Apoptosis in Cancer Cells

Name

Institution

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**Introduction**

Apoptosis refers to an orchestrated and ordered cellular process that takes place in pathological and physiological conditions. The topic emerges as one of the most studied in the cell biology continuum. It is important to understand the mechanism of apoptosis as it plays a crucial role in the learning of origin and development of several diseases. In cancer, resistance to apoptosis refers to the condition where malignant cells fail to die. Over recent years, biologists have undertaken intense research aimed at gained deeper insight into what causes the resistance to apoptosis, as one of the ways of combating cancer.

There has been a growing body of evidence suggesting that the treatment of cancer by focusing on apoptosis is viable. Nonetheless, a flurry of questions surfaces regarding the use of new treatment methods and drugs tailored to promote apoptosis. Critical tests have to be conducted and passed prior to safe use on human subjects. In cancer, a loss of balance occurs between cell death and cell division; cells supposed to die if fail to receive the triggering signals. For instance, during the down-regulation of one of the tumor suppressor gene known as p53, causes enhanced tumor development and growth, and diminished apoptosis. On the other hand, the inactivation of the gene is associated with a variety of human cancers irrespective of the mechanism. As much as it can be the cause of the problem, apoptosis is in many respects the solution as well. Not many people wonder that there has been an avalanche of interest and research into the development of new drugs targeting different facets of apoptosis. The role of apoptosis in both cancer treatment and carcinogenesis can not be underestimated. This paper gives a detailed description of the current research on apoptosis in cancer and gives recommendations for future research.

**Summary of Current Research**

Over recent years, primary cancer research has led to significant progress in the comprehension of cancer genetics and cancer biology. One of the most lauded of these progressions is that apoptosis along with the controlling genes bear a great degree of influence on the malignant phenotype. It is now apparent that tumor metastasis, progression or initiation is as a result of the disruption of apoptosis by a number of oncogenic mutations. In contrast, evidence suggests that apoptosis is enhanced by other oncogenic transformations which lead to selective pressure to supersede apoptosis, especially in multistage carcinogenesis. Additionally, it has been found that a majority of cytotoxic anticancer agents trigger apoptosis (Raj et al., 2011). The implication of this finding is that there is a higher probability that apoptotic programs’ flaws bring about treatment failure. There is currently an intense research effort aimed at unraveling the various mechanisms of apoptosis in order to provide information and create grounds upon which apoptosis may be exploited for therapeutic advantage.

A majority of the anticancer agents in use today were created through the use of empirical screens tailored to detect agents that selectively eliminated tumor cells. Hitherto, a great deal of research into the action of drugs concentrated on resistance mechanisms, the type of cellular damage or the intracellular drug targets. Nevertheless, pathologists discovered that chemotherapy and radiation could activate the death of cells with morphological characteristics of apoptosis. Current research has indeed ascertained that anticancer agents elicit apoptosis, and treatment sensitivity can be reduced via the interruption of apoptotic mechanisms. Given that agents with different basic targets have the ability to initiate apoptosis via parallel programs, mutations in apoptotic mechanisms yield multi-drug resistance.

Furthermore, current research has been able to identify the factors that spark apoptosis during tumor development. They include extracellular triggers such as loss of cell-matrix interactions, radiation, and hypoxia and survival/growth factor depletion, and internal balances, for example telomere malfunction and DNA damage (Martin et al., 2013). The discovery of apoptotic activators has helped bring into light tumor evolution forces. For instance, although prolonged skin exposure to UV radiation triggers apoptosis, it also helps destroy totaled cells. Ultraviolet radiation activates apoptosis. Likewise, the erosion of p53 function results in the survival of such damaged cells, hence the start of tumor development.

In concurrence with current research, apoptosis disruption also plays a role in tumor metastasis. For a tumor cell to metastasize, it has to survive within the bloodstream and attack a foreign tissue. However, this process often fails to occur since the epithelial cells are predisposed to die in suspension or where there is no apt tissue survival (Thedieck et al., 2013). It is therefore quite clear that these processes initiate apoptosis and lead to the selective pressure to modify apoptotic mechanisms during tumor growth. Signal transduction and focal adhesion pathways are some of the molecules that serve to control apoptosis in suspension. In retrospect, loss of apoptosis potentially affects the metastasis, progression, and initiation of tumors.

