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Current Advances in Environmental Epigenetics

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INTRODUCTION

Inflammatory Bowel Disease (IBD) is a complex multifactorial disease in which external and environmental factors significantly influence its onset and pathogenesis, especially in genetically susceptible individuals. Crohn's Disease (CD), one of the two types of his IBD, is characterized by transmural inflammation most commonly localized to the terminal ileum. Oxidative stress caused by excess reactive oxygen species is present both locally and systemically in patients with celiac disease and appears to be associated with the well-described imbalanced immune response and disease comorbidity. Oxidative stress may also underlie some of the proposed environmental risk factors for celiac disease. Although the exact etiology of CD remains unknown, an important role of oxidative stress in CD pathogenesis is widely recognized. Epigenetics can provide a link between environmental factors and genetics, and numerous epigenetic alterations associated with specific environmental risk factors, microbiota, and inflammation have been reported in CD. Further attention should be paid to whether these epigenetic changes associated with oxidative stress play a major role in the pathogenesis of CD.

DESCRIPTION

Epigenomic patterns, particularly DNA methylation, have emerged as potential objective biomarkers to address some of these study design and exposure measurement challenges. In this article, we summarize previous literature on epigenetic alterations associated with specific prenatal and early childhood exposure domains and combinations of exposures in human observational studies and their biomarker potential. In addition, we highlight evidence for other types of epigenetic patterns that serve as exposure biomarkers. Evidence strongly supports epigenomic biomarkers of exposure that are detectable across lifespans and across different exposure domains. Current and future research areas in this area can be used to extend these lines of evidence to other environmental exposures, determine their specificity, and assess early childhood risk factors for lifelong health outcomes. It aims to develop prediction algorithms and methylation scores.

In addition to environmental and genetic alterations, these disorders may be influenced by processes that do not affect DNA sequence but play an important role in gene expression and may be inherited. These so-called 'epigenetic' changes include DNA methylation, histone modifications and ATP-dependent chromatin remodeling enzymes that influence chromatin remodeling and gene expression. In addition, microRNAs are non-coding RNA molecules that silence genes after transcription. Both epigenetic factors and microRNAs are known to influence cardiac development and homeostasis in discrete ways, but also in complex regulatory networks. This review describes how epigenetic factors and microRNAs interact and how they work together to influence cardiovascular disease.

CONCLUSION

In post-genomic science, the development of etiologic models of neurobiological susceptibility to psychiatric risks has expanded exponentially in recent decades. Such studies include those of the McGill Group for Suicide Studies (MGSS). Its research has focused on identifying key risk factors and epigenetic traits that identify specific susceptibility profiles to psychiatric disorders (such as depression and predict high-risk behaviour (e.g., depression) help you to, suicidal tendencies). The MGSS has long focused on the environmental epigenetic model of suicide risk and the extension of findings from rodent studies to the human population, but its overall agenda includes the transition from retrospective studies to clinical studies and epidemiological studies, including multiple research axes. Common to these research axes is concern about the long-term effects of negative experiences on maladaptive courses and their negative consequences for mental health.

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