The APC gene regulates transcription, cell adhesion, microtubular cytoskeleton, cell migration, crypt fission (formation of the folds in the intestine), apoptosis, and the spreading of cells. The APC protein interacts with several proteins, one of which is β-catenin. With the mutated APC gene, the APC protein is usually shorter, causing higher levels of unbounded β-catenin molecules through the Wnt-1 signaling pathway. This shorter APC protein causes the destruction complex for the catenin molecule to not form during the Wnt-1 signaling pathway. These free molecules migrate to the nucleus where they bind to T cell factor 4. This activates gene expression at the wrong time and place. In " T-cell Factor 4 Functions as a Tumor Suppressor whose Disruption Modulates Colon Cell proliferation and Tumorigenesis," it states, " Data from Tcf4 mutant mice show a loss of proliferative cells, suggesting that Tcf4 is important for stem cell renewal in the small intestine and the general assumption that the formation of the Tcf4/β-catenin complex is cancer-promoting," (Angus-Hill, Elbert, Hidalgo, and Capechchi 4914). Since the Tcf4/ β-catenin complex is involved in cell proliferation and differentiation, it means that cells containing this complex will have abnormal cell growth in intestinal crypts. Normal people would not have this complex form at the magnitude found with people with the APC gene mutation. Normal people would just have normal intestinal crypt formation that result in no polyps.

The APC gene's second major role is its effect on cell cytoskeletons. Cells of the intestine are linked by calcium bonds that are connected to the cells actin filaments via β-catenin molecules and E-cadherin molecules. The increased β-catenin concentration messes with the cell's equilibrium between β-catenin and APC protein. The APC protein may also be over expressed, and since the APC protein competes with other proteins to bond with β-catenin, sites on the β-catenin may also be blocked.

There are also lesser explored roles of APC. " Intestinal trefoil factor 3 appears to interact with both APC and E-cadherin in complexes that modulate epithelial cell adhesion, migration and survival," according to Oxford journals. APC has also been observed to directly interact with DNA.