[10, 15]. The Mayo Sleep Questionnaire[19, 20] is a brief carer-report instrument that can be used to screen for parasomnias and other primary sleep disorders. In residential care or a clinical setting, it is generally more practical to rely on careful observations and unobtrusive recording of sleep times over 24 hours to provide a guide to the nature of the sleep disturbance (see below).

Polysomnography (PSG) is an objective tool that reports relevant physiological parameters including electroencephalography, airflow, electro-oculography, electromyography, electrocardiography, respiratory effort and pulse oximetry during sleep[21]. Although PSG and video recording may be used to rule out other sleep disorders with similar symptoms[15], it can be expensive and poorly tolerated making it impracticable for many people with dementia[21, 22].

Measuring nocturnal disruption

Assessment protocols and tools for monitoring sleep in neurodegenerative diseases include electronic devices such as remote monitoring technologies (RMTs), smartphone applications, wearables, and home-based sensors[23, 24]. Multiple sensor and other digital technologies are used currently in health and aged care settings[25], and new technologies and techniques such as

home EEG are emerging[22]. However, there is scarce evidence of the effectiveness of wearable devices[25] and smartphone applications in monitoring sleep in people living with dementia. This is due in part to the lack of representation in studies, perceived lack of benefit by some with dementia and design and support issues as well as non-compliance, rather than lack of digital competence[24-28]. Large discrepancies can occur between characterisation of sleep disturbances measured by actigraphy and questionnaires. Multiple methods are advised to avoid under- or over-estimating sleep problems[29].

Current practice guidelines recommend the use of actigraphy and a sleep log in the assessment of nocturnal disruption in dementia. Questionnaires may also be useful[4, 29, 30]. Self-report measures have limited usefulness in people with moderate to severe dementia.

- Actigraphy uses small devices with accelerometers, commonly worn on the wrist, to continuously record limb movement over time to detect sleep-wake state. Actigraphy is an unobtrusive and naturalistic way to estimate sleep-wake patterns which can be used to assess a treatment response. Accuracy is reduced when sleep is fragmented or in the event of a coexisting movement disorder. However, actigraphy data may offer no benefit over proxy report for people with dementia and can overestimate sleep problems leading to increased prescription of psychotropic medication[29].
- A sleep log or sleep diary is commonly used as an adjunct to actigraphy to record subjective sleep experiences and relevant events such as drug administration times. Sleep logs based on care staff observation have been shown to be reliable and useful where self-report is not feasible. The Consensus Sleep Diary is an example of a standardised prospective tool[31, 32].
- The Sleep Disorders Inventory (**SDI**) describes frequency, severity, and carer burden associated with sleep-disturbed behaviour over two weeks[33]. A 3-to-4-point change or more constitutes a clinically important difference[34].
- The sleep disorders subscale of the 12-item Neuropsychiatric Inventory (**NPI**) is completed during an interview with the carer, in which they rate the frequency and severity of the person with dementia's sleep disruption or night-time behaviours as well as their own subsequent distress[35].
- The NPI-Clinician (**NPI-C**) version has added an additional question relevant to nocturnal disruption regarding agitation or concern around night-time awakening or being able to fall asleep[36].
- The Pittsburgh Sleep Quality Index (**PSQI**) is a self-rated questionnaire that assesses subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of nocturnal sedation and daytime dysfunction over one month[37].
- The Epworth Sleepiness Scale (**ESS**) is a brief self-rated questionnaire used to assess excessive daytime sleepiness[38].
- The self-report Sleep Continuity in Alzheimer's Disease (**SCAD**) assesses the length and quality of sleep, nighttime waking and difficulty falling asleep[39]. The scale has been validated in people with cognitive impairment and in Italian.
- The Uppsala Sleep Inventory-25 (**USI-25)** assesses bedtime, sleep onset latency, nocturnal awakenings, morning awakening, sleep duration and daytime napping[40].
- The Behavioural Pathology in Alzheimer's Disease (**BEHAVE-AD**) includes one item to assess diurnal rhythm disturbance[41].

Prevalence of nocturnal disruption

Nocturnal disruption commonly occurs in people with dementia, with prevalence ranging from 33% to 100%[42, 43]. Variance in prevalence generally arises from differences in dementia severity and subtype of those sampled, the definition of nocturnal disruption (e.g. clinically significant

sleep disturbance versus the existence of any symptom of sleep disturbance) and/or the assessment instrument used. Higher prevalence of sleep disturbance is reported for DLB/LBD (90%), PDD (80-100%), HD (58%-90%) and frontotemporal dementia (FTD; 33%-76%) when compared to Alzheimer's disease (AD) at (45%)[21, 42, 44-47], and a higher prevalence in AD compared to vascular dementia (VaD)[43].

The occurrence of nocturnal disruption reportedly increases with dementia severity, the presence of APOE ε4 allele[48, 49], anxiety, depression, limited exposure to environmental cues and poor sleep hygiene. Nocturnal disruption has also been associated with advanced age, male gender[29, 49], poor health, lower levels of physical activity, incontinence and obesity in the general population which can also occur with dementia.

Effects of nocturnal disruption

The extent of sleep problems in people with dementia has been clearly demonstrated as a major clinical problem[50]. Neurodegenerative changes affect epigenetics[51], circadian rhythms and sleep quality. Circadian and sleep alterations can in turn impair memory and cognition, drive neuroinflammation and worsen disease processes[1, 10, 51]. The human circadian system impacts all major organs and systems[10]. Sleep loss can further interfere with social health and functioning by perpetuating a self-reinforcing cycle of social withdrawal and loneliness[52-54]. Disrupted sleep leads to daytime *slump*, fatigue/feeling tired and taking naps[6, 7]. Disrupted sleep and associated fatigue can leave people with dementia and carers feeling frustrated and irritated[6]. Memory problems, decision-making abilities and overall quality of life can worsen[3].

Nocturnal disruption is linked to significant detrimental effects in the person with dementia including[10, 55]:

- poorer cognitive functioning
- faster cognitive decline
- greater functional impairment
- reduced quality of life
- increased agitation
- institutionalisation
- dysregulated metabolism and hunger/eating
- poorer psychological functioning
- poorer motor control
- increased psychotropic prescription
- increased wandering
- depression
- higher risk of mortality.

Family carers may experience fatigue, poorer sleep quality and health[42] as well as increased depressive symptoms[56], burden[42, 57] and distress[58] related to nocturnal disruption in the person with dementia[59]. In residential care, other residents and staff are also impacted[55]. Nocturnal disruption contributes to consistent worsening of changed behaviours and symptoms in the late afternoon and early evening or sundowning[9]. Where nocturnal disruption prompts night wandering, safety may be an issue. See *Module 12 Wandering* for more information.

Results

A systematic literature review to set criteria (see *Appendix 5)* yielded eleven psychosocial and environmental as well as ten biological and pharmacological intervention studies with outcomes relevant to nocturnal disruption in dementia. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained but not included in the summary numbers.

Addressing nocturnal disruption

It is important to determine potentially treatable factors contributing to the nocturnal disruption. *Appendix 4* provides suggested questions to facilitate comprehensive assessment. Addressing underlying causes can include:

- relief from pain, hunger, thirst and/or the need to urinate

- cues to provide direction back to bed from the toilet
- monitoring evening fluid intake
- treating infection or adverse drug reactions
- addressing socio-emotional needs
- carer education around sleep hygiene
- assessment of noise or environmental disturbance occurring at night close to the person.

ASSESSMENT

To reduce Elaine's nocturnal disruption, potentially contributing factors must be identified:

- *Chronic or acute pain/discomfort/ illness/infection preventing her from settling at night*
- *Medication interactions, dosage, adverse effects, recently prescribed*
- *Unfamiliar physical environment*
- *Unfamiliar noise/disruption/light from night staff attending to the needs of other residents nearby*
- *Less flexibility with routine than Elaine previously had at home*
- *Some staff have little awareness that her previous lifestyle factors may be contributing to the nocturnal disruption.*

Assessing the situation:

- *Consult family members to identify possible strategies to assist in managing Elaine's disrupted sleep patterns.*
- *Directly observe her pattern across a 24-hour period.*
- *Monitor her food and fluid intake.*
- *With consent, contact the hospital for any additional information available with regard to Elaine's history.*
- *Is pharmacological intervention indicated and/or appropriate for treatment of the nocturnal disruption?*

Psychosocial and environmental interventions

Psychosocial/environmental intervention trials were mainly conducted in residential settings.

Animal-assisted interventions
Quality of research studies: 3/3 moderate
Outcomes of studies: 3/3 positive

Three residential care studies showed positive outcomes for daytime sessions with the companion robot *Paro*. A very large RCT of 15-minute non-facilitated, individual *Paro* sessions with artificial intelligence (AI) enabled, three afternoons per week, provided strong evidence of a small positive effect. Those in the AI-enabled group showed a significantly greater decrease in time spent in nocturnal physical activity and number of night-time steps after 10 weeks compared with the AI-disabled group. Benefits were also evident five weeks post-intervention compared with the AI-disabled group and usual care[60].

Two medium RCTs reported moderate evidence of medium effect on sleep for 30-minute *Paro* sessions compared with usual care[61, 62]. People with moderate to severe dementia demonstrated significantly increased daytime awake hours and decreased daytime sleep hours after 6 weeks of 30-minute non-facilitated *Paro* activity five times weekly[62]. Sleep efficiency increased significantly, and average total sleep time was significantly greater compared with usual care, after 12 weeks of 30-minute small group *Paro* activities[61].

Touch therapies
Quality of research studies: 3/3 moderate
Outcomes of studies: 2/3 positive, 1/3 negative

A large residential aged care (RAC) study of people with mild AD provided some evidence of a medium effect for acupressure wrist devices applying pressure on the *Shenmen HT7* point overnight. PSQI sleep quality scores and hours of effective sleep increased significantly at 8 weeks[63]. Significantly improved sleep was demonstrated for both, 20-minute ear acupressure and relaxing lower limb and back massage sessions 5 times weekly compared with usual care, after three months in a large RAC study of people with moderate to severe dementia[64, 65]. Bedtime 3-minute slow-stroke back massage did not outperform usual bedtime care in sleep disturbance after two nights in a medium study of RAC residents with moderate to severe dementia[66].

