New link seen between gene fusion and bladder cancer

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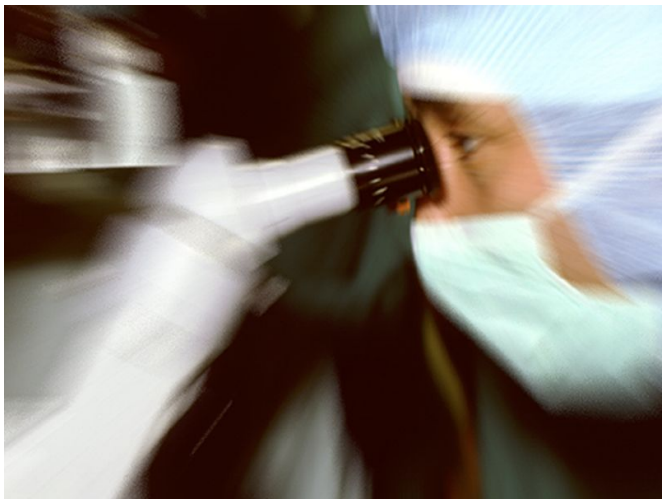

(HealthDay)—The fibroblast growth factor receptor 3 (FGFR3) transforming acidic coiled-coil containing protein 3 (TACC3) (FT3) gene fusion recruits endogenous TACC3 away from the mitotic spindle, resulting in errors in chromosome segregation in bladder cancer cells, according to a study published online Aug. 30 in *Open Biology*.

Noting that FT3 has been identified in many cancers, including those of the urinary bladder, Sourav Sarkar, Ph.D., from the University of Warwick in the United Kingdom, and colleagues explored potential changes in TACC3 function in FT3-positive cells.

The researchers note that TACC3 is a mitotic spindle protein required for accurate segregation of chromosomes, and errors in segregation can lead to aneuploidy, contributing to cancer progression. FT3-positive bladder cancer cells were found to have lower levels of endogenous TACC3 on the mitotic spindle, causing mitotic defects. FT3 was not localized to the mitotic spindle, and recruited endogenous TACC3 away from the spindle by virtue of its TACC domain. In FT3-positive bladder cancer cells, the chromosome segregation defects were partially rescued by knockdown of the fusion gene or low-level overexpression of TACC3. In these cancer cells, inhibition of FGFR3 signaling did not rescue the TACC3 level on spindle.

"In this paper, we have shown a TACC3-specific role of FT3 in inducing mitotic defects in bladder cancer cells," the authors write. "The mechanism we describe is general and is likely to translate to other FT3-positive cancer types."

More information: Abstract/Full Text