Report Title

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Paper 0
Title:Epigenetic Regulation in Neurodegenerative Diseases.

Abstract:Mechanisms of epigenetic regulation, including DNA methylation, chromatin remodeling, and histone post-translational modifications, are involved in multiple aspects of neuronal function and development. Recent discoveries have shed light on critical functions of chromatin in the aging brain, with an emerging realization that the maintenance of a healthy brain relies heavily on epigenetic mechanisms. Here, we present recent advances, with a focus on histone modifications and the implications for several neurodegenerative diseases including Alzheimer's disease (AD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS). We highlight common and unique epigenetic mechanisms among these situations and point to emerging therapeutic approaches.

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The impact factor:16.978

Paper 1

Title:The emerging field of epigenetics in neurodegeneration and neuroprotection.

Abstract:Epigenetic mechanisms - including DNA methylation, histone post-translational modifications and changes in nucleosome positioning - regulate gene expression, cellular differentiation and development in almost all tissues, including the brain. In adulthood, changes in the epigenome are crucial for higher cognitive functions such as learning and memory. Striking new evidence implicates the dysregulation of epigenetic mechanisms in neurodegenerative disorders and diseases. Although these disorders differ in their underlying causes and pathophysiologies, many involve the dysregulation of restrictive element 1-silencing transcription factor (REST), which acts via epigenetic mechanisms to regulate gene expression. Although not somatically heritable, epigenetic modifications in neurons are dynamic and reversible, which makes them good targets for therapeutic intervention.

DOI:10.1038/nrn.2017.46
The impact factor:38.755

Paper 2

Title:Epigenetic Mechanisms in Memory and Cognitive Decline Associated with Aging and Alzheimer's Disease.

Abstract:Epigenetic mechanisms, which include DNA methylation, a variety of post-translational modifications of histone proteins (acetylation, phosphorylation, methylation, ubiquitination, sumoylation, serotonylation, dopaminylation), chromatin remodeling enzymes, and long non-coding RNAs, are robust regulators of activity-dependent changes in gene transcription. In the brain, many of these epigenetic modifications have been widely implicated in synaptic plasticity and memory formation. Dysregulation of epigenetic mechanisms has been reported in the aged brain and is associated with or contributes to memory decline across the lifespan. Furthermore, alterations in the epigenome have been reported in neurodegenerative disorders, including Alzheimer's disease. Here, we review the diverse types of epigenetic modifications and their role in activity- and learning-dependent synaptic plasticity. We then discuss how these mechanisms become dysregulated across the lifespan and contribute to memory loss with age and in Alzheimer's disease. Collectively, the evidence reviewed here strongly supports a role for diverse epigenetic mechanisms in memory formation, aging, and neurodegeneration in the brain.

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The impact factor:6.208

Paper 3

Title:The Role of Epigenetics in Neuroinflammatory-Driven Diseases.

Abstract:Neurodegenerative disorders are characterized by the progressive loss of central and/or peripheral nervous system neurons. Within this context, neuroinflammation comes up as one of the main factors linked to neurodegeneration progression. In fact, neuroinflammation has been recognized as an outstanding factor for Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS), Parkinson's disease (PD), and multiple sclerosis (MS). Interestingly, neuroinflammatory diseases are characterized by dramatic changes in the epigenetic profile, which might provide novel prognostic and therapeutic factors towards neuroinflammatory treatment. Deep changes in DNA and histone methylation, along with histone acetylation and altered non-coding RNA expression, have been reported at the onset of inflammatory diseases. The aim of this work is to review the current knowledge on this field.

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The impact factor:6.208

Paper 4

Title:Epigenetic Changes in Prion and Prion-like Neurodegenerative Diseases: Recent Advances, Potential as Biomarkers, and Future Perspectives.

Abstract:Prion diseases are transmissible spongiform encephalopathies (TSEs) caused by a conformational conversion of the native cellular prion protein (PrP(C)) to an abnormal, infectious isoform called PrP(Sc). Amyotrophic lateral sclerosis, Alzheimer's, Parkinson's, and Huntington's diseases are also known as prion-like diseases because they share common features with prion diseases, including protein misfolding and aggregation, as well as the spread of these misfolded proteins into different brain regions. Increasing evidence proposes the involvement of epigenetic mechanisms, namely DNA methylation, post-translational modifications of histones, and microRNA-mediated post-transcriptional gene regulation in the pathogenesis of prion-like diseases. Little is known about the role of epigenetic modifications in prion diseases, but recent findings also point to a potential regulatory role of epigenetic mechanisms in the pathology of these diseases. This review

highlights recent findings on epigenetic modifications in TSEs and prion-like diseases and discusses the potential role of such mechanisms in disease pathology and their use as potential biomarkers.

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The impact factor:6.208

Paper 5

Title:The Essential Role of Epigenetic Modifications in Neurodegenerative Diseases with Dyskinesia.

