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Commentary on The Epidemiology of DSM-5 Marijuana Use Disorders among Adults in the USA: *Science to Inform Clinicians Working in a Shifting Social Landscape*

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The study of marijuana use disorders has become urgent because of several factors: Increasing marijuana legalization in multiple states and jurisdictions (1), the high and increasing prevalence of marijuana use (2), prospective evidence of the impact of marijuana use on future risk of psychiatric disorders (3), and evidence for multiple deleterious effects of marijuana exposure (4). It is against this backdrop of a rapidly changing legal landscape and emerging evidence of harms from marijuana use that we should consider Hasin and colleagues' rigorous analysis of the prevalence, demographic characteristics, psychiatric comorbidity, disability and treatment for DSM-5 marijuana use disorders in the US adult population (5). This study will go a long way towards helping psychiatrists and all clinicians to treat patients more effectively and participate more actively in policy discussions.

We knew, going into this study that 12.5% of persons age 18 or older in the US reported past 12-month use of marijuana in 2013 (2). This prevalence is about 19% higher than the 10.5% found in 2002 (2), an increase that was likely fueled, at least in part, by the inverse relationship between marijuana use and the perception of harmfulness (6). This trend is problematic because marijuana use is associated with increased risk for a number of adverse cognitive, psychiatric, physical, and social effects, including comorbid mental disorders (3–4).

The increasing prevalence of marijuana use combined with the evolving definition of marijuana use disorder in the revised DSM-5 nomenclature warranted reassessment of marijuana epidemiology. For instance, changes in DSM-5 (compared to DSM-IV) included the addition of withdrawal as a recognized component of marijuana use disorder. DSM-5 also included universal changes for all substance use disorders, including marijuana use

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disorders. Of particular note, DSM-5 eliminated the DSM-IV abuse and dependence disorders in favor of a single, unified substance use disorder with severity determined by the number of criteria endorsed (7).

Hasin's paper reports findings from the landmark National Epidemiologic Survey of Alcohol and Related Conditions-III, which included psychiatric interview data from 36,309 persons age 18 or older in 2012–2013, to determine the rates of DSM-5 marijuana use disorder and its correlates (5). Marijuana use disorder was found to be prevalent, with past year rates of 2.5% and 6.3% lifetime. Rates were higher among men, Native Americans, unmarried persons, younger persons and those with low incomes. Comorbidity was found to be common, with strong associations of marijuana use disorders with other substance use disorders, affective disorders, anxiety disorders and personality disorders.

Factual information about marijuana use disorder is sorely needed, and Hasin and colleagues' paper is likely to become the standard reference on the topic. Particularly compelling is the information about demographic correlates and comorbidity. The demonstration of strong associations with other substance use and psychiatric disorders (personality, anxiety, and mood) is consistent with previous studies based on DSM-III, III-R and IV criteria, but the addition of information about how increasing severity of DSM-5 marijuana use disorder is reflected in the increasing strength of the associations is quite novel. It appears that the severity subtypes implemented in DSM-5 are reliable predictors of such associations. Hasin and colleagues found that increasing DSM-5 severity was associated with poorer functioning, stronger correlation with risk factors, and increased prevalence of comorbid psychiatric disorders (5). These findings suggest that clinical problems associated with marijuana use disorder exist along a severity continuum, like the disorder itself.

The low rates of treatment documented by Hasin and colleagues are also noteworthy (5). Only 13.7% of adults with a lifetime marijuana use disorder ever sought any type of treatment or intervention. Even among those with a severe marijuana use disorder, only 24.3% reported seeking any such assistance. Needed are both effective interventions, including medications, for marijuana use disorders as well as increased patient motivation to seek such care.

