PIM1 accelerates prostate cancer cell motility by phosphorylating actin capping proteins

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Video Byte

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Abstract

Prostate cancer is one of the prevalent forms of cancer in men around the world. Prostate tumors become especially fatal when they spread to one and other vital organs. The main forces driving cell motility are the constant polymerization and depolymerization of actin filaments which lead to changes in cellular protrusions. This process is regulated by actin-binding proteins, such as capping proteins, which bind to the fast-growing ends of actin filaments. A new study shows that these capping proteins are substrates for cancer-inducing PIM kinases, proteins whose overexpression promotes cancer cell survival and motility. Phosphorylation of capping proteins decreases their ability to protect actin filament ends from disassembly, leading to enhanced cell motility. while PIM inhibitors or capping protein mutations have the opposite effect, reducing cell migration. This novel mechanism helps explain how PIM kinases enhance the motility and metastasis of prostate cancer cells and supports the development of PIM-targeted therapies to treat prostate cancer.