Physical activity
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A medium community-based study of people, with AD, DLB, PDD or mixed dementia at any stage, found ESS sleep quality scores did not improve after 12 weeks of twice-weekly 45-minute sessions of either chair yoga, customised participatory music or gentle chair-based exercise[67].

Education/training
Quality of research studies: 2/3 strong, 1/3 moderate
Outcomes of studies: 2/3 some evidence, 1/3 negative

Compared with usual care, strong evidence of significant improvements in carer-rated behaviour frequency and sleep time were demonstrated for staff/carer sleep education in a medium study over 1 month. Sessions were conducted by an experienced trainer to develop and implement individualised sleep plans for RAC residents with mild to severe dementia and disturbed sleep[68]. Another medium RAC study provided moderate evidence of a large effect for significantly less daytime sleep and more night-time sleep for staff training in person-centred dementia care, compared with usual care in residents with severe dementia at three months[69]. By contrast, a medium community-based study provided strong evidence of no change in ESS scores and no benefit over usual care for multicomponent psychologist-led carer training sessions focused on sleep disturbance (*DREAMS-START)* over 3 months[70].

Models of care
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A large retrospective chart review study reported no significant reduction in the number of people with sleep disturbance after receiving multidisciplinary inpatient care provided in a specialised hospital ward with homelike environmental features[71].

Multicomponent interventions
No multicomponent intervention studies published since 2012 met our quality inclusion criteria however, literature published prior to 2012 provided mixed support. Two community-based studies of carer sleep hygiene education, daily walking and increased daylight exposure (*NITE-AD*) showed significant improvements in the frequency and duration of night-time awakenings and sleep percentage[72-74]. A multicomponent environmental and daytime activities intervention reported RAC residents spent less time in bed during the day[75].

Summary

Some positive outcomes were reported for interventions involving a companion robotic seal[60-62], overnight acupressure[63], and RAC staff/carer training in person-centred dementia care and individualised sleep plans[68, 69]. Traditional measures such as warm milk, reassuring human contact, gentle massage and soothing music may be considered although scientific evidence is lacking in these areas. Likewise, effective continence strategies and avoiding caffeine may promote better sleep[76]. See *Appendix 2* for interventions reported above.

Biological interventions

Brain stimulation therapies
Quality of research studies: 3/3 moderate
Outcomes of studies: 1/3 positive, 1/3 limited evidence, 1/3 negative

Significant improvement in PSQI sleep quality scores was reported for daytime light therapy tailored for maximum benefit, and significantly greater improvement compared to lower-level placebo lighting after 4 weeks, in a medium trial undertaken in residential aged care homes and assisted-living facilities[77]. A small, community-based trial reported limited improvement in PSQI sleep efficiency scores for blue-white, fluorescent light when compared with usual lighting, but no difference in sleep efficiency when compared with placebo yellow-white light all during daytime hours[78]. A large

RAC study that compared fluorescent warm white and cold-white ceiling lights with warm-white conventional lighting over 8 weeks during autumn/winter months reported no significant effect of one type of lighting over the other on sleep efficiency in residents with severe dementia[79].

Four additional trials of various approaches to light therapy, located in our literature search, were excluded here due to modest quality but are included in *Appendix 3*. Clinical practice guidelines provide weak evidence for light therapy in combination with behavioural interventions but not in combination with melatonin, for people with dementia and irregular sleep-wake disorder[15, 80].

Analgesic medications
Quality of research study: 1/1 moderate
Outcome of study: 1/1 mixed evidence

Significantly improved total sleep time, sleep onset latency and early morning wakening were found for active pain treatment, with paracetamol or transdermal buprenorphine when compared with placebo, in RAC residents with dementia and depression not already receiving pain treatment in a large RCT after one week[81] but not at 13 weeks[82].

Cholinesterase inhibitors (ChEIs) and/or memantine
Quality of research study: 1/1 moderate
Outcome of study: 1/1 limited evidence

A small non-controlled study of people with mild to moderate AD provided no good evidence of benefit for initiating and up-titrating memantine over four weeks. Based on polysomnography (PSG) data, total minutes of sleep time and sleep efficiency increased compared with baseline, but average AIS insomnia scores did not decrease significantly[83]. Three ChEIs trials, located in our literature search, were excluded here due to modest quality but are included in *Appendix 3*. A medium trial conducted prior to 2012 reported increased REM sleep for donepezil but no effects on any other sleep parameters[84]. It is important to note that ChEIs can cause sleep disturbance[85, 86] and nightmares[87] and monitoring for adverse effects is indicated.

Antidepressants
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

Community-dwelling people with sleep disorders in AD reported no positive effect on sleep at two weeks for daily Mirtazapine. Actigraphy data indicated no significant change in nocturnal sleep duration or efficiency and daytime total sleep *increased* compared with placebo after two weeks in this medium RCT[88]. Clinical guidelines recommend against routine use of antidepressants for sleep disturbance[89] and mirtazapine has been reported as worsening RBD[15].

Psychostimulants
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A medium open-label, chart review of inpatients with severe dementia in an acute neuropsychiatric unit reported no significant change in number of observed night-time wakenings or sleep duration for the cannabinoid dronabinol over seven days[90].

Other pharmacological/biological treatments
Quality of research studies: 2/3 strong, 1/3 moderate
Outcomes of studies: 2/3 positive, 1/3 negative

A very large trial of suvorexant, an orexin receptor antagonist, provided strong evidence of significantly greater improvement in total sleep time and Clinician's Global Impression of insomnia severity, for suvorexant when compared with placebo with very small-small effect at four weeks[91]. Other reports regarding suvorexant are inconsistent regarding effectiveness and adverse effects. A lack of harmful effects is reported in one review[92] and a higher risk of sedation and somnolence highlighted in another[93]. Data on long-term effects are lacking.

Strong evidence was provided of a medium effect for prolonged-release melatonin. Total PSQI

scores and sleep efficiency scores decreased significantly when compared with placebo at 24 weeks in a large RCT of outpatients with mild-moderate AD[94]. A moderate quality community-based study reported no significant difference in PSQI sleep quality between melatonin and placebo at 8 weeks in people with mild to moderate dementia and sleep alteration[95]. A very large study conducted prior to 2012 reported improved nocturnal disruption with melatonin alone and in combination with bright light therapy[96]. International guidelines recommend against the use of melatonin due to an overall lack of evidence[80, 92, 97, 98]. See *Appendix 3* for interventions reported above.

STRATEGIES/OUTCOMES

- *After investigation via the hospital, Elaine's only surviving sibling was contacted. He reported that Elaine used to enjoy her garden and was once very proud of her azalea collection. Elaine would also spend time flicking through newspapers and 'junk mail' brochures.*
- *Elaine has had two recent relocations which will likely have increased her confusion and disorientation. Providing familiar items may help her identify her space, making it more comfortable and appealing at night. Although Elaine's brother had not been close to her for some years, he was willing to bring some personal items to the facility. Elaine was pleased to see her brother.*
- *It became evident that Elaine was further disrupted at night by activity around the nurses' station and staff attending to the high-level needs of the resident in the next room. She was moved to a quieter area.*
- *The relative inactivity and darkness within the RAC environment at night, provided reduced cues. A small night light in her room assisted with orientation.*
- *Supporting staff through Elaine's transition to residential care and providing education around the factors contributing to her night-time wandering improved understanding of, and tolerance for, the episodes of nocturnal disruption.*
- *Keeping Elaine's daily routine as predictable as possible, given her previous lifestyle, provided some structure and ultimately, familiarity.*
- *Staff members were able to chat to Elaine about her passion for gardening and source local newspapers for her to browse.*
- *A daily walk to the aged care home letterbox after multiple brochures (recycled by staff) were 'delivered' became a positive activity.*
- *To further help establish a daily routine, Elaine was encouraged to take a brief nap of 30 minutes and never more than 45 minutes after lunch. Staff monitored how long Elaine napped to ensure she didn't sleep all afternoon.*
- *Participation in activities relevant to Elaine's history were promoted during the day and if she fell asleep while sitting at the table to eat, care staff assisted her to continue eating.*
- *Better sleep hygiene minimised opportunities for prolonged sleep during the day. Gradually a somewhat earlier and regular bedtime was established for Elaine.*
- *While she was still awake later than other residents in the facility, the situation became more manageable and less disruptive to others.*

Antipsychotic medications

Evidence to support the use of antipsychotic medications for nocturnal disruption in dementia is lacking, although those with sedative effects, such as quetiapine or olanzapine, have been prescribed especially when sleep disturbance occurs with agitation[92]. Antipsychotics are not recommended for people with dementia unless the sleep disruption is secondary to psychosis and then only when non-drug approaches and other drug therapies are contraindicated or ineffective[98-100]. Their use has been associated with further cognitive decline and greater risk of somnolence, extrapyramidal symptoms, abnormal gait, oedema, urinary tract infections, incontinence, falls, cerebrovascular adverse events and mortality[101]. Any use of antipsychotic medication must be time-limited and closely monitored for adverse effects. See *Module 2, Table 2.3* for side effects associated with antipsychotics.

Consistent with others, no studies for sedative-hypnotic medications were found for this review although hypnotics such as the benzodiazepines are commonly prescribed for sleep disturbance[92]. These may help with sleep temporarily[98], but clinical guidelines recommend against the use of sleep-promoting medications including sedative-hypnotic medications for sleep disruption in people with dementia. These medications increase the risk of confusion and falls[55] and in the long term are addictive and lose

their potency. If such drugs are used as a rescue medication, shorter-acting hypnotics with no active metabolites, such as oxazepam, are preferable to longer-acting.

