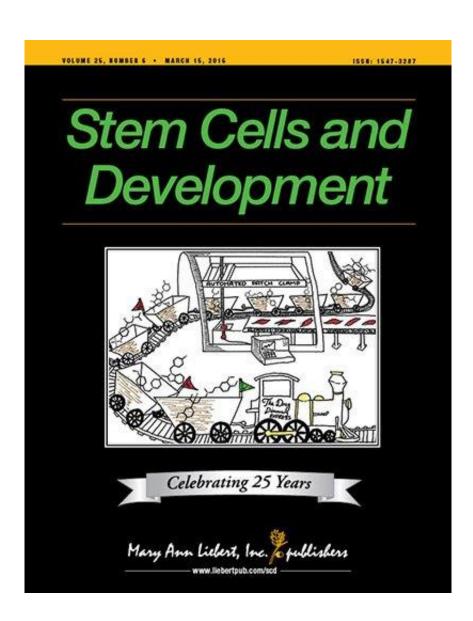


What can Frizzled7 reveal about breast cancer development?

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Credit: Mary Ann Liebert, Inc., publishers



A new study shows that Frizzled7 (FZD7), a protein present on human breast epithelial cells and a component of the Wnt signaling pathway is uniquely controlled by the Notch signaling pathway, both of which play key roles in mammary gland formation and breast cancer development. Evidence supporting the potential ability to regulate Notch-mediated Wnt signaling by targeting the NOTCH3-FZD7signaling network is presented in the study published in *Stem Cells and Development*.

Vasudeva Bhat, Yu Jia Sun, Steve Weger, and Afshin Raouf, University Manitoba and CancerCare Manitoba, Winnipeg, Canada, determined how the Notch signaling pathway uniquely regulates a specific component of the Wnt signaling pathway—the FZD7receptor—in human breast epithelial cells. A better understanding of how the two pathways interact could help identify an underlying cause of breast carcinogenesis.

In the article "Notch-induced Expression of FZD7 Required Noncanonical NOTCH3 Signaling in Human Breast E0pithelial Cells", the researchers provide evidence that Notch-induced expression of FZD7 is controlled only by the NOTCH3 receptor and in a unique way that differs from other demonstrated mechanisms of Notch-mediated modulation of the Wnt signaling pathway.

"This paper explodes the dogma that there is redundancy across all Notch receptors in binding, nucleation, and subsequent activation of target genes," says Editor-in-Chief Graham C. Parker, PhD, The Carman and Ann Adams Department of Pediatrics, Wayne State University School of Medicine, Detroit, MI. "These results are acutely of interest to breast cancer researchers but also have wider implications for our understanding of normal development."

More information: Vasudeva Bhat et al. Notch-Induced Expression of FZD7 Requires Noncanonical NOTCH3 Signaling in Human Breast



Epithelial Cells, *Stem Cells and Development* (2016). DOI: 10.1089/scd.2015.0315

Provided by Mary Ann Liebert, Inc

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