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Shared Genetics between Depression and Cardiometabolic Disorders

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Introduction: Several studies have discovered associations between depression and cardiovascular disease risk factors and patients with Coronary Artery Disease with comorbid Depression have worse prognosis. There is growing evidence of polygenic overlap between depression, Coronary Artery Disease and other Cardiovascular risk factors and may suggest molecular mechanisms underlying the association between depression and raised cardiovascular disease risk.
Objectives: Depression and cardiometabolic disorders are both heritable and both are caused by a mix of genetic and environmental factors. Genetic factors contribute to 31–42 % in MDD (Sullivan et al. 2000), 30–60 % in coronary artery diseases (Marenberg et al. 1994), 26–69 % in type 2 diabetes (Almgren et al. 2011 ; Poulsen et al. 1999), 24–37 % in blood pressure (hypertension) (Van Rijn et al. 2007), 35–48 % in heart rate variability (Kupper et al. 2004), 40–70 % in obesity (body mass index) (Willyard 2014), and 58–66 % in the level of serum lipids (Knoblauch et al. 1997). Moreover, there are fairly high genetic co-heritabilities (genetic correlations) between depression and the different cardiometabolic disorders suggesting the influence of pleiotropic genes and shared biological pathways within them.
Methods: Literature search was conducted and appropriate material was then extracted to examine the hypothesis.
Results: Identification of shared molecular pathways supports a growing evidence base for cross-diagnostic treatment. Besides, further exploration of overlapping molecular pathophysiology can unveil novel targets for drug development and repurposing of existing medications. Also, cardiometabolic disorders can increase the risk of poor response to standard treatments in mood disorders. Lastly, studying shared pathways of depression and somatic disorders can untangle the clinical and genetic heterogeneity that underlies in these illnesses.
Conclusions: Genetic studies have suggested the involvement of pleiotropic genes in the comorbidity between depression and cardiometabolic disorders. While our abstract attempts to provide some insight into the common mechanisms and role of pleiotropic genes, in-depth understanding of how these genes mediate the association between depression and cardiometabolic diseases requires future larger scale comprehensive cross-disorder research. This will enable us to better understand why patients suffer from multiple diseases at a time and how multimorbidities influence pharmacological treatment response to diseases.

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The Impact of Bisphenol A (BPA) on Serotonin Regulation and Its Implications for Mood

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Introduction: Bisphenol A (BPA), a widely used chemical in the manufacturing of plastics and resins found in everyday consumer products such as water bottles and food containers, is known to interfere with the body's endocrine system. Specifically, BPA acts as a xenoestrogen, meaning it can mimic the hormone estrogen by binding to estrogen receptors (ERs) in the body. This interaction raises concerns about BPA's potential impact on brain regions with high densities of these receptors, including the hippocampus and prefrontal cortex, which are critical for mood and cognitive functions. Estrogen plays a vital role in regulating serotonin, a neurotransmitter essential for mood stability. This review synthesizes existing literature on how BPA may disrupt serotonin regulation and its implications for mood disorders.
Objectives: To evaluate the mechanisms by which BPA interacts with estrogen receptors and serotonin in the brain with a key focus on its implications for mood disorders such as depression and anxiety.
Methods: A narrative literature review was conducted, gathering findings from relevant studies published over the past two decades. Sources were identified through database searches in PubMed, PsycINFO, and Google Scholar using keywords such as "BPA," "serotonin," "estrogen receptors," "hippocampus," "prefrontal cortex," and "mood disorders." The review focused on various types of studies that examine BPA's interaction with estrogen receptors and its effects on serotonin regulation allowing for the creation of an up-to-date integrative summary.
Results: The review reveals that BPA's interaction with estrogen receptors in the brain can lead to significant disruptions in serotonin regulation. BPA competes with endogenous estrogen for ER binding leading to altered serotonin synthesis and release. BPA exposure is associated with reduced expression of tryptophan hydroxylase, the enzyme critical for serotonin production, potentially lowering serotonin levels. BPA's interference with ERs impairs serotonin release from presynaptic neurons, particularly in the hippocampus and prefrontal cortex, which are vital for mood regulation.
Conclusions: The literature strongly suggests that BPA disrupts normal estrogenic regulation of serotonin, with significant implications for mood disorders such as depression and anxiety. The hippocampus and prefrontal cortex appear particularly vulnerable to these disruptions, which could theoretically exacerbate symptoms of mood dysregulation highlighting the importance of considering environmental factors like BPA in the treatment of mood disorders. Given the widespread exposure to BPA, these findings underscore the need for further research into its long-term effects on mental health and potential regulatory measures to mitigate exposure.

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