While there is no empirical basis for their use, sedating antihistamines, anti-epileptic medications and antidepressants are also prescribed[92]. Some of these medications can worsen symptoms of sleep disturbances including RBD. Clinical guidelines recommend against use of these medications for sleep-wake disturbance in people living with dementia due to a lack of evidence for efficacy and clear associated risks. Analgesics have a place where pain is obvious or suspected. As simple a remedy as paracetamol at night may be helpful and is well tolerated.

Understanding the potential causes underlying the nocturnal disruption for each individual person with dementia will assist. Sleep disturbances can be secondary to depression, anxiety, agitation and/or pain and pharmacotherapy for the underlying condition may be helpful. If antidepressants are indicated for major depressive disorder, SSRIs are preferred[89]. See other modules in this guide for interventions to address other possible underlying BPSD.

The potential risk/benefit ratio for the individual with dementia must always be considered before prescribing pharmacological agents for nocturnal disruption. Symptomatic, pharmacological treatment should be time-limited, closely monitored, frequently reviewed and withdrawn when possible[80, 89]. Jurisdictions vary in their requirement for informed or proxy consent for regular prescriptions of psychotropic medications and local requirements should be checked. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[89, 102]. For example, where the person presents with depression and nocturnal disruption, treating the depression may also help to reduce nocturnal disruption.

Limitations

Intervention studies, particularly those providing strong evidence, are limited in number and many report no benefit or mixed results. Nocturnal disruption is frequently not the primary outcome of reported intervention studies. Problems occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for. Few trials investigated the long-term effects of the interventions with only four studies conducting post-intervention follow-up assessments ranging from five weeks to five months[60, 63, 65, 68]. These factors limit their clinical utility in nocturnal disruption.

Conclusions/Principles of care

In summary, nocturnal disruption is common in dementia and it has significant consequences for people with dementia and their carers. There are limited intervention studies available and most provide moderate evidence at best. Interventions using a companion robot, overnight acupressure and training for carers and staff in residential aged care homes provided the best psychosocial evidence. In literature published before 2012, the *NITE-AD* multicomponent intervention, also indicated benefit. Paracetamol or buprenorphine may be beneficial for musculoskeletal pain and limited evidence is provided for light therapy and analgesic medications. Despite some evidence of benefit for suvorexant and melatonin, clinical guideline recommendations as well as concerns around risk and limited efficacy mean these medications cannot be recommended.

A lack of sound evidence should not prevent clinicians from considering strategies to reduce nocturnal disruption on an individual basis[103]. Traditional interventions to promote sleep may contribute to individualised strategies and shouldn't be overlooked. Good clinical practice suggests tailoring psychosocial interventions to individuals. For example, a person-centred approach[104] may indicate that for one person reducing nocturnal disruption may result from changing their personal care routine to include a relaxing bath before bed, for another it may be improving their pain management and for a third, it may be increasing daytime, outdoor activities with a carer or family member to ensure adequate daylight exposure. As always, the need for a multidisciplinary, individualised and multifaceted approach is stressed.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

11. Vocal disruption

MODULE 11: Vocal disruption

Key messages

- Differing definitions of vocal disruption yield varying prevalence rates and shape intervention studies.
- Vocal disruption causes distress for the person living with dementia and others within residential aged care, hospital and community environments.
- Three key areas can prompt vocal disruption and provide targets for intervention:
  - Pain and discomfort (physical and/or psychological and/or social)
  - Operant conditioning of vocalisations due to the increase in attention they attract
  - Reduced-stress thresholds due to cognitive impairment.
- The inclusion of 'disruptive' in the definition is based on the perception of others.
- Environmental modifications to support resident orientation and a staff education and support program provide the best psychosocial evidence for decreasing vocal disruption.
- The evidence for effective biological/pharmacological interventions for vocal disruption overall is lacking.
- Clinical guidelines recommend against the use of atypical antipsychotics for vocal disruption however, short-term use of risperidone may be considered for those with underlying psychotic symptoms.
- Emerging technologies may help to identify underlying pain in people who vocalise but who cannot express themselves verbally.
- As always, a multidisciplinary, individualised and multifaceted approach is recommended.
- McMinn and Draper provide a practice guideline for treating vocal disruption, based on potentially contributing factors (see *Figure 11.1*).

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
   - the person living with dementia
   - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
   - the care partner/care staff
   - the care environment
3. Checked for, and treated, reversible causes of the BPSD

   (See Module 1 for further details)

Vocal disruption summary

What is vocal disruption and what does it look like in dementia?

- Vocal disruption is also referred to as persistent vocalisation, screaming and verbal agitation.
- Vocal disruption can be described as any vocalisation that causes the person distress and/or causes stress to others.
- Vocalisations include calling-out, screaming, abusive or verbally aggressive comments, perseveration, repetitive questioning, groaning and sighing which can be intermittent or incessant.
- Pain, unmet needs, operant conditioning, environmental vulnerability and reduced stress-threshold in people with dementia can contribute to vocal disruption.

Causes of vocal disruption

Causes include pain, physical and/or psychological discomfort including trauma, loneliness and social isolation e.g., based on cultural and linguistic diversity. Vocal disruption can also result from reduced stress threshold due to cognitive impairment and operant learning e.g., increased attention is provided when calling out but the person is ignored when they are silent.

Differential diagnosis

- Vocal disruption should be excluded from a delirium.
- Symptoms of vocal disruption overlap with agitation in dementia.
- Vocalisations can be disruptive whether or not the person with dementia has an awareness of their needs.
- The inclusion of 'disruption' in the definition is based on the perception of others.
- The same vocalisation may be disruptive in one context and not in another.

Measuring vocal disruption

- Most scales measuring vocal disruption include it as a subset of BPSD or agitation.
- The original Neuropsychiatric Inventory (**NPI**) does not include a subscale relevant to vocal disruption, however the revised NPI-Clinician (**NPI-C**) includes an additional subscale for measuring aberrant vocalisations.
- The verbal behaviour section of the Disruptive Behaviour Scale (**DBS**) includes subscales for aggressive and agitated (nonaggressive) vocal behaviours.
- The Cohen-Mansfield Agitation Inventory (**CMAI**) includes six items relating to vocal disruption.
- The Pittsburgh Agitation Scale (**PAS**) includes a category which measures aberrant vocalisation.

Prevalence of vocal disruption

Different definitions of vocal disruption yield varying prevalence rates based on how inclusive they are and the setting e.g., residential aged care versus at home. The prevalence of vocal disruption overall is difficult to estimate as reported frequencies often relate to various aspects of vocal disruption. Examples include cursing and/or verbal aggression from 10% to 48%, repetitious sentences/questions from 3% to 31.1% and screaming from 10% to 15%.

Effects of vocal disruption

Vocal disruption causes significant stress and/or distress within residential aged care, hospital and community environments. It has been shown to cause concern, frustration, anxiety, anger as well as complaints from care staff, visitors, other residents and/or neighbours. Vocal disruption can lead to the person with dementia becoming socially isolated or the inappropriate use of restrictive practices.

Addressing vocal disruption

The initial step for the clinician in addressing vocal disruption is to attempt to identify and understand the underlying factors provoking the vocalisations for the individual person. Where this is not achievable, the aim of treatment is to minimise distress to the person with dementia and those around them.

Psychosocial and environmental interventions

- Only three psychosocial/environmental intervention studies met our quality criteria for inclusion.
- All trials were conducted in residential care settings.
- Environmental modifications to support resident orientation showed limited evidence of a decrease in the number of episodes of screaming.
- Mixed evidence was found for an educational program *Bathing Without a Battle* which provided training and support for care staff during personal care activities.
- No benefit was found for psychosocial intervention.
- The potential causes of the vocal disruption may provide clues to appropriate interventions for the individual person.

Biological and pharmacological interventions

- The evidence for biological/pharmacological treatments for vocal disruption overall is lacking and no biological/pharmacological intervention studies met our quality criteria for inclusion.
- Current expert guidelines recommend against the use of atypical antipsychotics due to risk of harms and lack of benefit, unless the vocal disruption is underpinned by psychosis.
- Any potential benefits to the person with dementia must be weighed against the side effects before considering the use of pharmacological treatments.

Limitations

Numerous definitions and aspects of vocal disruption impact on how it is measured and addressed, as well as the evaluation of interventions. There is a lack of good quality evidence available and vocal disruption is frequently not the primary outcome of intervention studies. None of the reported trials investigated the long-term effects of interventions. These factors limit the clinical utility of interventions and the ability to make recommendations.

Conclusions/Principles of care

- Vocal disruption is common in dementia, with significant and distressing consequences.
- Recognised expert guidelines and reports on the outcomes of interventions are limited.
- Environmental modifications and an education program for staff provide the best evidence for psychosocial intervention.
- No good evidence was found for biological or pharmacological interventions.
- Atypical antipsychotics are not recommended due to lack of benefit and risk of harm.
- A lack of sound evidence should not prevent clinicians from considering strategies to reduce vocal disruption on an individual basis.
- A multidisciplinary, individualised and multifaceted approach is required.

McMinn and Draper provide a practice guideline for treating vocal disruption, based on factors thought to contribute to vocal disruption including discomfort, reduced-stress thresholds and operant conditioning (see *Figure 11.1*).

What is vocal disruption and what does it look like in dementia?

Vocal disruption is commonly associated with dementia. It is also referred to as vocally disruptive behaviour, persistent vocalisations[1] and verbal/vocal agitation[2]. Vocal disruption can be described as vocalisations (both verbal and non-verbal) that are upsetting to the person and/or that cause stress within the person's environment[1, 3]. Examples of vocal disruption include screaming, abusive or verbally aggressive comments, perseveration, repetitive questioning, groaning, sighing and unwelcome singing[4]. Vocal disruption can be intermittent or incessant, with potential peak periods in the afternoon[5], often related to sundowning. Vocal disruption may be[1, 6, 7]:

- aggressive or non-aggressive
- purposeless and perseverative or goal-orientated
- a response to the environment
- an indication of distress or unmet needs or demands
- communication by people who are non-verbal
- Communication by people who are approaching the end of life
- noise made in the context of impaired hearing
- other vocal sound-making.

