Cell division is necessary for maintaining tissue homoeostasis, but at the same time its defects are closely related to the development of many diseases including cancer and premature ageing. Activation of oncogenes leads to replication stress and directly threatens genome stability. The right control of transition between interphase and mitosis is an important mechanism for the protection of genome integrity. Nuclear division is only possible with those cells in which flawless duplication of genetic information occurred. By contrast, cells with damaged DNA structure remain temporarily or permanently stopped at G2 phase of the cell cycle. The topic of this thesis is a detailed literature overview with the subject of molecular mechanisms of the G2/M transition regulation under unperturbed conditions and in the presence of damaged DNA.