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Cigarette Smoking and Epigenetics: Unveiling the Hidden Impact

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INTRODUCTION

Cigarette smoking is a major public health concern, responsible for a myriad of diseases, including lung cancer, cardiovascular disorders, and chronic obstructive pulmonary disease. While the direct carcinogenic and toxic effects of tobacco smoke are well-documented, emerging research is shedding light on the intricate relationship between cigarette smoking and epigenetics, a field that explores how environmental factors can alter gene expression without modifying the DNA sequence. This article aims to delve into the complex interplay between cigarette smoking and epigenetics, emphasizing the potential implications for public health and personalized medicine.

DESCRIPTION

Epigenetics encompasses various mechanisms that influence gene regulation, including DNA methylation, histone modifications, and non-coding RNAs. Among these, DNA methylation is perhaps the most studied epigenetic alteration associated with cigarette smoking. DNA methylation involves the addition of a methyl group to the DNA molecule, typically repressing gene transcription. Studies have revealed that cigarette smoke can induce hypermethylation of specific genes, leading to their reduced expression. For instance, tumour suppressor genes involved in lung cancer, such as p16 and RASSF1A, have been found to be hypermethylated in smokers. Furthermore, histone modifications play a pivotal role in gene regulation by modulating the accessibility of the DNA to transcription factors. Studies have shown that cigarette smoke can lead to changes in histone acetylation and methylation patterns, ultimately affecting gene expression. These changes can be linked to inflammatory and oxidative stress responses, which are well-known consequences of smoking. Non-coding RNAs, such as microRNAs, have also been implicated in the epigenetic response to cigarette smoke. MicroRNAs can post-transcriptionally regulate gene expression, and their dysregulation has been linked to smoking-related diseases. For example, miR-21, which is upregulated in response to cigarette smoke, has been associated with lung cancer development. Cigarette smoking induces widespread DNA methylation changes. Several studies have identified specific genes and genomic regions that are differentially methylated in smokers compared to non-smokers. Notably, these changes are not limited to the lungs but extend to other organs, emphasizing the systemic impact of smoking on the epigenome. These methylation alterations are often found in genes related to inflammation, oxidative stress, and cancer. For example, the gene AHRR (Aryl Hydrocarbon Receptor Repressor) has been consistently identified as hypermethylated in smokers. This gene plays a crucial role in detoxifying carcinogens found in cigarette smoke. Its hypermethylation may lead to its reduced expression, impairing the body's ability to eliminate harmful compounds. The effects of cigarette smoke on histone modifications are less well-documented but equally important. Cigarette smoke-induced oxidative stress and inflammation can lead to histone modifications, affecting gene expression. These changes have been linked to the development of smoking-related diseases. Non-coding RNAs, such as microRNAs, are responsive to cigarette smoking and can modulate gene expression in various ways.

CONCLUSION

Cigarette smoking has far-reaching effects on the epigenome, influencing DNA methylation, histone modifications, and non-coding RNA expression. These epigenetic changes play a critical role in the development of smoking-related diseases. Understanding the complex interplay between cigarette smoking and epigenetics offers opportunities for the development of targeted therapies, personalized medicine, and more effective smoking cessation strategies. It is imperative to continue researching this field to unravel the hidden impact of smoking on our epigenome and further enhance public health efforts.

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