Stem cell function may explain higher colon cancer rate in males

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A higher incidence of colorectal cancer has been seen in males. In this issue of *Stem Cell Reports*, Yu et al. discovered the role of androgen in proliferation and differentiation of intestinal stem cells, a mechanism that may explain the higher levels of colorectal cancer in males. The dichotomy of androgen action in the gut and its ink in colorectal cancer is illustrated by the yin-yang symbol divided the cross section of intestine into male part (black zone with structure formula of testosterone) and female part (white zone with formula of estrogen). Credit: Yu et al

Colon cancers have a higher incidence in males, which is in part related to much higher androgen levels in males compared to females. Scientists have now discovered a link between androgens and stem cells that could contribute to colon cancer. Some cancers in the intestine are thought to arise from intestinal epithelial stem cells, which are needed for normal

tissue maintenance and regeneration, but which can give rise to cancers when they grow improperly.

In research recently published in *Stem Cell Reports,* Jingxin Li, Dawei Chen and colleagues found that androgen levels can regulate intestinal stem cell proliferation, a new potential link between androgen levels and colon cancer.

By tuning androgen levels in mice with genetic or pharmacological tools, the researchers found that high androgen levels cause intestinal stem cells to divide more than usual and at the same time generated less mature epithelial cells, characteristic of changes that can lead to tumor formation.

Interestingly, findings show that this effect was not direct but involved the surrounding niche cells, a population of cells that regulate intestinal stem cell growth and function. While important follow up research is needed, these findings may have implications for treating or preventing colon cancers through androgen regulation.

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