

# New piece found in colorectal cancer puzzle

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Prostasin, a relatively unknown protease enzyme expressed in most epithelial cells, may play a role in the genesis of colorectal cancer. Researchers writing in the open access journal *BMC Cancer* have associated a reduction in the expression of inhibitors of the enzyme with malignant cellular behavior.

Lotte Vogel, from the University of Copenhagen, worked with a team of Danish and Norwegian researchers to investigate levels of prostasin and its [inhibitors](#) in colorectal tissue samples from 222 patients and 23 controls. They found that the mRNA levels of the inhibitor of prostasin, PN-1, increased at both the transition between normal tissue and mild/moderate [dysplasia](#) and again at the transition between severe dysplasia and colorectal cancer.

According to Vogel, "It has previously been shown that overexpression of prostasin in mammary and prostate cancer cells reduces the invasive properties of cancer cells and that high prostasin expression in gastric tumours is associated with longer survival. In what may be support for this trend, our data shows that elevated mRNA levels for prostasin's inhibitor, PN-1, coincides with the acquisition of malignant properties in colorectal tissue".

The [enzymatic activity](#) of prostasin is almost certainly influenced by levels of inhibitors other than PN1, and PN1 itself is known to inhibit many other enzymes. This complex web of interactions between relevant proteases and their inhibitors makes firm conclusions difficult to draw. As Vogel writes, "Future studies are required to clarify whether down-

regulation of prostatic activity via up regulation of PN-1 is causing the malignant progression or if it is a consequence of it".

More information: Expression of prostatic and its inhibitors during colorectal cancer carcinogenesis, Joanna Selzer-Plon, Jette Bornholdt, Stine Friis, Hanne C Bisgaard, Inger M. B. Lothe, Kjell M. Tveit, Elin H Kure, Ulla Vogel and Lotte K. Vogel, *BMC Cancer* (in press)

Source: BioMed Central ([news](#) : [web](#))

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