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Dysregulation of Wnt/β-catenin pathway has been reported to play a pivotal role in the development and progression of colorectal cancer. β-catenin is a downstream component of the Wnt signaling pathway. In the absence of Wnt stimulation, the levels of cytoplasmic β-catenin are low since the phosphorylated β-catenin is ubiquitinated and constantly degraded in the proteasome. In recurrent colon cancer, the coordinated phosphorylation and destruction of β-catenin is disrupted resulting in its accumulation and translocation to the nucleus, where it activates the transcription of its target genes, cyclin D1 and c-myc. The figure depicts indirect immunofluorescence staining of parental HT-29 cells showing membranous and cytoplasmic localization of β-catenin (green). Counter staining was performed with DAPI (blue) ×100. See article by Vasudevan and colleagues (beginning on page 1138) for more information.