People with lived experience of dementia may use different terms to refer to vocal disruption. Carers may describe people "calling-out" for someone who is unavailable or who has died, repeating things "over and over" due to memory lapses, that the person makes non-aggressive noises such as humming, that their vocalisations seem agitated or "frantic", or aggressive and "roaring"[8, 9]. People living with dementia describe distressing symptoms and powerful emotions including anxiety and fear motivating them to shout or "call for help"[8].

Causes of vocal disruption

Vocal disruption is caused by a complex interaction between factors related to the person with dementia and environmental contributors[10]. Seeking to identify potential causes of vocal disruption is important as these may provide clues to the appropriate choice of intervention[7]. The presence of vocal disruption is associated with:

- pain, discomfort[11, 12]
- dementia severity[5, 6, 13]
- impaired expressive communication[5, 11, 14]
- under-stimulation or hypersensitivity to stimulation[11]
- hypersensitivity to care or receiving care that is delivered without communication/warning[11, 15]
- impairment in activities of daily living (ADLs)[1, 5, 13, 16]
- reduced threshold for stress due to cognitive impairment[17]

PRESENTATION

Sarah has lived in residential care for two years. During the past year her tendency to repeatedly call out has increased. She constantly calls her daughter's name, at increasing volumes, until she is shouting. Other times she makes grunting noises. Her increasing distress and vocalisations appear to correspond to increasing anxiety, particularly when care staff attend to her personal hygiene needs. Staff and other residents are distressed and exhausted by Sarah's vocalisations. Some of her fellow residents can become very agitated when Sarah's calling out escalates. Families and visitors of other residents have complained to staff and management. Sarah's son and his family are embarrassed and uncomfortable when they visit, reporting to staff that they feel "helpless to ease mum's distress".

Sarah's daughter lives in America. Although she has always phoned her mother frequently and visits each year, Sarah has a history of missing her daughter's company. Staff attempt to assist Sarah to communicate with her daughter during her calls from America, but these currently provide little to no quality contact for either of them.

- mobility limitations such as wheelchair immobile or bedfast[6]
- current and historical trauma e.g., PTSD[11, 18]
- environmental restraints[19]
- psychotropic medication[13]
- depression[20, 21]
- poor quality relationships[21-23]
- reduced social interaction[19, 24]
- cultural and/or linguistic background not shared by other residents or staff[25, 26].

Higher levels of vocal disruption are associated with wandering/aberrant motor behaviours, irritability, agitation, anxiety, nocturnal disruption, euphoria, and visual and/or auditory hallucinations[8, 10, 13, 27-30]. Aggressive vocalisation is more likely to occur in those of younger age and better health[27]. A large Australian study used a computerised clinical system and pain assessment app[16] to explore pain and vocalised expressions in people with dementia who were unable to self-report. Outcomes indicated that sighing and screaming corresponded to high pain scores, vocalisations increased with pain intensity and vocalised pain expressions were graded and not discrete[12].

Differential diagnosis

Vocal disruption in dementia should be excluded from a delirium[6, 31]. Symptoms of vocal disruption overlap with agitation in dementia. Regardless of whether the person with dementia has an awareness of their needs or not, vocal disruption is distressing. The inclusion of 'disruption' in the definition is based on the perception of others[1, 27] and is not necessarily inherent to the vocalisations. It has been suggested that the situation may be compared with labelling the cries of a hungry baby as 'disruptive'[32]. Further, vocalisations may be disruptive in one context and not in another.

Measuring vocal disruption

Scales specifically measuring vocal disruption are limited and scales which include aspects of vocal disruption incorporate it as a subset of BPSD or agitation. The following are widely used[33]:

- The Cohen-Mansfield Agitation Inventory (**CMAI**) includes six relevant items: making strange noises, cursing or verbal aggression, screaming, repetitious sentences/questions, complaining and constant requests for attention[34].
- The Pittsburgh Agitation Scale (**PAS**) includes aberrant vocalisation based on vocalisation intensity and disruptiveness within the environment as well as effort required for redirection over four to eight hours[35].
- The 45-item Disruptive Behaviour Scale (**DBS**) includes aggressive and agitated vocal behaviours: screaming/yelling, hostile or accusatory language, threatening language, repetitive words or phrases, constantly talking and repetitious noises[27, 36].
- The revised Neuropsychiatric Inventory, the NPI-Clinician (**NPI-C**) includes an aberrant vocalisations subscale. Items include making strange noises, yelling, repetitive requests, abusive language, verbally sexual advances, muttering to oneself, nonsensical conversation, angry noises and verbally manipulative requests[37]. The original NPI[38] did not include a subscale relevant to vocal disruption.
- The Screaming Behavioral Mapping Instrument (**SBMI**) records multiple types of vocal disruption including shouting, screaming or howling, constant requests for attention, repeating words, complaining, cursing, verbal aggression, nonsense talk and talking to someone who is not present[39].
- The Typology of Vocalizations (**TOV**) scale measures verbal (e.g. singing, yelling) or non-

verbal (e.g. groaning, howling), meaning/reason/content (e.g. pain, ADL requests), timing (e.g. constant, random) and level of disruptiveness[40].

Prevalence

Different definitions, instruments used and target populations yield varying prevalence rates for vocal disruption based on how inclusive they are. Some prevalence studies focus only on specific types of vocal disruption. It is therefore difficult to estimate rates of vocal disruption overall. Reported frequencies of various types of vocal disruption in dementia are listed below.

Table 11.1 Types and frequency of vocal disruption

Types of vocal disruption	Frequency
Complaining	37.7% - 63.4%[28, 41]
Crying	3.3% - 9.1%[12, 42]
Cursing and/or verbal aggression	40%[28, 41]
Fearful comments	34%[7]
Howling	1.2%[12]
Groaning/moaning	7% - 8.2%[12]
Loud talk	7.8%[12]
Negative comments	37.6% - 67.5%[28, 41]
Sounds indicating pain	8.3%[12]
Repetitious sentences/questions	57.2% - 78.8%[28, 41]
Requests for attention or help	6.9% - 61.7%[12, 28, 41]
Screaming	2.4% - 34.1%[12, 28, 41]
Sighing	13%[12]
Nonspecific vocalisations	33.3% - 44%[28, 41]

Effects of vocal disruption

Vocal disruption causes considerable stress and/or distress in residential aged care, hospital and home environments[5, 6, 30]. These can manifest as concern, frustration, anxiety, anger and/or complaints from staff, visitors, other residents and neighbours[19]. Nursing staff may feel overwhelmed, powerless, frightened, and express a desire to 'block-out' or ignore calling-out and distance themselves from residents exhibiting vocal disruption[6, 43]. Further, the person with dementia may be socially isolated, subject to inappropriate restrictive practices and/or victim to verbal and/or physical aggression from other residents with reduced tolerance[15, 19, 22-24]. See important information below regarding the use of restrictive practice through seclusion.

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded three psychosocial and environmental intervention studies. No biological or pharmacological intervention studies met our quality criteria for inclusion. Refer to Principles of care for guidance. Where the evidence from studies published before 2012, previously included in the *BPSD Guide* (2012), met quality criteria they have been retained. Only one pharmacological trial was retained.

Addressing vocal disruption

The initial step in addressing vocal disruption is to attempt to understand the underlying factors that may be provoking the vocalisations, where possible. *Appendix 4* provides suggested questions to facilitate comprehensive assessment. Where this is not achievable, addressing vocal disruption may involve minimising distress to the person with dementia and those around them.

Disruption may be reduced by changing the immediate environment of the person. Transferring

them to an area where they may not be as disruptive for a period, such as a safe garden or a quiet space, may be appropriate if it provides the person and others with relief and supervision is maintained. Be aware however, socially isolating the person may increase their distress and worsen symptoms. The use of restrictive practice through seclusion requires careful consideration. "Seclusion is a practice or intervention that is, or that involves, the solitary confinement of a care recipient in a room or a physical space at any hour of the day or night where: (a) voluntary exit is prevented or not facilitated; or (b) it is implied that voluntary exit is not permitted; for the primary purpose of influencing the care recipient's behaviour"[44].

Part 4A of the Quality of Care Principles 2014[45] sets out the requirements that must be met for the use of restrictive practices in residential aged care settings. Under Commonwealth law very stringent legal obligations and protections for care recipients exist ensuring that restrictive practices are only used when absolutely necessary. If an aged care home uses restrictive practices, these are expected to be best-practice, used as a last resort, for as short a time as possible and compliant with relevant legislation. If a restrictive practice is used outside of these circumstances and does not meet the requirements in the Quality of Care Principles 2014, providers will be required to report that use of restrictive practices to the Aged Care Quality and Safety Commission as a serious incident under the Serious Incident Response Scheme[46]. See *Module 2* for additional information on restrictive practices.

ASSESSMENT

To reduce Sarah's distressing vocalisations, potentially contributing factors must be identified:

- *Unreported pain/discomfort/infection*
- *Medication review: interactions, dosage, adverse effects, recently prescribed*
- *Overstimulation (noise, people, activities) or understimulation/boredom*
- *Altered routines, new staff, particular staff and/or family members prompting anxiety/ distress*
- *Unfamiliar/altered/deprived physical environment*
- *Identification of potentially unmet needs*
- *Reduced threshold for coping with stress*

Assessing the situation:

- *Encourage Sarah to indicate her needs as far as she is able*
- *Directly observe for specific triggers for the vocalisations*
- *Ask staff who know Sarah well if they can assist in identifying potentially unmet needs or possible reasons for her calling out*
- *Consult Sarah's life history as well as behaviour and clinical charts for further information with regard to triggers*
- *Assess the immediate environment for possibly modifiable triggers*
- *Consult family members to identify other potential triggers that are unknown to staff and not previously documented*

The following theories may contribute to vocal disruption[17] and provide some insight into potentially reversible precipitants. Psychosocial intervention studies tend to focus on these areas[1, 12].

