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Abnormal interactions of neurodevelopment with drugs of abuse trigger permanent deficit in the endocannabinoid system-dependent brain natural ability to alter and protect itself Predictors of involuntary hospitalizations to acute psychiatry

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Cannabis abuse is considered to be the serious, if not the greatest known environmental risk for neuropsychiatric disorders. Individuals who are exposed to cannabis experience variety of psychoactive effects such as general alteration of conscious perception, euphoria, problems with social interactions, memory and learning, and occasionally anxiety and paranoia. Our research indicates that abnormal interactions of neurodevelopment with the environment triggered by drugs of abuse during neonatal or adolescent periods may permanently impair brain function including the brain natural ability to alter and protect itself, i.e. endocannabinoid system (eCB)-dependent inherent neuroprotection of circuit integrity and neuroplasticity. The eCB system represents a major activity-dependent regulatory system in the central nervous system and has been implicated in multiple brain functions, including synaptic plasticity and the homeostatic regulation of network activity patterns. Noteworthy, deficiency in eCB signaling found in developmental model for psychosis is associated with abnormalities in prefrontal cortex-dependent fear discrimination learning. We also show that mouse model of adolescent cannabis abuse shows deficits in an endocannabinoid -mediated signaling and neuroplasticity in adult prefrontal cortex, a brain region encompassing neural circuit for decision-making. Blockade of the primary gene product responsible for degrading the endogenous endocannabinoid, with the specific drug ameliorates these deficits. The observed deficit in endocannabinoid-dependent signaling in the brain may represent a neural maladaptation underlying cortical network instability and abnormal cognitive functioning triggered by overactive CB1 receptor during adolescence. The validity of these observations to human conditions relies on their criteria and translating data obtained in mouse to human behavior and physiology. These results suggest that transiently overactive CB1 receptor in the brain during adolescence leads to permanent alterations in the endocannabinoid system dependent signaling and neuroplasticity. Cannabis abuse during adolescence increases the risk of schizophrenia, which involves developmental maladaptations in neural circuitry that result in impaired brain functioning. In addition, the endocannabinoid system is altered in the brains of people with schizophrenia. These findings provide additional insights into the pathological processes related to increased risk for neuropsychiatric disorders and point towards developing new treatment strategies.

Biography

Korzus is an Assistant Professor in Neuroscience and Psychology at University of California Riverside. He completed his Ph.D. in Molecular Biology and Biochemistry at the University of Georgia-Athens. He completed a postdoctoral fellowship at Howard Hughes Medical Institute at University of California San Diego and is the recipient of numerous Medical Research Awards. Currently, he is a P.I. of the National Mental Health Institute's research program "Prefrontal circuit and function in memory accuracy" and the holder of NARSAD Award.

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