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Alterations of Epigenetic Regulators in Pancreatic Cancer

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Abstract

Pancreatic cancer is one of the deadliest cancers, with a five-year survival rate of less than 10%. Despite advances in the treatment of other cancers, pancreatic cancer remains a difficult disease to treat due to its aggressive nature and late diagnosis. In recent years, there has been increasing interest in the role of epigenetics in the development and progression of pancreatic cancer. Epigenetics refers to modifications of DNA and histones that regulate gene expression without altering the underlying genetic code. Alterations in epigenetic regulators can lead to aberrant gene expression, resulting in the development and progression of cancer. In pancreatic cancer, several epigenetic regulators have been found to be deregulated, including DNA methylation, histone modifications and non-coding RNAs.

Keywords: Pancreatic cancer • Gene expression • Genetic code

Introduction

DNA methylation is a common epigenetic modification that involves the addition of a methyl group to cytosine residues in CpG dinucleotide. DNA methylation can lead to the silencing of tumour suppressor genes, allowing cancer cells to proliferate uncontrollably. In pancreatic cancer, several key tumour suppressor genes, such as CDKN2A, have been found to be hyper methylated, leading to their down regulation and promoting tumour growth. Histone modifications, such as acetylation, methylation and phosphorylation can also regulate gene expression. In pancreatic cancer, there is evidence of aberrant histone modifications, including increased histone deacetylase activity and decreased Histone Acetyl Transferase (HAT) activity. HDAC inhibitors have shown promise in preclinical studies as a potential therapeutic strategy for pancreatic cancer.

Literature Review

Non-coding RNAs, such as microRNAs and long non-coding RNAs, can also regulate gene expression at the post-transcriptional level. In pancreatic cancer, deregulation of microRNAs has been shown to promote tumour growth and metastasis. For example, miR-21 is up regulated in pancreatic cancer and has been associated with poor prognosis. The deregulation of epigenetic regulators in pancreatic cancer has significant clinical implications. Epigenetic alterations can serve as diagnostic and prognostic biomarkers and potential therapeutic targets. DNA methylation patterns, for example, can distinguish pancreatic cancer from normal tissue and can be used as a diagnostic tool. Histone modifications and non-coding RNAs also have potential as biomarkers for pancreatic cancer. Pancreatic cancer is a devastating disease that affects the pancreas a glandular organ located in the abdomen. The pancreas plays an important role in digestion and the regulation of blood sugar levels. Pancreatic cancer occurs when abnormal cells in the pancreas grow and multiply uncontrollably, forming a mass or tumour [1].

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Discussion

Pancreatic cancer is a particularly deadly form of cancer, with a five-year survival rate of less than 10%. This is largely due to the fact that pancreatic cancer is often diagnosed at an advanced stage, when the cancer has already spread to other parts of the body. Symptoms of pancreatic cancer can include abdominal pain, weight loss, jaundice (yellowing of the skin and eyes) and digestive problems. However, these symptoms can be vague and nonspecific and may not appear until the cancer has already advanced. There are two main types of pancreatic cancer: exocrine pancreatic cancer and endocrine pancreatic cancer. Exocrine pancreatic cancer is the most common type, accounting for more than 90% of all cases. This type of cancer originates in the ducts that carry digestive enzymes from the pancreas to the small intestine. Endocrine pancreatic cancer, also known as Pancreatic Neuroendocrine Tumours (PNETs), originates in the hormone-producing cells of the pancreas [2,3].

The exact causes of pancreatic cancer are not fully understood, but certain risk factors have been identified. These include smoking, obesity, a family history of pancreatic cancer and certain genetic mutations. Chronic pancreatitis, a long-term inflammation of the pancreas, is also a risk factor for pancreatic cancer. Treatment for pancreatic cancer depends on several factors, including the stage of the cancer, the location of the tumour and the overall health of the patient. Surgery is often the preferred treatment for pancreatic cancer, but it is only an option if the cancer has not spread to other parts of the body. Chemotherapy and radiation therapy may also be used, either alone or in combination with surgery. Despite advances in treatment, pancreatic cancer remains a difficult disease to treat, largely due to the fact that it is often diagnosed at an advanced stage. In recent years, there has been increasing interest in the role of genetics and epigenetics in the development and progression of pancreatic cancer, which may lead to the development of more effective treatments in the future [4,5].

Targeting epigenetic regulators has emerged as a promising therapeutic strategy for pancreatic cancer. HDAC inhibitors, such as vorinostat and panobinostat, have shown promise in preclinical studies and are currently being tested in clinical trials. Other epigenetic drugs, such as DNA methyltransferase inhibitors and inhibitors of non-coding RNAs, are also under investigation. Pancreatic cancer is a devastating disease that is difficult to diagnose and treat. It is important to be aware of the risk factors for pancreatic cancer and to seek medical attention if any symptoms are present. Early detection and treatment can improve the chances of survival, but more research is needed to fully understand the underlying mechanisms of pancreatic cancer and to develop more effective treatments for this deadly disease [6].

Conclusion

In conclusion, alterations in epigenetic regulators play a significant role in

the development and progression of pancreatic cancer. Deregulation of DNA methylation, histone modifications and non-coding RNAs can lead to aberrant gene expression and promote tumour growth. Epigenetic alterations can serve as diagnostic and prognostic biomarkers and potential therapeutic targets. Further research is needed to fully understand the mechanisms underlying epigenetic deregulation in pancreatic cancer and to develop more effective epigenetic therapies for this deadly disease.

Acknowledgement

None.

Conflict of Interest

No potential conflict of interest was reported by the authors.

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