The taxanes are a class of commonly used anticancer agents, which are very effective in treatment of breast, ovarian, prostate or lung cancer. Taxanes bind to the β-tubulin subunit of microtubules and lead to their stabilization and inhibition of depolymerization. Such microtubules lose their function to form mitotic spindle, thus arresting cells in G2/M phase and resulting in apoptosis. Unfortunately some cells are able to escape from taxanes-induced apoptosis by developing various mechanisms of resistance including alteration in taxanes target microtubules or upregulation of specific transporters that pump the drug out of cells. Other types of resistance are connected with process of programmed cell death (PCD), especially with proteins that after taxane application participate in its successful progress. Taxanes can directly or indirectly modify the activity of Bcl-2-family proteins that control mitochondrial and endoplasmic reticulum integrity, thus regulating the initiation of PCD. Caspases are executioners of PCD and caspase-2 activated by cytoskeletal disruption seems to be especially important in taxanes-induced apoptosis. In some cases can taxane treatment also result in caspase-independent cell death. Special role has protein p53 that seems to be involved only in apoptosis caused by low taxanes concentration.