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Commentary

Challenges in Recovery Research from a Neuroscience Perspective

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Abstract

This commentary addresses some of the contemporary challenges to recovery research from the neuroscience perspective. The authors examine advances in the neuroscience of substance use disorder while highlighting the fact that equal advances have not been made in advancing understanding of recovery from substance use disorder. They note particular challenges in the inclusion of emerging adults and minority populations in recovery research and make recommendations for ways to move forward in establishing a unifying theoretical framework of the neurobiology of recovery from substance abuse disorders that may benefit researchers, practitioners, and policy makers working in the recovery community.

Keywords: Emerging adults; Executive function; Preclinical models; Recovery research; Substance use disorders

Introduction

Neuroscience has advanced our understanding of the chronic, relapsing brain disorder referred to now as Substance Use Disorder (SUD). Despite these advances from within the field of neuroscience the same systematic investigation has not been equally applied to resolution of Substance Use Disorders (SUD), also referred to as recovery, in a similar manner. George Koob and Nora Volkow [1] synthesized twenty years of neuroscience research into the medical model of SUDs. The widely accepted and updated framework of the neurobiology of SUDs includes well defined constructs of preoccupation, binge/intoxication, and withdrawal corresponding with brain systems of the prefrontal cortex, basal ganglia, and amygdala respectively [2]. While this elucidates the process of intermittent use to compulsive use similar investigations have not been applied to those in long-term recovery. Based on existing literature, we propose executive function (the metacognitive and mental skills needed for optimal decision making), as a starting point to characterize neurocognitive processes of recovery as a distinct and independent process from developing substance use disorders [3]. This commentary explores the obstacles in conducting research in recovery populations, solutions to advance research efforts using clinical and preclinical models, and a call to action for further neurobiological investigations of recovery processes and milestones to address the gaps in literature necessary to synthesize a unifying theoretical framework of the neurobiology of recovery from substance use disorders.

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Challenges in Recovery Research

Complicating the issue of research is a lack of a universally adopted definition of the construct of recovery. The National Institute of Alcohol Abuse (NIAAA) and Alcoholism Alcohol Use Disorder (AUD) Recovery Roundtable [4] conceptualizes recovery as a process of "remission from AUD and cessation from heavy drinking" but no universal recovery definitions exist for other substances, further complicating efforts to disentangle recovery processes from addiction processes. For this commentary we define recovery as intentional, motivated behaviors to resolve problematic substance use and prevent return to use and can include harm reduction and moderation of use [5,6]. There are many pathways and methods for recovery support services and we do not endorse a singular, specific pathway. Each individual has a unique and variable path including differences in age at first use, drug of choice, route of administration, and other psychosocial variables that lead to dysregulated brain function and compulsive substance use. Rather, we argue that there are likely universal neurobiological mechanisms that occur during recovery efforts that, once uncovered by addressing the obstacles in recovery research, can be synthesized into an encompassing theory on the neuroscience of recovery. Complicating the issue of addressing gaps in our understanding is the difficulty in conducting research in a population with loosely defined constructs and with limited clinical guidelines. For example, results from the National Survey on Drug Use and Health estimated that 21.9% of people aged 12 or over reported using illicit drugs in the last year, with the highest rate in young adults ages 18-25 [7]. Further, 15.3% of the population reporting alcohol or illicit drug use are estimated to meet the criteria for having a use disorder based on current clinical definitions. Efforts should go towards prevention and intervention for these individuals and include best practices based on recovery-informed theories, and further bolstered by including neuroscience of recovery theories in the future. For the focus of this paper, we emphasize that results from SAMSHA showing perceptions of substance use disorder match closely with diagnostic criteria such that 11.6% of the population report they perceived themselves to have a use disorder and within those that perceive they have ever had a use disorder nearly 72% consider themselves in recovery or have recovered from an alcohol or substance use disorder [7]. Thus, self-awareness of problematic alcohol or drug use corresponds to self perceived recovery status.

Notably, these perceptions are based on self reports and perceptions without an objective measure of recovery, unlike diagnostic

criteria for a substance use disorder. Thus, this sample may represent individuals that may not engage with recovery support services if they perceive themselves to be recovered. Subsequently, this population would not encounter efforts for recruitment for research to advance recovery science. On the other hand, individuals seeking treatment for an active substance use disorder through physician offices, treatment centers, or other means have more opportunities to engage in research efforts for recovery research. Systematic and controlled studies exist with patients seeking treatment for AUD and SUDs but outcomes focus on treatment effectiveness in maintaining abstinence and not on elucidating mechanisms of behavioral change [8]. Further, relying on neuropsychological data collected from those early in SUD treatment, such as receiving care in residential facilities, gives only a brief snapshot into the timeline of recovery mechanisms. Together these issues create obstacles at every level of study design including recruitment, efficacy studies of interventions, generalizability, and creating a consistent, unifying framework.

