There is convincing evidence that Runx 3 is a new and important tumour suppressor in gastric cancer.

Runx3/Pebp2alphaC null mouse gastric mucosa exhibits hyperplasias due to stimulated proliferation and suppressed apoptosis in epithelial cells, and the cells are resistant to growth-inhibitory and apoptosis-inducing action of TGF-beta, indicating that Runx3 is a major growth regulator of gastric epithelial cells. Between 45% and 60% of human gastric cancers significantly express RUNX3 due to hemizygous deletion and hypermethylation of the RUNX3 promoter region. Tumor-specificity of human gastric cancer cell lines in nude mice was inversely related to their level of RUNX3 expression, and a mutation (R122C) occurring within the conserved Runt domain abolished the tumor-suppressive effect of RUNX3, suggesting that a lack of RUNX3 function is causally related to the genesis and progression of human gastric cancer.
excellent example where failure to appropriately activate physiological apoptosis contributes to tissue expansion and neoplasia. Thirdly, this paper illustrates the importance of epigenetic changes in the aetiology of disease. Promoter methylation is now commonly recognised as a major determinant of gene expression. The fact that Runx 3 expression could be restored by promoter demethylation in cell culture suggests that strategies to reactivate silenced genes may one day be of therapeutic value clinically.

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