Invasion by colorectal carcinomas is characterized by an epithelial-mesenchymal transition (EMT)-like dedifferentiation of the tumor cells. However, a redifferentiation towards an epithelial phenotype, resembling a mesenchymal-epithelial transition, is detectable in metastases. This indicates that malignant progression is based on dynamic processes, which cannot be explained solely by irreversible genetic alterations, but must be additionally regulated by the tumor environment. The main oncoprotein in colorectal cancer is the Wnt pathway effector beta-catenin, which is overexpressed due to mutations in the APC tumor suppressor in most cases. EMT of the tumor cells is associated with a nuclear accumulation of the transcriptional activator beta-catenin, which is reversed in metastases. Nuclear beta-catenin is involved in two fundamental processes in embryonic development: EMT and stem cell formation. Accumulating data demonstrate that aberrant nuclear expression of beta-catenin can also confer these two abilities to tumor cells, thereby driving malignant tumor progression.