

Protein found to regulate spread of pancreatic cancer cells

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Researchers at Queen Mary, University of London have identified a new protein that makes pancreatic cancer cells less 'sticky' and therefore less able to attach to and invade other tissue.

The [protein](#), known as S100PBP, does this by suppressing a second protein called cathepsin Z. The research team has shown that cathepsin Z makes [pancreatic cancer](#) cells sticky, allowing them to [spread](#) to their surrounding environment. Prior to this study nothing was known about the function of S100PBP in the body or the role that cathepsin Z plays in pancreatic cancer.

The findings, funded by the UK charity, Pancreatic Cancer Research Fund (PCRF), are reported today in The [American Journal of Pathology](#).

Lead researcher Dr Tatjana Crnogorac-Jurcevic of Barts Cancer Institute at Queen Mary said: "We believe these findings are significant. A greater understanding of the role these proteins play in the adhesion and spread of pancreatic cancer to other organs, which is almost always the case in this deadly cancer, could help us to develop novel preventive and therapeutic targets."

Pancreatic cancer has the worst five year survival rate of any common cancer – 3 per cent - and this figure has not improved in forty years. With no early diagnostic test available, and symptoms that are often mistaken for less serious conditions, the majority of sufferers are diagnosed too late for surgery - currently the only possible curative

option.

The team found that the production of cathepsin Z is regulated by S100PBP. When large quantities of S100PBP are present, less cathepsin Z is produced by the cancer cells, limiting their spread. PhD student and co-author Kate Lines said that the team hopes to secure further funding to progress this line of research: "We're especially keen to find out exactly how S100PBP controls the levels of cathepsin Z, so we can try to prevent its increase in [cancer cells](#)".

"Pancreatic cancer is an extremely complex cancer, and these findings add to the growing bank of knowledge regarding the number and roles of proteins involved in its aggressive spread throughout the body. We hope that these newly discovered proteins may ultimately prove to be key in paving the way for new therapies that could make a real difference to patients' prognosis."

Provided by Queen Mary, University of London

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