1. Meeting unmet needs:
    - pain
    - discomfort during routine care such as bathing, feeding or toileting
    - social isolation
    - boredom
    - low self-esteem
    - communication difficulties.
2. Operant conditioning, learning and behavioural interventions:
    - vocal disruption reinforced by increased attention, whereas silence is ignored

- repetitive questioning related to memory deficits (i.e. forgetting the answer that has been provided previously).

3. Environmental vulnerability and interventions aimed at supporting the person's reduced threshold for stress
   - relaxation
   - reducing noise
   - impoverished environment
   - environment inappropriate for needs.

STRATEGIES/OUTCOMES

- *Sarah has a history of chronic back pain following an accident many years ago. She is prescribed analgesia as required. A retrospective review of her medication charts indicated that Sarah currently receives pain relief irregularly and less frequently than when she was able to request it herself.*
- *Some staff members had little knowledge of dementia and were unaware that pain can prompt BPSD.*
- *Pain assessment indicated that Sarah may be experiencing frequent discomfort and/or pain, particularly in relation to personal care activities. Her analgesic medication was reviewed and regular day and night-time doses prescribed. Non-pharmacological pain relief interventions, such as gentle heat, were also implemented.*
- *Sarah's care plan was updated to schedule her shower for 30 mins after the morning dose of analgesia was administered.*
- *An OT assessment was requested to ensure the most appropriate and comfortable shower chair for Sarah's back pain.*
- *Resident room allocation throughout the aged care home was reassessed and Sarah was relocated to a quieter room with less stimulation to prompt her calling out, particularly at night.*
- *A redirection and relaxation program was developed to support Sarah. Gaining Sarah's attention by taking her hand, using her first name and ensuring eye contact enabled staff to engage her in up to ten slow breaths. This strategy initially stopped Sarah calling out for up to 20 minutes. Staff were trained to ensure consistency across shifts.*
- *Sarah was positively reinforced by giving her attention when she had not been calling out for a period of time.*
- *Behavioural observation charts indicated that Sarah responded positively to gentle touching and stroking. A volunteer visitor was trained to provide appropriate touch several times weekly.*
- *Sarah's favourite music and aromatherapy were also trialled with mixed results.*
- *Sarah's family members were trained and encouraged to participate in the redirection and relaxation program during their visits.*
- *Willing family members subsequently developed an informal roster to complement the volunteer's visits. They reported that they felt their contribution was beneficial to Sarah.*
- *Phone calls from Sarah's daughter were coordinated with times when Sarah was supported by family or a volunteer and hence, more relaxed. Short positive phone contact with her daughter left Sarah quite content.*
- *With time, the redirection and relaxation program resulted in longer vocalisation-free periods when Sarah was better able to communicate her needs and answer questions.*
- *Follow-up assessment indicated Sarah's episodes of disruptive vocalisation continued to reduce in frequency, intensity and volume. Her family felt Sarah's anxiety and overall quality of life had improved under the program. When she did call out, staff felt better able to reduce Sarah's distress.*

Psychosocial and environmental interventions

Psychosocial/environmental intervention trials were all conducted in residential care settings.

Therapeutic recreation
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A large trial that compared thrice weekly one-to-one positive psychosocial intervention sessions (IPPI) and standardised one-to-one activities to usual care for people with mild to severe dementia found no evidence of benefit for either intervention. After three weeks, very negative verbal behaviours were significantly *greater* for standardised one-to-one activities compared with individualised activities and usual care[47].

Education/training
Quality of research study: 1/1 moderate
Outcome of study: 1/1 mixed evidence

Evidence of a very small positive effect for significantly decreased calling for help or protesting during bathing was reported for a very large study of a staff educational program *Bathing Without a Battle*, but no decrease was found for yelling or aggressive language[48].

Models of care
Quality of research study: 1/1 moderate
Outcome of study: 1/1 1 limited evidence

A small study provided limited evidence of a small positive effect for environmental modifications to support orientation. Number of episodes, but not average duration, of screaming during 24-hour periods in people with moderate to severe dementia significantly decreased after three months. Modifications included painting walls, 'skylike' ceiling tiles, coordinating staff clothing, lighting to better differentiate day and night, oversized clocks and streaming soothing music[49]. See *Appendix 2* for interventions reported above.

Biological and pharmacological interventions

Research into biological/pharmacological interventions is lacking. A pooled analysis of three RCTs of people with psychosis of Alzheimer's disease (AD) or mixed dementia, published prior to 2012, provided some evidence that risperidone was more effective than placebo at reducing cursing or verbal aggression, repetitive sentences or questions and verbal outbursts[50]. Current guidelines recommend against the use of atypical antipsychotics due to risk of harms and lack of benefit for vocal disruption such as calling-out[51]. However, short-term use of risperidone may be considered for those with vocal disruption and distressing, underlying psychotic symptoms that represent a threat to themselves or others[51]. Wherever possible, the use of symptomatic, pharmacological agents, when required for treatment of vocal disruption, should be time limited, closely monitored, reviewed, reduced and/or discontinued when indicated, and prescribed with appropriate psychosocial interventions. When more than one BPSD occurs and medication is considered necessary, avoid polypharmacy by prescribing medication only for the primary behaviour or symptom[51, 52]. For example, where the person presents with anxiety and vocal disruption, addressing the anxiety may also help to reduce vocalisations. As always, the potential benefits to the person with dementia must be weighed against the side effects of pharmacological treatments. See *Module 2, Table 2.3* for side effects associated with antipsychotics.

Limitations

The many and varied definitions and types of vocal disruption shape how vocal disruption is measured and addressed. The different instruments used to measure vocal disruption also impact on the evaluation of interventions, particularly where instruments are limited in their measurement. Vocal disruption is frequently not the primary outcome of intervention studies for BPSD. Problems occur when multiple, individual BPSD subscale scores are analysed in

the same study and multiple comparisons are not accounted for. None of the reported trials investigated long-term effects of the interventions. These factors and the heterogeneity of the evidence limit the clinical utility of interventions and the ability to make recommendations for practice[1].

Conclusions/Principles of care

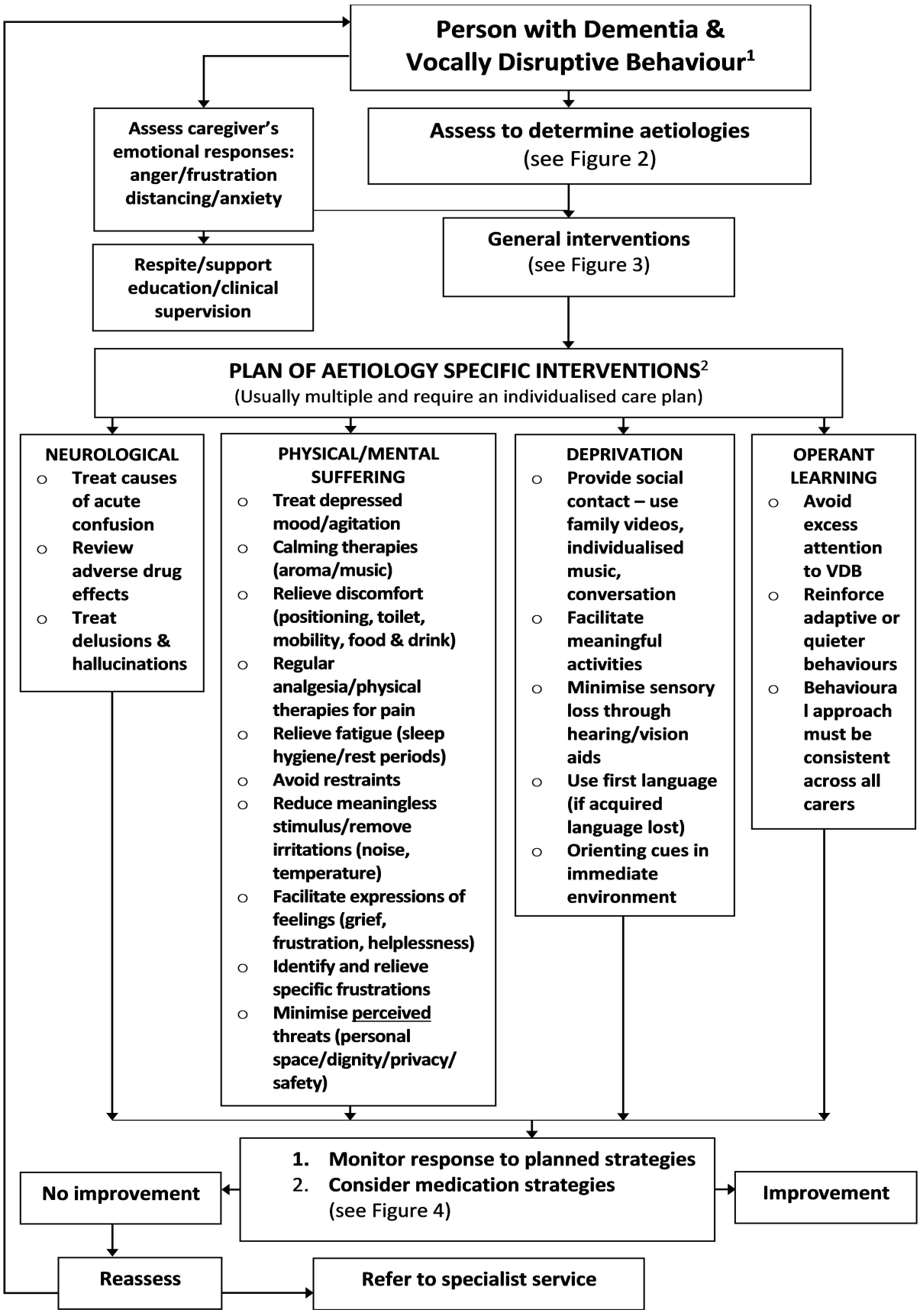
In summary, various types of vocal disruption are common in dementia, with significant and often distressing consequences. Recognised expert guidelines for addressing vocal disruption are limited. Reports on the outcomes of interventions are limited in number and quality. Environmental modifications to support orientation[49] as well as education and support for staff to apply strategies that address resistance during personal care[48] may help to reduce vocal disruption. The use of atypical antipsychotics is not recommended for vocal disruption that is not underpinned by psychosis.

