Dev. Reprod. Vol 10, No 1, 2006(Suppl). pp 3

Molecular Aspects of the Mammalian Cell Cycle Regulation and Cancer

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Uncontrolled cell proliferation is one of the main hallmarks of cancer, and tumor cells have acquired damage to genes that are directly involved in regulating the cell cycle. Damage is caused by mutations producing an oncogene with a dominant gain of function, and/or by mutations in tumor suppressor genes causing a recessive loss of function. Regardless of the genetic damage or type of cancer, the common feature is a disrupted cell cycle. The cell cycling process is carefully regulated and responds to the specific needs of a certain tissue or cell type. Normally, in adult tissue, there is a delicate balance between cell death (programmed cell death or apoptosis) and proliferation (cell division) producing a steady state. Disruption of this equilibrium by loss of cell cycle control may eventually lead to tumor development. The highly organized and regulated cell cycle process is responsible for duplication of the cell. Tight regulation and timing ensure that DNA is replicated once during the S phase (without errors), and that identical chromosomes are equally delivered to daughter cells during the M phase. The cell cycle is, therefore, an alteration of two main processes: A) the "doubling" process (S = synthesis phase) where DNA is synthesized, and B) the "halving" process (M = mitosis phase) where the cell and its contents are divided equally into two daughter cells. Cell cycle progression is driven by the coordinated regulation of the activities of cyclin-dependent kinases (Cdks). Of the several mechanisms known to regulate Cdk activity in response to external signals, regulation of cyclin gene expression, post-translational modification of Cdks by phosphorylation-dephosphorylation cascades, and the interaction of cyclin/Cdk complexes with protein inhibitors have been thoroughly studied. The purpose of the present review is to summarize the most important aspects of the various mechanisms implicated in cell cycle regulation.