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Fast-acting antidepressant and the circadian clock

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Major depressive disorder is one of the most serious of the psychiatric disorders and is associated with high rates of suicide. Worldwide, depression affects over 300 million people of all ages and is the leading cause of lifetime disability out of all medical disorders according to the World Health Organization (WHO). Despite widespread use of classic antidepressants, globally, suicide accounts for more than 800,000 deaths per year, reflecting the limitations of monoaminergic-based treatments. Therefore, the development of a novel generation of antidepressant therapies acting remarkably fast (within 24 hrs to a few days) not only represents a new hope for the patients suffering from this disease, but also opens new directions to investigate the pathophysiological mechanism of depression and concomitantly complex brain functions. Epidemiological, clinical, and experimental evidence over the past 30 years has clearly established a causal link between circadian disruptions including sleep disturbances, hormonal secretions, temperature and mood in a subset of mayor depressive disorder and bipolar disorder patients. This coincides with the permeation of modern lifestyles which induces sleep disorders, stress and circadian disruption, stemming from the so-called “social zeitgebers”. These environmental factors can disrupt the normal circadian rhythms. Circadian abnormalities associated with mayor depressive disorder include dysregulated rhythms of sleep, temperature, hormonal secretions, and mood all of which are modulated by clock genes. Fast-acting antidepressants such as low-dose ketamine and sleep deprivation therapy can improve symptoms within 24 hrs in a subset of depressed patients in striking contrast to conventional treatments which sometimes require weeks for a full clinical response. Analyzing the mouse anterior cingulate cortex, a region associated with depressive symptoms, we found by comparative transcriptomics analyses, common transcriptional responses to both ketamine and sleep deprivation therapies. Interestingly these responses were implicated in the circadian clock and processes involved in neuronal plasticity. Our findings open new research avenues to help design chronopharmacological strategies to treat major depressive disorder.

Biography

Ricardo Orozco-Solis is from National Institute of Genomic Medicine which is in Mexico.