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MOLECULAR MECHANISMS OF CANCER DEVELOPMENT

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The complex interplay among the cell cycle, proto-oncogenes, tumour suppressor genes, and apoptotic pathways is crucial for understanding cancer biology, as these components collectively regulate cellular growth, division, and survival. Disruptions in the cell cycle and the conversion of proto-oncogenes to oncogenes can lead to unchecked proliferation, while the loss of tumour suppressor gene function compromises genomic stability. Furthermore, the balance between pro-apoptotic and anti-apoptotic signals is essential for determining cell fate.

In this investigation the interactions between the cell cycle, proto-oncogenes, tumour suppressor genes, and apoptotic pathways in cancer development were established and the role of external factors in this pathway disturbing was identified.

The information from PUBMED, WHO, Google scholar, ScienceDirect, SpringerLink, Wiley Online Library and ResearchGate was collected and analysed.

Analysis of the literature demonstrated that disruptions in the cell cycle, driven by mutations in proto-oncogenes and tumour suppressor genes, lead to uncontrolled cell proliferation, a key characteristic of cancer. It is observed that overexpression of proto-oncogenes promotes tumour formation, while inactivation of tumour suppressor genes contributes to cancer progression. Additionally, the imbalance between pro-apoptotic and anti-apoptotic signals allows cancer cells to evade programmed cell death, enhancing their survival and resistance to therapies. The findings underscore the critical roles of the cell cycle, proto-oncogenes, tumour suppressor genes, and apoptotic pathways in cancer biology. By elucidating how these components interact within signalling networks, we highlight potential targets for therapeutic interventions. Restoring normal function in these pathways could improve treatment efficacy and overcome resistance mechanisms in cancer therapy.

Cancer development stems from disruptions in the cell cycle, proto-oncogenes, tumour suppressor genes, and apoptosis. Knowing these mechanisms is the therapeutic targets for restoring normal cell function and improving treatment.