A Novel Diagnostic And Therapeutic Target Within The Wnt Pathway

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Technology description

UCSD researchers have identified and characterized a novel non-receptor for trimeric G proteins that works synergistically with the Wnt pathway receptors to enhance PI3K and beta Catenin signals to trigger oncogenesis. Since multiple cancers depend on aberrant enhancement of Wnt signaling to progress, this invention may be extremely significant for cancer research diagnosis and therapy.
Wnt signaling is essential for tissue homeostasis and its dysregulation causes cancer, Wnt ligands trigger signaling by activating Frizzled receptors, which belong to the G-protein coupled receptor superfamily. However, the mechanisms of G-protein activation in Wnt signaling remain controversial. The invention provides that frizzled receptors activate G proteins and trigger non-canonical Wnt signaling via the target, which contains binding and activating motifs which associate with binding to frazzled receptors, thereby linking Wnt stimulation to G protein activation. This then triggers non-canonical Wnt responses suppressing tumorigenesis but enhancing tumor cell invasiveness.
It has been shown that in colorectal cancer, the target is suppressed during adenoma to carcinoma transformation, and expressed later in metastasized tumor cells. Therefore, dysregulation of the target can impact both tumor initiation and progression to metastasis. The target has been cloned and therefore will be a useful tool for testing and or generation of novel agents. A patent application has been filed.

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