Focus on Young Adults and Executive Function

Our best efforts towards understanding recovery processes is to study groups that have spent more time resolving moderate to severe use disorders than in active addiction. Previous studies making this attempt have utilized individuals in early treatment, usually in residential treatment settings, where the average age of those entering treatment is 34 years [9]. Young adult populations are an ideal candidate population to study to understand recovery processes as distinct, and separate from chronic substance use. Young adulthood is characterized as a transition between adolescence and adulthood and encompasses several developmental themes including identity exploration, experimentation/possibility, independence and social connections [10]. Young adults in recovery are a heterogenous group in how they started their recovery journey but likely share common biopsychosocial traits, yet these commonalities remain understudied. In contrast, the last 20 years of research in addiction routinely report individuals in active addiction share characteristics in impaired prefrontal cortex control [11], increased attention to drug stimuli [12,13], and recruitment of stress and amygdala system [14] despite varying pathways to dependence (e.g. different age of initiation, reasons to start, social supports etc) and substance of choice. Thus, young adults likely share neuropsychological features that support sustained recovery, but recovery mechanisms are yet to be determined.

We propose a logical first step to developing a framework of the neuroscience of recovery is to characterize executive function in young adults based on self-reported recovery status and integrate these findings into the construct of recovery. Executive Function (EF) is a neuropsychological construct of higher-order cognitive processes that govern planning and executing goal-directed behavior and domains including inhibitory control, working memory, and cognitive flexibility. Together, these cognitive processes orchestrate complex evaluations of situations, expectations, and outcomes to guide behaviors such as reasoning, decision-making, and problem solving. Chronic substance use results in impaired executive functioning (decreased prefrontal cortex activation) in older adults but little is known regarding the impact of chronic substance use on EF in young adults.

The neurodevelopment sequalae of young adulthood also makes this epoch an ideal group to further our understanding of neurocognitive milestones achieved during recovery. For example, the prefrontal cortex reaches maturity during young adulthood with resulting increases in executive functioning capacity. Improvements in executive function occur throughout adolescence and emerging adulthood (ages 18-25) and reach stabilization in young adulthood [15,16]. In fact, EF assessments in college-aged students predict academic outcome [17], identity achievement [18] and increased resiliency that buffers stress-induced impairments and reduces health complaints [19]. In contrast, executive dysfunction in adolescents and young adults predicts maladaptive coping [20] and increases in alcohol use, substance use, and other risky behaviors. Further, among young adult heavy drinkers, EF impairments measured at baseline predicted increased alcohol consumption when working memory was taxed [21]. Further, early initiation of cannabis impairs cognitive flexibility, independently of increased use or frequency [22]. Despite the known role impaired executive function plays in risk of initiation and development of a use disorder, efforts should be taken to characterize executive function in young adults in recovery as distinct and separate mechanisms and integrate these findings into a generalized neurobiological theory of recovery. Doing so can advance refined interventions that can facilitate shorter windows to achieving stable recovery across the lifespan.

Additional challenges

Young adults report the highest frequencies of substance use and about 1 in 7 meet the diagnostic criteria for substance use disorder [7]. Unfortunately, several studies report unfavorable outcomes in emerging adults seeking treatment for Alcohol and Substance Use Disorders (ASUD) [23,24]. Emerging adults report the lowest motivation to enter treatment [25,26] and poor outcomes compared to adolescents and adults, including reduced recovery capital, treatment dropout, and return to use. Young adults are an under-investigated population regarding neuropsychological profiles (e.g., psychosocial factors, milestone achievements, and executive function) of Alcohol and Other Drug (AOD) use and/or resolution of ASUD largely because they are underrepresented in studies of intensive ASUD treatment due to poor retention. Further, most studies on young adults in recovery report outcomes for young adults in recovery that concern service utilization, demographics, and phenomenological assessments of experiences in SUD treatment or recovery [27-31]. Yet, without corresponding evidence of improvements in objective measures, such as EF, we cannot address the issues of barriers to treatment. There is a need to overcome the barriers in efforts made to advance recovery science that can aid in developing recovery-informed interventions that can generalize across the lifespan.

