Early drivers of gastric cancer

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Gastric cancer – a leading cause of cancer-related deaths worldwide – is commonly diagnosed at a late stage when most patients have a poor prognosis.

Wael El-Rifai, M.D., Ph.D., and colleagues are exploring the molecular alterations that drive gastric carcinogenesis, to improve early detection, treatment and prevention. They previously showed that knocking out (eliminating) the gene Tff1 in mice induces gastric lesions that include low- and high-grade dysplasia and adenocarcinomas.

Now, they have investigated gene expression in low-grade dysplastic lesions and normal stomach tissue from mice and in gastric cancer and normal stomach tissue from humans. Using bioinformatics approaches, they identified transcription (gene expression) networks that were consistently deregulated in both mouse and human lesions.

The findings, reported in the July issue of *Genes, Chromosomes and Cancer*, suggest that activation of MYC, STAT3, and beta-catenin transcription networks could be an early molecular step in gastric tumorigenesis. The findings also support using the Tff1-knockout mouse model for in vivo studies of molecular mechanisms in gastric cancer.

More information: Zheng Chen et al. Integrated expression analysis identifies transcription networks in mouse and human gastric neoplasia, *Genes, Chromosomes and Cancer* (2017). DOI: 10.1002/gcc.22456

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