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Intracellular checkpoint controls constitute a network of signal transd- tion pathways that protect cells from external stresses and internal errors. Ext- nal stresses can be generated by the continuous assault of DNA-damaging agents, such as environmental mutagens, ultraviolet (UV) light, ionizing radiation, or the reactive oxygen species that can arise during normal cellular metabolism. In response to any of these assaults on the integrity of the genome, the activation of the network of checkpoint control pathways can lead to diverse cellular responses, such as cell cycle arrest, DNA repair, or elimination of the cell by cell death (apoptosis) if the damage cannot be repaired. Moreover, internal errors can occur during the highly orchestrated replication of the cellular genome and its distribution into daughter cells. Here, the temporal order of these cell cycle events must be strictly enforced?for example, to ensure that DNA replication is c- plete and occurs only once before cell division, or to monitor mitotic spindle assembly, and to prevent exit from mitosis until chromosome segregation has been completed. Thus, well functioning checkpoint mechanisms are central to the maintenance of genomic integrity and the basic viability of cells and, the- fore, are essential for proper development and survival. The importance of proper functioning of checkpoints becomes plainly obvious under conditions in which this control network malfunctions and fails. Depending on the severity and timing, failure of this machinery can lead to embryonic lethality, genetic diseases, and cancer.

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