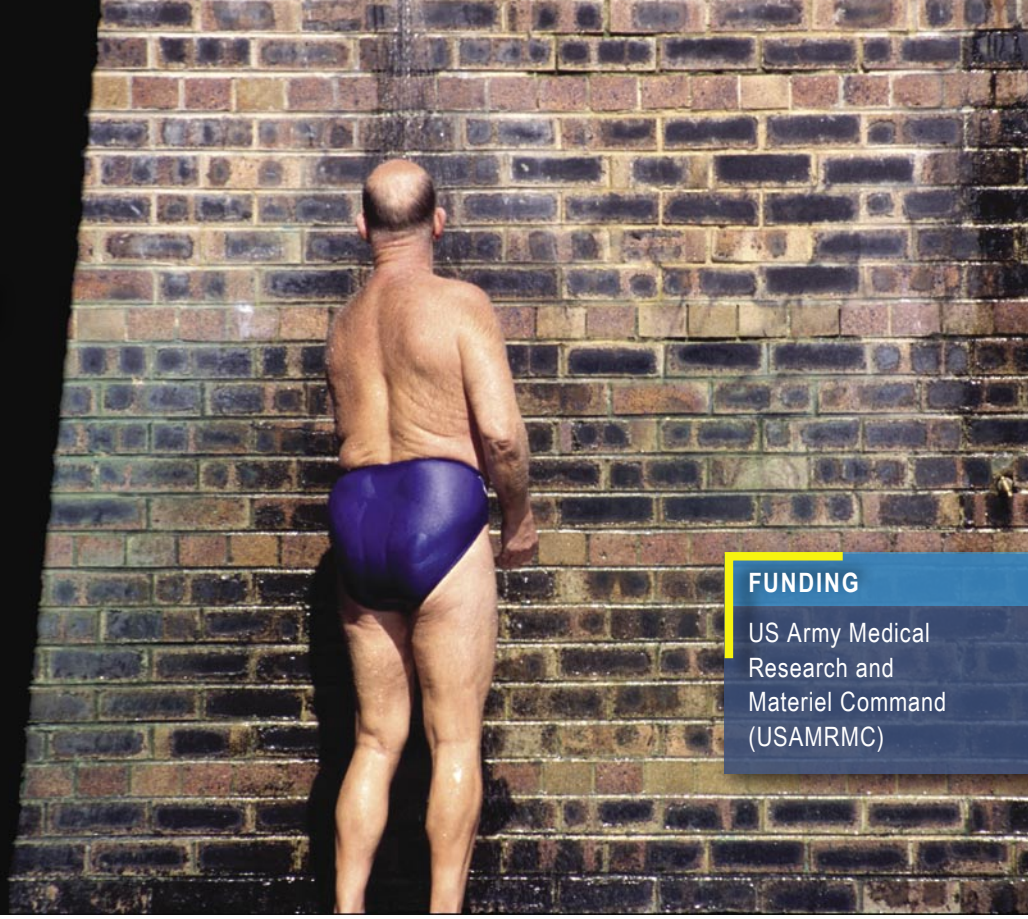
New *treatments for prostate* cancer



FUNDING

US Army Medical Research and Materiel Command (USAMRMC)

With a new case diagnosed every hour and 2500 deaths each year, prostate cancer has become the biggest cancer threat facing Australian men. Could genetic 'errors' play a role in the spread of prostate cancer throughout the body, and is it possible to produce new drug delivery methods with fewer side effects? Professor Pamela Russell, AO, and her team at the Prince of Wales Hospital's Oncology Research Centre (research arm of the Institute of Oncology and affiliated to UNSW's Faculty of Medicine), are investigating.

With new funding from the US Army Medical Research and Materiel Command (USAMRMC), which has a mandate to 'provide solutions to medical problems' for the military, both on and off the battlefield, Pamela Russell and her team are expanding their research into these two areas.

"The first study will investigate the role of genes thought to be involved in the spread of prostate cancer to other, more life-threatening parts of the body," said Pamela Russell, "while the second project is concerned with new treatments for prostate cancer that seek to target cancer cells while ignoring their healthy neighbours."

Researchers already know that errors in genetic material or DNA are responsible for all cancers. Previous studies at the Oncology Research Centre have shown that additional genetic errors might also explain why some prostate cancers metastasise or spread throughout the body, while the vast majority of prostate cancers which lack these errors remain slow growing and less likely to cause harm.

According to Pamela Russell, one of the main suspects involved in the spread of prostate cancer is a gene called p53. "It's thought that genetic mistakes or mutations within certain regions of the p53 gene might make the cancer more likely to spread to the bone – the most common site of metastasis in advanced prostate cancer patients. Using human prostate cancer cells grown in the laboratory, our research involves creating a series of artificial prostate cancers, each carrying a different error within the p53 gene.

"We should then be able to determine whether the genetic mutation the p53 gene acquires allows the cancer cell to spread and grow inside the patient's bone," she said.

"In the second project, we may have found a way to make chemotherapy more effective and to protect patients from some of the debilitating side-effects of the cancer drugs.

"Ever since chemotherapy for prostate cancer was developed more than 50 years ago, doctors have been limited by the amount of drugs they could give a patient because of the toxic effect on other cells. With our colleagues from CSIRO, in particular Dr Peter Molloy, we have now enlisted a gene to help target chemotherapy directly to the prostate and leave healthy cells in other organs unscathed.

"We use a cancer drug which requires chemical conversion to make it active. This 'pro-drug' has no side-effects and is unable to kill either normal or cancer cells. The trick has been to create a 'genetic switch' which only works inside prostate cells to reactivate the pro-drug into its lethal form," said Pamela Russell.

The three-year study involves inserting this artificial gene into a patient's prostate cancer cells, using a friendly virus to carry the genetic cargo. So far, the results have been impressive with growth rates significantly reduced in prostate cancers grown in the laboratory.

With a Phase One clinical trial for men with advanced prostate cancer planned for 2004, the Oncology Research Centre shows how quickly laboratory research can be translated into clinical practice.

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