Future work might benefit from a longitudinal design. While cross-sectional research such as the work by Hasin and colleagues (5) helps to demonstrate important correlates of disorder, details about possible reasons for the associations (i.e., causal pathways) are better addressed in longitudinal studies. For instance, the recently launched National Institutes of Health Adolescent Brain Cognitive Development (ABCD) study proposes to study exposure to marijuana and other substances in repeated longitudinal examination of children from pre-adolescence though their teen years. The ABCD study will include careful assessment of substance exposure along with psychosocial risk factors and mental illness and so will be well positioned to test the associations found by Hasin and colleagues. Second, examining whether and how changes in rates of marijuana use may inform the associations found by Hasin and colleagues remains a research challenge. It remains to be determined whether and how many of the associations are with marijuana use disorder or, more directly, with use of

marijuana itself. Future research may be needed to consider whether the associations are fully explained by greater use of marijuana or by greater risk of a disorder, over and above the association with marijuana use, *per se*.

Limitations notwithstanding, the Hasin et al. paper should be received with enthusiasm for it is the first full epidemiological study of DSM-5 marijuana use disorder. The current findings supporting the dimensional approach in DSM-5 are particular noteworthy in this context, for validation of the severity subtype may have significant implications for screening and treatment planning.

To capture their true significance, the findings from Hasin and colleagues (5) should be analyzed in conjunction with the growing neurobiological evidence pertaining to the potential disruptive impact of marijuana use on brain development and mental health. We now know that the endocannabinoid system supports a core signaling mechanism that optimizes information processing and performance by fine-tuning the balance between inhibition and excitation throughout the brain (Figure 1A). This mechanism is the key to understanding not only the endocannabinoid system's involvement in psychiatric disorders but also in synaptic pruning and white matter development, two neurodevelopmental processes that are highly orchestrated and particularly active during adolescence. Exogenous administration of a cannabinoid (such as THC) perturbs normal signaling through the endocannabinoid system (Figure 1B). Thus, repeated THC exposure may lead to persistent dysregulation in a broad range of neurotransmitter systems, including dopamine, serotonin, GABA and glutamate, across a vast network of circuits that rely on the endocannabinoid system to optimize developmental processes, adaptive behaviors, and overal brain performance (8–10). This helps explain some of the adverse consequences that have been associated with marijuana use, particularly when it is used regularly beginning in adolescence (4).

Given the shifting marijuana legal and socio-cultural environment, clinicians require accurate information to guide practice development. When seen in light of a growing body of neurodevelopmental work on marijuana's effects on adolescent brain maturation processes, concerns about the potential harms associated with marijuana use and marijuana use disorder require public health vigilance. Findings from Hasin and colleagues help to address this gap and make a strong case for the need to enhance marijuana prevention and education efforts.

