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Combining Epigenetic and Immunotherapy Improves Outcomes for Cancer Patients

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DESCRIPTION

There is an integration of maintenance signals generated by genetic, epigenetic, immunological, and environmental factors (such as diet) that help maintain balance in the gut-liver axis. It is well known that the imbalance in this complex and interdependent system greatly increases her risk of EH. Therefore, relevant studies were performed to decipher how each signal separates and acts together in the progression of EH. Recent literature has shown that both macronutrients and micronutrients interfere with the expression of regulatory miRNAs and alter several cellular processes that contribute to EH and its comorbidities. We highlight how carbohydrates, lipids, proteins, salt, and potassium alter miRNA signatures during her EH. Disruption of miRNA expression can adversely affect communication systems such as: B. Overactivation of the renin-angiotensin-aldosterone system, modulation of vascular smooth muscle cell phenotype, and promotion of angiogenesis in favor of EH. We also describe the prognostic value of miRNAs in EH and discuss the advantages and disadvantages of surgical and dietary prophylactic approaches in EH prevention. We propose that dietary perturbation of miRNA profiles is a mechanism within the gut-liver axis that determines the development of EH.

It is known that changes in the epigenome affect the development and progression of cancer. Epigenetics is strongly influenced by the environment, such as diet, which is the source of metabolic substrates that influence the synthesis of cofactors or substrates for chromatin and RNA-modifying enzymes. Additionally, plants are a common source of bioactive compounds that can directly modify the activity of these enzymes. Here, we review and discuss the effects of diet on epigenetic mechanisms such as chromatin and RNA regulation and their potential implications for cancer prevention and therapy.

Dietary salt intake increases Blood Pressure (BP), and blood pressure salt sensitivity varies from person to person. Interplay of aging, genetics, and environmental factors, including malnutrition and stress, contribute to BP salt sensitivity. Similarly, offspring of mice fed a low-protein diet during pregnancy develop salt-sensitive hypertension associated with aberrant DNA methylation of the gene encoding the type 1A angiotensin II receptor (AT1AR) in the hypothalamus Causes up regulation of hypothalamic AT1AR, leading to renal damage. Aging is also associated with salt-sensitive hypertension. In aging mice, promoter methylation results in decreased renal production of the anti-aging factor Klotho and reduced circulating soluble Klotho. In Klotho deficiency, salt-induced activation of the Wnt5a-RhoA vascular pathway causes age-related salt-sensitive hypertension, possibly as a result of decreased renal blood flow and increased peripheral resistance. Therefore, renal mechanisms and aberrant DNA methylation of specific genes are involved in the development of salt-sensitive hypertension during fetal development and in old age. Her three different paradigms of epigenetic memory operate on different timescales of prenatal malnutrition, obesity, and aging.

CONCLUSION

Epigenetic alterations in our genome can result in heritable alterations in the risk, clinical presentation, course and outcome of many diseases. Understanding these epigenetic mechanisms will help identify potential therapeutic targets. This is of particular importance in Necrotizing Enterocolitis (NEC), where both ante-mortem and postnatal factors influence susceptibility to this devastating disease, but treatment options are limited. Developmental factors affecting intestinal structure and function, the immune system, gut microbiota, and postnatal enteral feeding are thought to play important roles in this disease. In this manuscript, we have identified the epigenetic mechanisms involved in NEC. These include key developmental alterations in DNA methylation in the immature gut, the role of long noncoding RNAs (lncRNAs) in maintaining intestinal barrier function, Toll-like receptor 4 (TLR4) and enteral nutrition related Epigen-

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etic changes lead to up regulation of pro inflammatory genes. We have extensively reviewed the literature using key terms in multiple databases such as PubMed, EMBASE and Science Direct and assimilated research from our own laboratories.

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CONFLICTS OF INTERESTS

The authors declare that they have no conflict of interest.