Disruption of a proper regulation of cell proliferation can ultimately cause cancer. Most human B cell malignancies are driven by chromosomal translocations or other genetic alterations which directly affect the function of critical cell cycle proteins, such as cyclins and cyclin-dependent kinases. In addition, the transformation of indolent lymphomas into aggressive malignancies is often accompanied by a loss of tumor suppressors controlling important cell cycle checkpoints. A better understanding of cell cycle deregulations in human tumors has promoted the introduction of a new class of antiproliferative drugs into cancer therapies. These drugs exert their function by specifically blocking important cell cycle proteins. In the present review we discuss how alterations in the cell cycle control contribute to the malignant transformation of B cells. Furthermore, we provide an overview of novel direct and indirect cell cycle inhibitors and their impact on the treatment of patients with B cell lymphomas.