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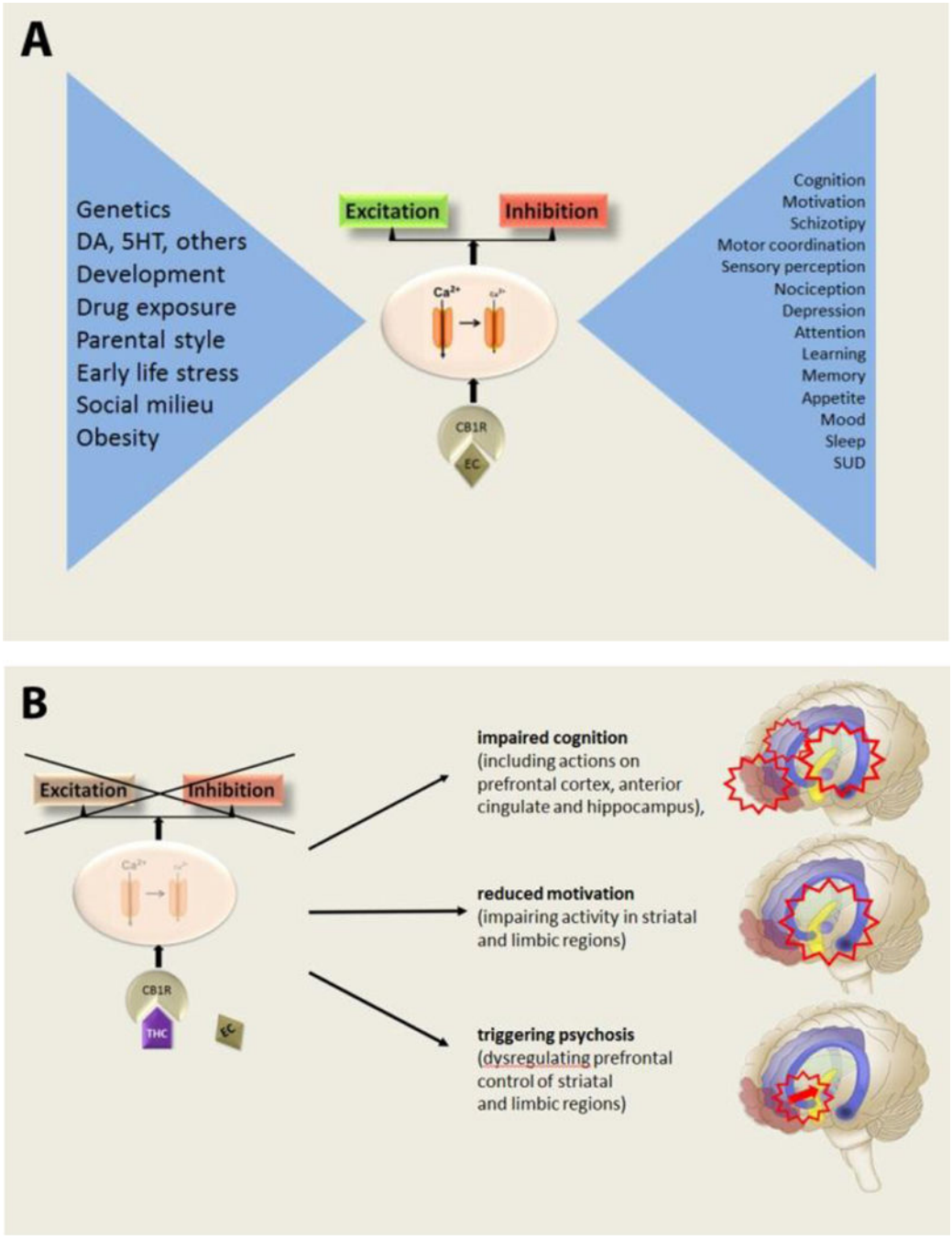

Figure 1.

A. The endocannabinoid system (ECS) is a core signaling mechanism that optimizes the balance between inhibition and excitation in multiple brain circuits. Retrograde activation of type 1 cannabinoid receptors (CB1Rs) by ECs (i.e., 2-AG and anandamide) inhibits Ca2+ channels thereby decreasing neurotransmitter release. Thus, EC signaling is endowed with the strategic ability to enhance both local inhibitory and excitatory tone through depolarization-induced suppression of stimulation or depolarization induced suppression of inhibition, respectively. As a result, like many other key control mechanisms, and because of

its fundamental pharmacological properties and broad distribution, the ECS can be viewed as the core of a “bow tie’ regulatory architecture where EC signaling is influenced by and in turn can influence a large number of normal and pathological processes (afferent and efferent blue triangles, respectively). **B.** A robust association between regular marijuana use, marijuana use disorder, and other, comorbid psychiatric conditions is consistent with the fact that exogenous administration of a cannabinoid like THC perturbs normal signaling through the ECS, leading to dysregulation of a broad range of neurotransmitters, inlcuding dopamine, serotonin, GABA and Glutamate, throughout a vast network of circuits that rely on the ECS to fine tune devepmental processes, adaptive behaviors, and overal brain performance. Thus, the neurobehavioral impact of THC-mediated interference of ECS function will vary depending on the time (developmental effects) and location (regional effects) of THC’s actions. Highlighted in this panel are the prefrontal cortex (maroon), anterior cingulate cortex (light purple), striatum with the NAc (yellow), and the hippocampus (blue) and some of the specific disruptions that may account for marijuana’s impact on cognition, motivation, and schizotipy.