If discomfort, reduced-stress thresholds and operant conditioning contribute to vocal disruption, interventions focusing on these elements will likely be the most successful in reducing this distressing symptom. McMinn and Draper[3] suggest an approach where these factors are considered (See *Figure 11.1*). Assessment of the person with dementia with the Typology of Vocalisation scale and a medical evaluation are recommended to determine possible aetiologies (e.g. pain, depression, environmental stressors). Effective pain management should always be considered when addressing BPSD[19]. Merging technologies may further assist identification of pain in people who vocalise but are unable to express themselves verbally[12].

Vocalisations are not necessarily inherently disruptive, and defining them as such is perception-based[1, 17]. Where vocal disruption is not harmful to self or others, not receptive to interventions and no specific underlying cause can be identified, supporting others to change their perception and understanding may be beneficial. A lack of sound evidence should not prevent clinicians from considering strategies to reduce vocal disruption on an individual basis[53]. Good clinical practice suggests tailoring psychosocial interventions to individuals. For example, a person-centred approach[54] may indicate that for one person reducing vocal disruption may result from changing their personal care routine to include a relaxing bath at their preferred time, for another it may be implementing a tailored pain management regimen and for a third, it may be regular time outdoors in the garden. The need for a multidisciplinary, individualised and multifaceted approach is stressed.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.

Figure 11.1 Practice Guidelines for Vocal Disruption in people with dementia. Additional information referred to in this figure can be obtained from McMinn and Draper[3]. Reprinted by permission of the publisher Taylor & Francis Ltd, http://www.tandf.co.uk/journals



MODULE 12: Wandering

Key messages

- Wandering in dementia is frequently distressing for a person living with dementia and their carers; the safety risks are significant.
- Consensus on a unifying definition has not been reached. An operational definition proposes that wandering occurs over time and space and includes four patterns of ambulation: restlessness, agitation, aberrant motor activity, and night-time disturbances.
- Prevalence rates for wandering in dementia reportedly range from 12% to 63% in the community and 5% to 100% in residential care.
- Adverse effects of wandering are numerous. Absconding and becoming lost can have severe negative consequences including injury and death.
- By contrast, independent but safe wandering can potentially have positive effects.
- The crucial task for the clinician is to understand what the wandering means for the individual person although this can be difficult to determine.
- Some evidence was provided for environmental modifications to support orientation.
- The inappropriate use of restrictive practices increase the person's risk of harm and is not recommended.
- Treating underlying depression or pain should be considered.
- Sound evidence of the effectiveness of interventions to prevent or reduce wandering is lacking.
- Comprehensive assessment must include aspects of the person with dementia that enable tailored strategies, to support an individual approach.

Before you move on, have the following been done?

1. A risk assessment to identify any immediate risks to the person with dementia or others within the care environment
2. A comprehensive person-centred assessment that considers the following key aspects:
    - the person living with dementia
    - the changed behaviour(s) or psychological symptom(s): details, frequency, severity, precursors and sequelae
    - the care partner/care staff
    - the care environment (See *Appendix 4* for further details)
3. Checked for, and treated, reversible causes of the BPSD

    (See *Module 1* for further details)

Wandering summary

What is wandering and what does it look like in dementia?

The construct of wandering has been used to summarise a range of observable motor behaviours. An operational definition, based on observed patterns of movement in aged care home residents with dementia proposes that wandering can manifest in patterns of lapping, pacing, random or direct locomotion. A descriptive typology of wandering has also been outlined, as opposed to a single definition. People living with dementia have described wandering as "searching" which can occur after distraction or memory lapses interfere with reaching a desired destination.

Causes of wandering

Recognising potential antecedents to wandering can aid in prevention. Wandering can have different meanings and causes for people living with dementia. These may include:

- a response to pain, infection, discomfort or increased confusion
- looking for assistance, a toilet, company or a familiar face/place
- habitual pattern of activity
- medication reaction
- underlying depression, anxiety, delusions or hallucinations
- perceived escape from trauma, threat or an unpleasant situation.

Wandering has been associated with greater cognitive and functional impairment, greater gait and balance impairment, conscientiousness, poorer response to stress, younger onset of dementia, and lower level of awareness.

Differential diagnosis

Wandering is often subsumed within the syndromes of agitation, restlessness and night-time disturbances. The term is also used interchangeably with aberrant motor activity. A medication review should be undertaken to exclude adverse effects of antipsychotics, hypnotics or stimulants.

Measuring wandering

Wandering in dementia is differentiated by pattern, severity, rate, duration, peak period of occurrence and frequency. The Revised Algase Wandering Scale for Long Term Care (**RAWS-LTC**) and the community version (**RAWS-CV**) are the only assessment tools specifically designed to measure wandering. The Neuropsychiatric Inventory (**NPI**), the Cohen-Mansfield Agitation Inventory (**CMAI**), the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) Behavior Rating Scale for Dementia and the Dementia Behavior Disturbance Scale (**DBDS)** include items pertaining to wandering.

Prevalence of wandering

Prevalence rates for wandering reportedly range from 12% to 63% in the community and 5% to 100% in care homes. The wide disparity in prevalence rates can be partly attributed to the imprecise definition of wandering and the variety of measurement tools used in reporting. The rate and duration of wandering tends to increase as cognition declines but then subsides in late-stage dementia.

Effects of wandering

Wandering has been associated with high carer burden and anxiety around the associated risks as well as earlier admission to residential aged care services (RACS). Adverse effects of wandering include falls and subsequent injury and/or fractures, weight loss, resident to resident violence, the inappropriate use of restrictive practice and social isolation. Absconding and

becoming lost present additional safety risks, at times resulting in death. Conversely, providing opportunities for independent but safe walking can potentially be therapeutic and improve wellbeing.

Addressing wandering

The first step is to understand the person and what underpins the wandering for the individual. Clearly, addressing the cause of the wandering is crucial, although finding the cause is not always possible. Comprehensive assessment to exclude comorbid illness and pain as well as psychological assessment to identify underlying mood or psychotic symptoms is essential. It is important to identify the significant aspects of the wandering including the issues for the person with dementia versus the issues for carers and/or staff.

Psychosocial and environmental interventions

- Good quality evidence for psychosocial/environmental interventions is lacking and only three studies met our quality criteria for inclusion.
- Some evidence was found for the effectiveness of supportive environmental changes in residential care.
- Negative outcomes were reported for tailored music therapy and interactive robot sessions.

Biological and pharmacological interventions

- Clinical guidelines recommend against the use of benzodiazepines, z-drugs, antidepressants, antihistamines and antipsychotics for wandering as these drugs can worsen confusion and increase the risk of falls.
- Antipsychotics can also exacerbate motor restlessness through akathisia.
- No biological/pharmacological intervention studies met our quality criteria for inclusion.

Technologically mediated devices

Technological monitoring devices have been developed in attempts to reduce the risks associated with wandering in the community and RACS. The level of evidence to support the use of such devices is low and further real-world research is required to demonstrate their effectiveness.

Limitations

Wandering is multifaceted and it has been understudied. This has limited the development of effective strategies to date. There is a lack of sound intervention research to guide clinicians and carers in strategies to address wandering. Overlapping of symptoms with other BPSD can mean that a diagnosis of wandering may not be straightforward, and wandering is rarely studied in isolation, further hindering the development of effective strategies.

Conclusions/Principles of care

- Treating underlying pain and depression should be considered.
- The inappropriate use of restrictive practices is not recommended.
- Although the scientific evidence is limited, strategies to reduce wandering should be considered on an individual basis.
- Some evidence for environmental modifications to support visual orientation is outlined.

What is wandering and what does it look like in dementia?

Wandering can be one of the most challenging and problematic co-morbid behaviours of dementia[1]. Researchers and clinicians have failed to reach a consensus on a unifying definition. The construct of wandering has been used to encapsulate a range of observable behaviours that involve walking and locomotion as well as the use of assistive devices or wheelchairs[2, 3]. While wandering is differentiated from other changed behaviours including restlessness[4], physical agitation[5] and night-time disturbances[6] symptoms overlap. Wandering is conceptually different but has been used interchangeably with broader terms such as aberrant motor activity/ behaviour and motor disturbance[3, 7]. Wandering has also been referred to as 'getting lost behaviour'[8, 9].

The DSM-V-TR does not provide a clear definition for BPSD[10]. The World Health Organisation International Classification of Diseases, Eleventh Revision[11] includes "clinically significant wandering that puts the person at risk of harm" under a classification for "behavioural or psychological disturbances in dementia that are severe enough to represent a focus of clinical intervention" under Neurocognitive disorders, Dementia[11; ICD 11, 6D86.6, Parent: 6D86].

An operational definition has been proposed to aid clinical recognition, research validity and the standardisation of language in relation to wandering[2]. According to this definition, wandering occurs over time and space and can manifest in the following patterns of ambulation:

- Lapping - circular locomotion
- Pacing - locomotion back and forth between two points
- Random - locomotion without a direct path and with multiple directional changes
- Direct - locomotion from a point to a destination without diversion (also termed non-wandering because no deviation occurs between point A and point B).

People with lived experience of dementia describe "walking with a purpose", such as exercise or being near nature, and "searching" which can occur after distraction or memory lapses interfere with reaching a desired destination[12, 13]. People with dementia, carers, and others may prefer the use of alternative, less stigmatising terms, such as 'exiting'[14], 'wayfinding'[15] or 'walking with purpose' rather than wandering[16].

Wandering has been defined clinically as 'repetitive locomotion that makes one susceptible to harm due to its incongruence with boundaries and obstacles which may culminate in exiting, elopement and/or becoming lost'[17]. Wayfinding is more complex and relies on multiple cognitive skills, including choosing a destination, planning a route as well as executing strategies to follow a path and reach the end point[18, 19]. Wayfinding impairments can occur at any disease stage but wandering tends to occur in the later stages of dementia. Elopement occurs when wandering 'around' becomes wandering 'away'[20]. A descriptive typology for wandering, as opposed to a single definition, has also been outlined[21].