Abstract:Epigenetics play an essential role in the occurrence and improvement of many diseases. Evidence shows that epigenetic modifications are crucial to the regulation of gene expression. DNA methylation is closely linked to embryonic development in mammalian. In recent years, epigenetic drugs have shown unexpected therapeutic effects on neurological diseases, leading to the study of the epigenetic mechanism in neurodegenerative diseases. Unlike genetics, epigenetics modify the genome without changing the DNA sequence. Research shows that epigenetics is involved in all aspects of neurodegenerative diseases. The study of epigenetic will provide valuable insights into the molecular mechanism of neurodegenerative diseases, which may lead to new treatments and diagnoses. This article reviews the role of epigenetic modifications neurodegenerative diseases with dyskinesia, and discusses the therapeutic potential of epigenetic drugs in neurodegenerative diseases.

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The impact factor:4.231

Paper 6

Title:Neuroepigenetic mechanisms in disease.

Abstract:Epigenetics allows for the inheritance of information in cellular lineages during differentiation, independent of changes to the underlying genetic sequence. This raises the question of whether epigenetic mechanisms also function in post-mitotic neurons. During the long life of the neuron, fluctuations in gene expression allow the cell to pass through stages of differentiation, modulate synaptic activity in response to environmental cues, and fortify the cell through age-related neuroprotective pathways. Emerging evidence suggests that epigenetic mechanisms such as DNA methylation and histone modification permit these dynamic changes in gene expression throughout the life of a neuron. Accordingly, recent studies have revealed the vital importance of epigenetic players in the central nervous system and during neurodegeneration. Here, we provide a review of several of these recent findings, highlighting novel functions for epigenetics in the fields of Rett syndrome, Fragile X syndrome, and Alzheimer's disease research. Together, these discoveries underscore the vital importance of epigenetics in human neurological disorders.

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The impact factor:5.465

Paper 7

Title:Histone Modifications in Alzheimer's Disease.

Abstract:Since Late-onset Alzheimer's disease (LOAD) derives from a combination of genetic variants and environmental factors, epigenetic modifications have been predicted to play a role in the etiopathology of LOAD. Along with DNA methylation, histone modifications have been proposed as the main epigenetic modifications that contribute to the pathologic mechanisms of LOAD; however, little is known about how these mechanisms contribute to the disease's onset or progression. In this review, we highlighted the main histone modifications and their functional role, including histone acetylation, histone methylation, and histone phosphorylation, as well as changes in such histone modifications that occur in the aging process and mainly in Alzheimer's disease (AD). Furthermore, we pointed out the main epigenetic drugs tested for AD treatment, such as those based on histone deacetylase (HDAC) inhibitors. Finally, we remarked on the perspectives around the use of such epigenetics drugs for treating AD.

DOI:10.3390/genes14020347
The impact factor:4.141

Paper 8

Title:Biological aging processes underlying cognitive decline and neurodegenerative disease.

Abstract:Alzheimer's disease and related dementias (ADRD) are among the top contributors to disability and mortality in later life. As with many chronic conditions, aging is the single most influential factor in the development of ADRD. Even among older adults who remain free of dementia throughout their lives, cognitive decline and neurodegenerative changes are appreciable with advancing age, suggesting shared pathophysiological mechanisms. In this Review, we provide an overview of changes in cognition, brain morphology, and neuropathological protein accumulation across the lifespan in humans, with complementary and mechanistic evidence from animal models. Next, we highlight selected aging processes that are differentially regulated in neurodegenerative disease, including aberrant autophagy, mitochondrial dysfunction, cellular senescence, epigenetic changes, cerebrovascular dysfunction, inflammation, and lipid dysregulation. We summarize research across clinical and translational studies to link biological aging processes to underlying ADRD pathogenesis. Targeting fundamental processes underlying biological aging may represent a yet relatively unexplored avenue to attenuate both age-related cognitive decline and ADRD. Collaboration across the fields of geroscience and neuroscience, coupled with the development of new translational animal models that more closely align with human disease processes, is necessary to advance novel therapeutic discovery in this realm.

DOI:10.1172/JCI158453
The impact factor:19.456

Paper 9

Title:The m(6)A epitranscriptome: transcriptome plasticity in brain development and function.

Abstract:The field of epitranscriptomics examines the recently deciphered form of gene expression regulation that is mediated by type- and site-specific RNA modifications. Similarly to the role played by epigenetic mechanisms - which operate via DNA and histone modifications - epitranscriptomic modifications are involved in the control of the delicate gene expression patterns that are needed for

the development and activity of the nervous system and are essential for basic and higher brain functions. Here we describe the mechanisms that are involved in the writing, erasing and reading of N(6)-methyladenosine, the most prevalent internal mRNA modification, and the emerging roles played by N(6)-methyladenosine in the nervous system.

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The impact factor:38.755