One intriguing feature of the current research is the biological link between cancer therapy and cancer genetics; the same genetic changes that affect apoptosis during tumor development influence treatment sensitivity as well. Now that the manipulation of apoptotic programs to produce extensive alterations in cell deaths is practicable, the proteins and genes regulating apoptosis are possible drug targets. There are several empirically obtained cytotoxic drugs that currently target apoptosis either non-exclusively or indirectly. More so, the drugs are toxic and mutagenic to normal tissues. Conversely, agents that trigger apoptosis directly tend to offer less chance for acquired drug resistance, reduce toxicity, and decrease mutagenesis. As far as current research is concerned, two observations indicate the viability of such methods. The first one is that a majority of anti-apoptotic alterations act considerably upstream in the program; this means that tumor cells still keep the underlying potential and ‘machinery’ for apoptosis. The second one is that tumor-specific changes in apoptotic programs offer selective cell death targeting (Raj et al., 2011).

Additionally, there are a number of current strategies being analyzed as discussed. In situations where dominant oncogenes disable apoptosis, the agents that interfere with their anti-apoptotic function have been shown to cause significant rise is cell death. The over-expression of anti-apoptotic agents can enhance chemoresistance and tumor development; it implies that the functional inhibition of such proteins may eliminate cancer cells. For instances, characterized by the loss of apoptosis due to a recessive mutation, substantial cell death may be promoted through the restoration of a dysfunctional activity or gene. Reintroducing mutant tumor cells may activate apoptosis or promote treatment sensitivity in tumor xenografts or cell lines (Martin et al., 2013). Techniques employing this approach are tested in clinical tests and trials. The justification and rationale of this approach are connected to the fact that normal cells are intrinsically less sensitive to inhibition compared to tumor cells. Current research has revealed that counter recessive transformations which are anti-apoptotic do not necessarily have to depend on protein or gene therapy. As a matter of fact, the inactivation of p, a gene that is pro-apoptotic, may foster cell survival by alleviating the effects of a downstream death suppressor inhibition (Thedieck et al., 2013). In this case, the downstream effector serves as a more appropriate drug target. Despite the fact that death receptors rarely mutate in the scope of human tumors, modifications accompanying tumor development can change the control of these pathways. Current research has led to the discovery of a viral protein referred to as a point which has the capacity to eliminate tumor cells.

**Conclusion**

Recent years have witnessed a remarkable increase in the comprehension of apoptosis along with its impact on cancer and cancer therapy. In addition, there has been an increasing focus on the molecular mechanisms in relation to apoptotic cell death. Current research has brought forth a platform for further advancements in cancer therapy, prognosis, and diagnostics. The recent breakthroughs described above such as anti-apoptotic activity targeting, restoration of pro-apoptotic activities, and the use of death ligands are very critical in cancer therapy. They help present information necessary for the design of drugs aimed at eliminating cancer cells. The future outlook in the field promises that coherent methods to structure cell suicide programs will lead to a realization of new, less mutagenic and less toxic therapies compared to the existing ones (Indran et al., 2011).

I have learned that the pathogenesis of cancer is a complex issue that requires careful and intense study if there is any chance to combat the disease. I have also learned that apoptosis plays a vital role not only in cancer but also in other diseases as well. Current research on apoptosis in cancer cells has also enlightened me as to the strategies used in the treatment of cancer. Last but not least, I have come to appreciate the efforts of cell biologists in this specific field given that it calls for special attention and focus.

There is no direct cure for cancer. In my opinion, the study of apoptosis in cancer cells is a major step towards understanding, treatment, and possible cure for cancer. Over the past decade, pathologists and other health care professionals have been at the forefront of combating cancer. Although their efforts have realized remarkable strides, a lot remains to be done. More research should be directed in the study of apoptosis in cancer cells, since therein lies the cure for the malignant disease.

References

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