- increased motor activity
- perceived aimless walking
- appropriate but excessive walking
- attempts to leave home or current place of residence
- night-time walking
- inappropriate walking
- being brought back home
- trailing.

Spatial disorientation can cause disrupted gait patterns e.g., slowing-down or speeding-up, taking shorter or longer strides, increasing or decreasing steps and trigger psychophysiological stress responses such as increased heart rate and/or anxiety[22]. In turn, these can predict negative outcomes such as falls, injury, getting lost or elopement[23]. People with Alzheimer's Disease (AD) may:

- have difficulty forming mental representations to support navigation i.e., impaired cognitive mapping and orientation strategies,
- have difficulty planning how to get to destinations,
- find route instructions challenging,
- use ineffective wayfinding strategies,
- be easily distracted by other stimuli in the environment or irrelevant visual information,
- have difficulty identifying errors and self-correcting,
- experience impaired decision-making or anxiety at points such as crossroads and/or
- have difficulty remembering why or where they are going and recognising their destination.

Causes of wandering

Wandering is multifaceted. It can vary between and within individuals in its expression. Biomedical, psychosocial and person-environment factors are proposed as contributing factors. Attempting to understand the cause of the wandering will enhance the clinician's ability to reduce wandering. Wandering in people with dementia can occur as a consequence of many potential antecedents. See *Module 1, Table 1.2* for a list of contributory factors. In addition to cognitive deficits[24, 25], wandering can have different meanings and causes for each individual[24-29]. These may include:

- attempting to navigate/explore a changed or unfamiliar environment e.g., when traveling, moving into a new home, transition to RACS
- unmet needs such as hunger, boredom, looking for a toilet, incontinence, soiled clothing and looking for assistance/staff
- lack of dementia-friendly environmental design elements in care settings and communities[28]
- looking for a loved one, a pet or a person they recognise, including those who are no longer living[30]
- a wish to return to a familiar environment including places from their past such as the person's childhood home or other place they have lived[20]
- feeling a need to leave because they have a job to do or a role to perform e.g., going to work, picking-up children from school
- social isolation, loneliness
- excess energy or lack of regular physical activity
- disruption to diurnal rhythm, dreams or confusing night with day[29]
- being prompted by delusions or hallucinations e.g., believing that 'this place' is not my home
- escaping from perceived imprisonment or persecution
- reliving historical trauma, history of incarceration, institutionalisation
- misperceiving incidents in the environment as frightening, prompting a need to flee
- avoiding unpleasant stimuli or routine or problematic interactions/relationships with other residents, staff
- a habitual pattern of activity[31]
- a reaction to medication[32]
- a symptom of stress, anxiety and/or depression[33]
- a response to pain, infection or discomfort such as constipation[30, 32-34].

Various explanations have been suggested for the different patterns of wandering, some of which are related to cognitive deterioration. Changes to structures in the medial temporal lobe [9], parietal lobes, frontal lobe, the hippocampus and amygdala are associated with spatial navigation and orientation deficits which can result in disrupted wayfinding and becoming lost[24]. Spatial navigation and orientation deficits differentiate AD neuropathology from frontotemporal dementia (FTD) and other dementias[24]. Cognitive deficits in AD such as impairments in spatial navigation/orientation, visual motion processing, path integration, working memory, attention, executive function can contribute to wayfinding difficulties and getting lost[15, 25].

Wandering has been associated with greater functional impairment, greater gait and balance impairment, conscientiousness and poorer response to stress[26]. Wandering is also associated with younger onset of dementia, greater cognitive impairment[35] and lower level of awareness [36]. Recognising potential antecedents to wandering can aid in prevention. The following may indicate that the person is at risk of a negative outcome[23, 37]:

- persistent walking
- poor gait
- stating their intention to leave a safe space
- pacing and waiting near, and/or attempting to unlock or open, exit doors
- requesting staff, visitors or others unlock exit doors/gates, sometimes with plausible explanations
- preparing to go outside e.g., putting on more clothing, seeking their jacket/bag/keys/purse/wallet, looking for their car
- packing-up belongings
- contacting/calling someone to open a door or to pick them up
- frequent glances at their watch
- being drawn to events or objects outside i.e., people walking past the home, animals.

PRESENTATION

Eddie is a 65 year old Aboriginal man who moved to Katherine from a remote NT community when he was a young man. He lived with his wife until she died recently. While raising their family of five children, they maintained strong connections with friends and family in Eddie's original community. His connection to Country has remained very important to him.

Family and community members have been supporting Eddie in the family home with the assistance of Aboriginal-specific services. Since their mother's death it has become evident to the family how much she was compensating for Eddie's functional deficits due to his dementia. On three occasions in the past month Eddie has been found after dark some distance from home, underdressed for the weather and distressed. On the most recent occasion, a concerned passer-by alerted police after Eddie was unable to provide his address or contact details for his family. When police approached Eddie became uncooperative and verbally aggressive. Police ultimately located Eddie's daughter who collected him from the local police station to take him home.

Differential diagnosis

Presentation of wandering is not always consistent in those with dementia, and it is often subsumed within the syndromes of agitation and restlessness[38]. Restlessness and a physical, non-aggressive form of agitation have been used interchangeably to refer to disordered motor behaviours or wandering[39]. Wandering in the form of restlessness, with a compelling need for movement or pacing, has been linked to side-effects of psychotropic medications, particularly akathisia with antipsychotics[4, 40]. A medication review should be undertaken to exclude adverse effects of antipsychotics, hypnotics or stimulants.

Measuring wandering

The following are widely used assessments of dementia-related wandering[41].

- The Revised Algase Wandering Scale for Long Term Care (**RAWS-LTC**) is a 19-item

scale which was designed for formal carers to rate wandering according to three subscales: persistent walking, spatial disorientation and eloping[42].

- The community version **RAWS-CV** includes five subscales with a total of 39 items: persistent walking, spatial disorientation, eloping behaviours, routinised walking and negative outcomes. Respondents rate observations as they occurred in the preceding week[18]. The RAWS is a reliable and valid tool that has been widely used for research purposes, however further evaluation is indicated to extend its clinical applicability[42].
- The aberrant motor subscale of the carer-rated Neuropsychiatric Inventory (**NPI)** includes a question on pacing[43] which rates the frequency and severity of the person's motor behaviour and the carer's subsequent distress. The reliability and validity of the NPI overall is well established[44]. The NPI-Clinician (**NPI-C**)[45] has added an additional question relevant to wandering in the revised aberrant motor disturbance subscale.
- The Cohen-Mansfield Agitation Inventory (**CMAI**) is a 29-question, three-factor carer questionnaire that assesses the frequency of a given behaviour as observed in the preceding fortnight[46]. The physically non-aggressive subscale of the CMAI includes an item relevant to pacing and aimless wandering. Algase and colleagues, however, indicate that physically non-aggressive behaviours and wandering are overlapping, but not equivalent, phenomena[39].
- The clinician-administered, carer-informed 48-item Consortium to Establish a Registry for Alzheimer's Disease (**CERAD**) Behavior Rating Scale for Dementia (**C-BRSD**) includes three items relevant to wandering[47]. Further evaluation is indicated regarding its use as a screening tool for wandering[48].
- The 28-item carer-rated Dementia Behavior Disturbance Scale (**DBDS**)[49] has items addressing pacing, getting lost outside, wandering at night and aimless wandering. Further evaluation is indicated regarding its use as a screening tool[50].

Multiple desktop-, tablet- and smart phone-based apps that assess navigation and orientation ability using virtual wayfinding tasks are undergoing reliability and validity testing with older adults and people living with dementia and may be appropriate in clinical settings[24, 51-53].

Prevalence of wandering

The prevalence of wandering in dementia is difficult to assess due to its imprecise definition and the variety of measurement tools used. Accordingly, the literature reports wide disparity in prevalence rates for those living in residential settings ranging from 5% to 100%[54-56] and 12% to 63% in community-based samples[9, 26, 57]. Wandering is reportedly more prevalent in younger people with dementia[36, 56, 58]. Those with AD are more likely to wander than those with vascular dementia (VaD)[55]. People with FTD reportedly have a greater tendency to pacing and lapping whereas those with AD are more inclined to engage in a higher proportion of random locomotion[24].

It has been demonstrated that the rate and duration of wandering increases as cognition declines, but then subsides in late-stage dementia[3]. Frequency of wandering has been linked to greater independence in mobility and dependence in activities of daily living (ADLs) associated with hygiene[23, 59]. These findings are consistent with the Need-driven Dementia-compromised Behaviour (NDB) model which postulates that cognitive impairment and mobility directly impact on the expression of wandering[3].

Effects of wandering

Wandering can be challenging for formal and informal carers, as well as dangerous for the person with dementia. Absconding (or elopement), becoming lost and going missing pose significant safety risks, at times resulting in death[8, 60-62]. Those at risk of wandering and becoming lost should have identification on them with their address and carers' contact details[63]. In residential aged

ASSESSMENT

To reduce Eddie's wandering which is putting him at risk, potentially contributing factors must be identified:

- *Investigate possible pain/discomfort and/or illness/infection/constipation*
- *Medication review: interactions, dosage, recent changes, adverse effects*
- *Assess the immediate environment for potential triggers*
- *Searching for his wife or other family members*
- *Searching for his childhood home environment*
- *Exclude underlying depression, particularly with recent significant loss*
- *Lack of stimulation/boredom*
- *Changes to the physical environment*

Assessing the situation:

- *Encourage Eddie to express his needs and concerns as far as he is able.*
- *Arrange medical and pharmacological review to exclude potentially reversible contributing factors.*
- *Directly observe and note Eddie's behaviour preceding wandering incidents and also on the occasions when he makes no attempt to leave home.*
- *Ask community workers who have become familiar with Eddie if they have identified situations which provoke his wandering.*
- *Consult Eddie's life history for further information.*
- *Consult family members to identify possible strategies that may discourage Eddie's wandering attempts.*

care services (RACS), a 'whole of facility' approach to resident safety is required. All visitors and staff, including those indirectly involved in resident care, must be aware of the potential risks and necessary precautions to reduce the likelihood of residents wandering[20]. A person who is attempting to exit a RACS will likely incite interest from others with dementia, which may prompt additional restlessness and attempts to abscond in other residents.

