

Discover Novel, Shared and Disorder-Specific Genetic Architectures in Major Depression, Insomnia and Chronic Pain

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Abstract

Chronic insomnia, a public health crisis affecting 10-15% of the US population and costing billions of dollars per year, is frequently associated with one or more comorbid psychiatric or organic conditions. Chronic insomnia has historically been classified as "secondary" to a presenting comorbid condition, resulting in under-recognition and under-treatment of both the insomnia and the comorbid condition (s). Chronic insomnia receives little, if any, public policy attention despite being critical in any model of comorbid disease management.

Keywords: Antidepressant drug • Depression • Genetics/genomics

Introduction

All psychiatric disorder controls were screened for the absence of lifetime SA. If possible, controls from general population cohorts were screened for the absence of SA; however, due to the low prevalence of SA in the general population, some cohorts included unscreened controls. In this study, no controls were screened for suicidal ideation or non-suicidal self-injurious behaviour. In the primary SA GWAS, 29,782 cases and 519,961 controls from 18 cohorts were included. An independent replication cohort of 14,089 SA cases and 395,359 controls from the Million Veteran Program was used to test for genome-wide significant associations with SA [1].

Description

During cancer screening, diagnosis, therapy and recurrence, anxiety is frequently evident. It can sometimes influence a person's health-related behaviour, adding to the delay or omission of cancer-prevention measures. Women with high levels of anxiety, for example, may undertake breast self-examination less frequently if they realise that they have a genetically higher risk of having breast cancer than they previously assumed. Anxiety can increase the likelihood of pain, other distressing symptoms and sleep difficulties in cancer patients and it can also play a role in anticipatory nausea and vomiting [2]. Identifying the potential causes of psychological anguish and then resolving to take efforts to reduce or overcome it is the first stage in effective dealing with psychological distress. This may entail psychiatric counselling in order to identify the source of the psychological suffering. A psychiatrist, psychologist, or other mental health practitioner may recommend a variety of therapeutic treatments to assist relieve psychological discomfort as part of the counselling.

Fear is an emotion that evolved to protect us and improve our ability to survive when we perceive danger or threat. Fear is an understandable feeling

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given the pandemic's primary concerns of illness and death. Validating the person's emotional experience, within reason, is the first step in treating with pandemic-related dread. It's also crucial to let the person understand that fear may be used as an ally rather than something to be conquered. When fear is channelled correctly, it protects both the individual and others by causing protective behaviours such as hand washing, mask wearing, social distancing and limiting non-essential activities [3-5].

Conclusion

All psychiatric disorder controls were screened for the absence of lifetime SA. If possible, controls from general population cohorts were screened for the absence of SA; however, due to the low prevalence of SA in the general population, some cohorts included unscreened controls. In this study, no controls were screened for suicidal ideation or non-suicidal self-injurious behaviour. In the primary SA GWAS, 29,782 cases and 519,961 controls from 18 cohorts were included. An independent replication cohort of 14,089 SA cases and 395,359 controls from the Million Veteran Program was used to test for genome-wide significant associations with SA.

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Conflict of Interest

There are no conflicts of interest by author.

References

1. McEwen, Bruce S., Nicole P. Bowles, Jason D. Gray and Matthew N. Hill, et al. "Mechanisms of stress in the brain." *Nat Neurosci* 18 (2015): 1353-1363.
2. McEwen, Bruce S. and John H. Morrison. "The brain on stress: vulnerability and plasticity of the prefrontal cortex over the life course." *Neuron* 79 (2013): 16-29.
3. Miller, Bradley R. and René Hen. "The current state of the neurogenic theory of depression and anxiety." *Curr Opin Neurobiol* 30 (2015): 51-58.
4. Price, Joseph L. and Wayne C. Drevets. "Neurocircuitry of mood disorders." *Neuropsychopharmacology* 35 (2010): 192-216.
5. Scorza, María Cecilia, Laia Lladó-Pelfort, S. Oller and R. Cortés, et al. "Preclinical and clinical characterization of the selective 5-HT1A receptor antagonist DU-125530 for antidepressant treatment." *Br J Pharmacol* 167 (2012): 1021-1034.

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