Background: The Adenomatous Polyposis Coll (APC) tumor suppressor gene is mutated in most familial and sporadic colorectal cancers, and encodes a large cytoplasmic protein, which is implicated in cell migration, cell adhesion, and proliferation (1). APC binds directly to microtubules, and lack of APC leads to defective mitotic spindles and aneuploidy due to mis-segregation of chromosomes (2). APC is well characterized as a scaffolding protein, binds to beta-catenin and is involved in the regulation of its intracellular concentration: in the absence of a Wnt signal, GSK3beta phosphorylates all three members of the APC-beta-catenin-axin complex and this phosphorylation of beta-catenin creates a recognition site for ubiquitin, the signal for proteosome mediated degradation. In the presence of a Wnt signal, dishevelled inactivates GSK3beta and beta-catenin can now coordinate gene transcription of proteins important for the control of cell cycle progression and proliferations such as cyclin D1 and c-myc (3).

Specificity/Sensitivity: APC Antibody detects endogenous levels of total APC protein.

Source/Purification: Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to the sequence of human APC. Antibodies are purified by protein A and peptide affinity chromatography.

Background References:

Western blot analysis (lane 1) and Immunoprecipitation (lanes 2-3) of extracts from SW480 cells. APC was immunoprecipitated with APC Antibody (lane 2) or with rabbit IgG (lane 3) to confirm specificity. Western blot was detection was performed using APC Antibody.