Wandering has behavioural, emotional, and social consequences[25]. It has been identified as a salient factor for earlier entry into RACS, due to increased carer burden and anxiety around the associated risks[64, 65]. The person with dementia can experience emotional discomfort, anxiety, insecurity and overwhelm when they are lost or disoriented[25], even within a safe environment[18]. Likewise, their distress may increase when they are barred by locked doors and/or agitated by alarms or surveillance devices[66, 67]. Adverse effects of wandering include falls and subsequent injury and/or fractures[68], weight loss, resident to resident violence, communicable disease infection risk for self- and others[28, 69], the inappropriate use of restrictive practices[69-71] and social isolation[3].

Conversely, safe walking can have positive effects through exercise by improving circulation and oxygenation[72]. Further, independent but safe walking can potentially be therapeutic in that it can improve wellbeing and agency, stimulate appetite, relieve boredom, improve mood as well as encourage feelings of empowerment and control[12, 35, 67]. While safe walking within the least restrictive environment has long been the goal of many strategies, recent times have seen a shift to promote greater independent mobilisation in the presence of impaired wayfinding[67].

It has been suggested that interventions to reduce wandering should only be implemented where the wandering negatively affects the physical or emotional health and well-being of the person with dementia or others, or when safety of the person or others is an issue[8, 64, 69, 71, 73]. Managing risk by limiting the person's liberty reduces personal autonomy and constitutes restrictive practice, which raises concerns[74]. See *Module 2* for further information on restrictive practices.

Results

A systematic literature review to set criteria (see *Appendix 5*) yielded only three psychosocial and environmental intervention studies, and no biological or pharmacological studies, that met our quality criteria for inclusion. As the evidence from studies assessing wandering published before 2012, previously included in the *BPSD Guide* (2012), did not meet our updated quality criteria none of these studies have been retained.

Addressing wandering

The first step is to understand the person and what underpins the wandering[62]. Clearly, addressing the cause of the wandering is crucial, although this can be difficult to determine when the person with dementia may be unable to express their needs verbally. Comprehensive assessment to exclude comorbid illness, pain and discomfort and psychological assessment to identify underlying mood or psychotic symptoms is essential. Clinical judgement in this situation frequently relies on historical factors, nonverbal cues and/or informant knowledge. *Appendix 4* provides suggested questions to facilitate comprehensive assessment. Assessment of social health, opportunities for physical activity/exercise, environmental factors such as visual cues, lighting, temperature and odours as well as risk factors for wandering should also be considered[8, 23, 75]. Additionally, it is important to identify the significant aspects of the wandering such as intensity, duration, peak period, boundary crossing and the issues for the person with dementia versus issues for carers and/or staff.

STRATEGIES/OUTCOMES

- *In discussions with the family it became evident that Eddie sometimes forgets that his wife has died. When he does not remember this and he cannot find her in their home, Eddie becomes distressed. Family members have put their numbers in Eddie's phone and reminded him to call whenever he needs help, but Eddie is apparently unable to manage this.*
- *Consultation with family and community workers indicated that Eddie was also more restless after phone contact with his younger brother who still lives in the town near their childhood home.*
- *Although Eddie's daughters are grieving they are very supportive. They are feeling the stress of caring for their father while meeting the needs of their own families and they are feeling increasingly concerned when leaving him at home alone. They are at a loss as to how to best support their father in his grief when this is complicated by dementia.*
- *Eddie's multiple comorbid medical conditions are contributing to his mobility and vision limitations, and the family are concerned that he may fall when he wanders from home. Eddie tends to forget to use his walking stick.*
- *Family and community members are experiencing greater difficulty communicating with Eddie as he increasingly reverts to his traditional language.*
- *Eddie's history, as outlined by the family, explained his reaction to contact with the police. With the progression of dementia, traumatic experiences from his past have exacerbated his fear of authority figures.*
- *The community workers and family have little understanding of the association between Eddie's dementia and his wandering. Information was provided to increase their awareness of potential triggers for Eddie's wandering and the impact of historical trauma and discrimination within the context of dementia.*
- *Eddie's younger brother travelled to Katherine to participate in a family/community meeting. The family determined that Eddie may benefit from staying with his brother and wife.*
- *Eddie responded well to returning to Country and the company of the older family members and community.*
- *The additional contact with community meant that Eddie was afforded greater supervision, companionship and support for his grief.*
- *Aboriginal services in Katherine referred Eddie to a service provider in the town where Eddie's brother lives for additional support.*

Psychosocial and environmental interventions

Good quality evidence for psychosocial and environmental interventions is lacking. Additional studies of staff education[76], multisensory room[77], doll therapy[78], pet therapy[79], aromatherapy[80], an individualised OT intervention[81], a multicomponent intervention[82] and a tailored physical activities program[5] across various care settings reported mixed outcomes for wandering but did not meet our criteria for inclusion. All studies were of modest quality and recommendations cannot be made on the basis of these one-off studies alone. Environmental distractions may sometimes reduce wandering but the evidence is also limited[83].

Models of care
Quality of research study: 1/1 moderate
Outcome of study: 1/1 some evidence

In a small RACS study of people with moderate to severe dementia, the number and the average

duration of episodes of wandering during 24-hour periods significantly decreased three months after environmental changes to support orientation, compared with the previous three months[84]. Changes included painting walls light beige, installation of 'skylike' ceiling tiles, coordination of staff clothing, lighting changes to better differentiate day and night, installation of oversized clocks and streaming soothing music[84].

Music
Quality of research study: 1/1 moderate
Outcome of study: 1/1 negative

A large RACS trial compared two weeks of small group tailored music therapy sessions delivered by certified music therapists, using preferred music and musical instruments, with usual care. AWS wandering scores did not significantly change in people with moderate to severe dementia[85].

Animal-assisted
Quality of research studies: 1/1 strong
Outcomes of studies: 1/1 negative

A significant *increase* in RAWS wandering scores was demonstrated for thrice weekly group sessions with a *Paro* interactive robot compared with interactive reading at 5 weeks in a small RACS study[86].

The limited psychosocial/environmental evidence for wandering is not unexpected. In literature published prior to 2012, environmental interventions often took the form of subjective barriers which may present ethical issues[87]. Subjective barriers generally involved two-dimensional visual manipulation of the environment, including mirrors and the use of concealment and camouflage techniques. See *Appendix 2* for interventions reported above.

Biological and pharmacological interventions

The use of pharmacological or physical restraints to address wandering and excessive motor activity is widely accepted as unethical hence, a lack of biological and pharmacological intervention studies is not unexpected. Furthermore, wandering has been identified as a behaviour/symptom associated with dementia that does not justify the use of antipsychotic medications[88]. Although agitation with motor restlessness can be secondary to depression, clinical guidelines recommend against use of antidepressants for agitation.

Sedation to control wandering (sometimes called chemical straitjackets), such as with benzodiazepines, z-drugs (i.e. zopiclone, eszopiclone, zaleplon, zolpidem), antihistamines or antipsychotics, can worsen confusion, increase the risk of falls and, in the case of antipsychotics, may exacerbate motor restlessness through akathisia[88, 89]. See *Module 2, Table 2.3* for side effects associated with antipsychotics. Pain management should always be considered when addressing any BPSD. Analgesics, even as simple as paracetamol 1 gram three times per day, may reduce agitation with motor restlessness if underlying pain is present.

Technologically mediated devices

In response to the risks associated with wandering, devices to disguise exits and a range of technological devices are available to alert carers when people with dementia attempt to exit. Such devices have been developed for use in the community and RACS. Wearable sensors and monitoring devices have been implemented in an attempt to reduce the risk of elopement and/or locate the person where wandering has occurred. The level of evidence to support the use of such devices in real-world settings is low. Further research is required to demonstrate their effectiveness[90, 91] and increase awareness[92]. Trials are ongoing[93-95]. Sensing technology is now common and its application for assessment and monitoring of treatment response will increase in the future[90]. Such technologies should only be implemented after careful consideration of the autonomy of the individual with dementia and the practical, ethical and legal ramifications of their use[90, 96].

Limitations

There is a paucity of sound intervention research for addressing wandering. The evidence for sustainability of effects is limited with no reports of long-term follow-up and just one study indicating no benefit at two weeks post-intervention follow-up[85]. Wandering is multifaceted and interventions have been understudied, limiting the development of effective strategies. A diagnosis of wandering in dementia may not be straightforward, due to overlapping of symptoms with other BPSD. Wandering is often subsumed within other categories such agitation and aberrant motor behaviour, is rarely studied in isolation, further hindering the development of effective strategies. Problems also occur when multiple, individual BPSD subscale scores are analysed in the same study and multiple comparisons are not accounted for.

Conclusions/Principles of care

In summary, wandering in dementia can have significant and dangerous consequences. Recognised expert guidelines are limited for addressing wandering in those with dementia. The inappropriate use of restrictive practices is not recommended however, treating underlying depression or pain should be considered. Sound research to guide clinicians and care partners in psychosocial interventions is limited[14, 97, 98] but this should not prevent clinicians from considering strategies to reduce wandering on an individual basis[99]. Good clinical practice suggests tailoring psychosocial interventions to the person with dementia. For example, a person-centred approach[100] may indicate that for one person reducing wandering may result from discovering a distracting activity that provides pleasure at high-risk times of the day, for another it may be reviewing their pain management and for a third, it may be arranging outdoor walks with a carer or family member to alleviate feelings of imprisonment. Environmental interventions to support visual orientation provide some evidence. A multidisciplinary, individualised and multifaceted approach is recommended.

For references cited in this Module see *Appendix 1: Reference lists for each Module* available in electronic format.