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Epigenetic Regulation Underlies the Vulnerability to Drug Addiction caused by Early-Life Trauma

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Abstract

Almost 70,000 Americans died from drug overdoses in 2019 alone, making drug addiction the world's largest cause of disability. Although while only a small percentage of long-term drug users develop addiction, little is known about the exact mechanisms behind this predisposition. Adversity in early life has a causal relationship to adult psychiatric illness and raises the possibility of addiction. Here, we review recent pre-clinical research that demonstrates how exposure to stress and/or drugs during adolescence controls changes in behaviour, gene expression, and the epigenome. Our review of the preclinical literature highlights research that show the frequently significant disparities between female and male subjects, as well as noteworthy results and gaps in the existing body of knowledge.

Keywords: Drug addiction; Adversity; Drug overdose

Introduction

In the past year, 20.8 percent of adults aged 12 or older used illegal drugs, according to the 2019 United States National Survey on Drug Use and Health, yet only 3.0 percent of respondents reported having an illicit drug use disorder (Substance Abuse and Mental Health Services Administration, 2020). So, drug addiction can be viewed as a disease of individual vulnerability. Despite this, it is still unclear what characteristics of an individual lead to the development of addiction. According to a large body of research, both in humans and in animal models, early life adversity increases susceptibility to adult psychiatric disorders like addiction, depression, and post-traumatic stress disorder [1, 2].

Stress in adolescence controls drug-reward behaviour in adults

Early life stress is a sort of early life adversity linked to behavioural changes, altered gene expression, and the epigenome. Early life stress in humans, such as that caused by parental separation, parental abuse, and social deprivation, alters brain activity and structure and may result in mental deficiencies Negative childhood experiences also have an impact on the processing of rewards and the speed at which rewards are learned [3,4]

Several forms of maternal separation that result in the stimulation and attenuation of the hypothalamic-pituitary-adrenal axis are used to simulate early life stress in rodents. Changes in the expression of corticotropin releasing factor (CRF) and glucocorticoid receptors (GRs) in the hypothalamic-pituitary-adrenal axis and brain reward areas are one of the neurobiological impacts of early life stress on reward exposure and motivation. Different types and lengths of early life stress have varied effects on the epigenome, transcription, and behavioural outcomes (Levis et al., 2021). The age of the pups at the time of separation (usually postnatal day 1–15), the length of the separation, and differences in severity are all variants to the mother separation paradigm [5, 6].

15 minutes of mother absence is seen as somewhat stressful, while 180–360 minutes is regarded as severely traumatic. The limited nesting and bedding paradigm, in which dams have restricted access to the availability of nesting and bedding materials, is another way to represent early life stress [7, 8].

Conclusion

Adversity in early life is a significant factor in both men and women's vulnerability to addiction. Adversity in early life has sex-, brain-region-, and exposure-specific impacts on addiction susceptibility, depending on the particular early adversity and adult drug experience. The underlying biological mechanisms include stress in early life, sex-specific regulation of genome-wide epigenetic alterations in neurons, and gene expression. Although age of exposure and sex both affect vulnerability to addiction, research has primarily used data from adult males [9, 10].

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Conflict of Interest

None.

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