New target for cancer therapy identified

September 21 2006

A new target for cancer therapy has been identified by Monash University scientists investigating the cell signalling pathways that turn on a gene involved in cancer development.

A team led by Associate Professor Jun-Ping Liu, from the Department of Immunology, has identified two proteins that are involved in stopping the gene from producing a protein called telomerase that is essential if cancer cells are to proliferate.

Telomerase plays a key role in controlling the life span of cells by modifying structures called telomeres that are found at the end of chromosomes.

Although it is involved in tumour development, telomerase is also found in modest quantities in most cells. It is plentiful in stem cells where it keeps the telomeres long, allowing the cells to keep dividing without limit which is necessary for the repair of damaged and worn out tissues throughout the human body.

However, studies have shown that telomerase also plays a key role in the formation of cancerous tumours. "It's the best indicator of cancer – 85 per cent better than any other tumour marker," Associate Professor Liu said. "What's more, telomerase is not associated with benign tumours; it's a marker for malignant tumours only.

"If we can control the production of telomerase we can prevent the immortality of cancer cells and therefore cancer formation."

Associate Professor Liu and his colleagues have been investigating breast cancer cells to identify the molecular signalling that is required to turn on, and also inhibit, the gene that produces telomerase. They have found two proteins Smad3 and c-Myc that are involved in turning off telomerase production. Their findings are published in the current issue of the Journal of Biological Chemistry.

"It's significant to find inhibitors of telomerase and we have found, for the first time, the pathway that inhibits telomerase in human cells," Associate Professor Liu said.

"This reveals an important mechanism for developing anti-cancer agents that mimic these proteins and thereby inhibit the production of telomerase. "

Source: Monash University