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Genome Conservation of Actinomycetes with Small Epigenomes

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INTRODUCTION

In mammals, genomic instability and aging are closely linked. This is evidenced by the growing list of progeria patients and animal models with congenital DNA repair defects. Until recently, DNA damage was thought to accelerate aging by impairing transcription and DNA replication, leading to age-related cellular dysfunction and somatic mutations that lead to cancer. However, recent evidence suggests that DNA lesions also induce widespread epigenetic changes that threaten cellular homeostasis as a function of age. This review describes the functional implications of persistent DNA damage to the epigenome in the context of aging and age-related diseases.

DESCRIPTION

Single-cell sequencing technologies, including transcriptome and epigenome assays, are changing our understanding of the cellular building blocks of neural circuits. By directly measuring multiple molecular signatures of 1000s to 1,000,000s of individual cells, single-cell sequencing methods can comprehensively characterize the diversity of brain cell types. These measurements reveal gene regulatory mechanisms that shape cellular identity and provide insight into the developmental and evolutionary relationships between brain cell populations. Single-cell sequencing data can support the design of tools for targeted functional study of brain circuit components by linking molecular signatures to anatomy, connectivity, morphology and physiology. Here, we describe the basic principles of single-cell transcriptome and epigenome sequencing, the integrative computational analysis of the data, and their important applications in neuroscience.

A ketogenic Diet (KD) is a low-carbohydrate, high-protein, high-fat and normal or low-fat dietary strategy with or without calorie restriction (Very Low Calorie Ketogenic Diet, VLCKD). KDs have been shown to be useful in the treatment of obesity, metabolic and related disorders, neurological diseases, and various pathological conditions such as cancer, non-alcoholic liver disease, and chronic pain. Several studies have examined the intracellular pathways that contribute to these beneficial effects of diet. Epigenetic changes are one of the most important determinants of an organism's ability to adapt to environmental changes, but data on epigenetic changes associated with these foraging pathways are still limited.

Cell fate determination as a fundamental problem in cell biology has been extensively studied at different levels of regulation over the years. However, the mechanisms of multilevel regulation of cell fate decisions remain unclear. Recently, we proposed an Epigenome-Metabolome-Epigenome (E-M-E) signaling cascade model to describe crossover cooperation during reprogramming of mouse somatic cells. In this review, we summarize the broad roles of E-M-E signaling cascades in various cell biological processes, including cell differentiation and de-differentiation, cell specialization, cell proliferation and cell pathological processes. Precise E-M-E signaling cascades are critical in these cell biological processes, and each step of the E-M-E signaling cascade is worth investigating. E-M-E signaling cascade models can shed light on the mechanisms of multilevel regulation of cellular biological processes and open windows for investigating mechanisms.

CONCLUSION

Recent findings indicate the increasing importance of 'non-genetic factors' in the pathogenesis of atherosclerotic vascular disease. In fact, the inherited genome only partially determines the risk profile, as genomic approaches cannot account for the additional layer of biological regulation due to 'epi' genetic alterations. Epigenetic modifications are defined as plastic chemical changes in the DNA/histone complex that have a decisive effect on gene activity without altering the DNA sequence. These modifications include DNA methylation, post-translational histone modifications, and non-coding RNA, and have the ability to regulate gene expression at both the transcriptional and post-transcriptional level.

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