Cyclin-dependent kinase

In the beginning, cyclin-dependent kinase 1 CDK1 for example, is a cell division cycle protein homolog 2, regarding a research paper that explains the role of CDK1 in the human breast cancer cells. They found that CDK1 rather than any types of CDK is fatal to the mutated version of MYC-dependent cancer that leads to a depletion of the oncogene in human cancer cells.

The study reveals that the reason for the MYC breast cancer cells duplation is targeting CDF1 exhibit any other CDK cell lines. The primary purpose was to figure out the mechanism of CDK1 inhibition which controls and target the breast cancer cells in human and both phosphorylation and expression of MYC during the cell cycle process.

To confirm that, they used siRNA knockdown to measure the expression level of MYC. Further, other than any mutants, apoptosis leading to the uncontrolled cell proliferation especially in human breast cancer disease that was measured by the detection of caspase-cleaved cytokeratin 18 using flow cytometry technique.

The controls that were used are estrogen receptor ER-positive and ER-negative, they found increased at the number of the protein activity for the positive and the negative correlation, but the gene expression was high at the positive one and decreased to zero at the negative control (that means no reaction between them).

They confirmed that CDK1 is essential for cell division and necessary for driving the cell cycle in all cell types. To initiate intracellular signaling pathways and stimulate the cell cycle entry, mitogens substance bind to the

cell surface receptor with the activation form of GTPase Ras that activates MAP kinase cascade. That will lead to the expression of encoding gene of the transcription regulatory protein like MYC.

Moreover, MYC will increase the expression of many late response genes, including some genes that lead to increased G1-CDK activity (cyclin D Cdk4), which triggers the phosphorylation of members of the tumor suppressor protein (Retinoblastoma protein)family. The inactive form of RB proteins can release the E2F target genes to initiate the transcription process in G1 and S phases, to enhance the G1- CDK and S- CDK activities and the phosphorylation of RB proteins forming a positive feedback loop.