Another challenge is the lack of inclusion of minority populations in the research. There are many barriers to accessing recovery services for minority populations, making it even less likely they will be included in recovery research [32,33]. Historically marginalized and under-represented populations tend to be distrustful of research and researchers as a result of a history of unethical research conducted on minorities, and a higher rate of stigma and criminalization of substance use disorders for group members. Subbaraman and colleagues [34] emphasize the need to use non-traditional recruitment strategies to increase the representation of minorities in recovery research. Community based participatory research, such as the equitable community and academic partnership proposed by Gilgoff and colleagues [35] are another strategy to reduce distrust, repair relationships between the academic researchers and ethnic minority communities in order to increase representativeness in recovery research. Using strategies that improve recruitment and engagement in minority populations could

be equally applied to strategies to engage the recovery population at large and address the barriers to access in minority populations at the same time.

Preclinical Models: Ecological Validity and Challenges

Preclinical models have provided insight into the neurobiological underpinnings of abstinence. These models rely on chronic exposure to a singular drug and measure outcomes following a forced period of abstinence. Results from these studies have elucidated changes in the brain and in behavior that underly vulnerabilities to relapse after protracted periods of abstinence from a variety of substances, including changes to the PFC leading to reinstatement of drug seeking behaviors [36], deficits in learning and memory [37,38], and altered responses to subsequent exposures [37,39]. Research has framed these outcomes as important to address and prevent relapse but not in a purposeful way to the process of achieving long-term recovery. Preventing relapse is a quantifiable and tangible measure of SUD treatments but does not encompass the wholistic approach to model recovery mechanisms. The field can achieve this by placing efforts into systematic investigation into a natural return of function regarding these brain systems at varying timepoints during abstinence from chronic substance exposure. Thus, future studies should examine the effects of long-term abstinence at multiple time points and include easily accessible and translatable interventions.

Certainly not all human experiences can be replicated, and the advantage of model organisms and laboratory studies is the tight control of extraneous variables to develop casual mechanisms leading to compulsive intake. For example, individuals seeking SUD treatment often report polysubstance use [40]. The rate of co-occurrence alcohol use disorder for individuals entering treatment for opioid is estimated to be 26.4% [41] and for co-occurring use in cocaine users it is 77% [42]. Yet, many studies using rodents as model organisms look at the effects of a singular substance for the purpose of elucidating specific mechanisms within biological systems. This further complicates issues in developing new therapies by ignoring potential pharmacological interactions for the sake of statistical significance supporting mechanistic hypotheses. Designing experiments that emphasize polysubstance exposure may require greater upfront costs, but the results are necessary to appropriately address current trends in substance use and their future consequences.

Efforts to include experimental timepoints across the lifespan are crucial to advancing our understanding of the neuroscience of recovery. For example, high incidences of adverse childhood experiences can motivate initial substance intake and also increase the risk for developing a severe substance use disorder during adolescence or young adulthood [43-45]. Preclinical models can use early life stressors to model this phenomenon but less is understood on how childhood trauma and substance use interact to impact neurobiological functioning later in life or during recovery efforts [46]. An obstacle to conducting preclinical protracted abstinent models is the associated costs of housing rodents for longer periods with no active data collection but can be overcome by strategic experimental planning. The urgent need for such studies despite associated costs and limitations is crucial to elucidate the neurobiological systems engaged in recovery processes vs. use disorders and ultimately inform better recovery theories and interventions.

Conclusion

The biopsychosocial characterization of recovery mechanisms in behavior changes and cognitive processing is a gap in understanding that this commentary seeks to address as a means to reduce the societal burden of substance use disorders. Understanding differences in executive functioning and identifying other factors that may mediate this relationship (stress, trauma, stigma, recovery capital etc.,) in young adults in recovery can improve services delivered to prevent treatment dropout and return to use. Thus, including measures of executive function refines our understanding of recovery mechanisms and provides a path towards developing objective markers and measurable indications of neuropsychological changes that are necessary for motivated behavior changes and overall improve our delivery of SUD services.

Finally, we acknowledge that recovery is often conceptualized as an intangible spiritual transformation that is immeasurable and agree that recovery is a highly individualized experience [47,48]. Advances in neuroscience research techniques allowed for addiction to become less stigmatized by showing indisputable evidence of brain changes. Similar efforts must be made to understand the changes in the recovering brain that are undetected by the human eye. Understanding the neuroscience of addiction and recovery as two sides of the same coin will lead to better strategies to reduce substance use and support the recovering brain in innovative ways that reduce time to stable recovery and ultimately reduce the number of lives lost to substance use disorders.

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