Mutations in genes coding for components of the wnt signalling pathway are relatively common in certain human cancers (Giles et al., 2003; Polakis, 2000). Mutations in the tumor suppressor APC occur frequently in colorectal cancer whereas mutations in Axin and β-catenin are detected in hepatocellular cancers. However, some human cancers exhibit evidence of deregulated signalling, yet mutations in the known components of the wnt pathway are rarely detected. This is the case for breast cancer, where histological evidence indicates that β-catenin is activated but mutations in APC, Axin or β-catenin are not prevalent (Howe and Brown, 2004). Therefore, we have searched for epigenetic events that might contribute to wnt signalling in breast tumour progression. To this end we have examined the expression patterns of wnts, soluble wnt inhibitors, the wnt receptors and the transcription factors of the TCF/LEF family in breast cancers. As an in vitro model we have enlisted the quasi-normal MCF10a human breast cell line. The data in aggregate indicate that wnt signalling is active in human breast epithelium and that alterations in the expression levels and activities of wnt pathway components are common in human breast cancer.

