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Transitioning from DSM-IV Abuse to Dependence: The Essence of Harmful Compulsive Substance Use is Ontogenetic and Dynamic

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Flórez-Salamanca and colleagues' study¹ of transitioning from DSM-IV substance abuse to dependence in this issue of *The American Journal of Drug and Alcohol Abuse* is important, in part, because the topic represents the juncture of several contemporary unresolved issues that are critical to healthcare. Harmful compulsive substance use, termed substance use disorder (SUD) in Diagnostic and Statistical Manuals (DSM), is among the most prevalent medical conditions. Lifetime DSM-IV SUD prevalence in the U.S. consequent to habitual use of tobacco, alcohol and illegal drugs, respectively, is 24%, 20% and 10%.^{2–4} Globally, smoking is the 4th leading contributor to disease burden, alcoholism is the 5th leading cause and illegal drug use is the 15th.⁵ In the U.S. alone, harmful substance use costs the nation well over \$400 billion annually⁶ due to consequences such as SUD treatment, crime, traffic accidents, medical and psychiatric disorder, domestic violence, child abuse/neglect, underemployment and job-related injury, suicide, traumatic injuries, and sexually transmitted disease including HIV/AIDS. Thus, research on SUD etiology and developmental transitions has tremendous potential to benefit at-risk individuals, their families and society.

A fundamental unmet need in SUD research and nomenclature is to characterize harmful compulsive substance use,⁷ including SUD diagnosis per se. The lack of a singular epitome of SUD obviously hinders the study of SUD transitions. To illustrate the heterogeneity of the population that qualifies for an SUD, DSM-IV criteria allow for 466 combinations of symptoms/criteria to qualify for an SUD related to use of a particular drug (not withstanding additional variance due to clinical judgment).⁸ This number of combinations increases over four-fold to 2,036 in the anticipated DSM-5 nomenclature as abuse and dependence criteria will be merged into a single diagnostic category.⁹ DSM-5 nomenclature provides a quantitative basis for severity, as diagnoses are based on a count of criteria (similar to factor-analysis based scoring of psychological measures),¹⁰ which simplifies clinical diagnosis compared to parsing abuse criteria (the putative lesser form of SUD) from dependence criteria (the more severe form). However, the change from DSM-IV to DSM-5 may further complicate the study of transitions from milder to more severe forms of SUD (e.g., if a simple count of symptoms does not well-represent severity as is suggested by item response theory research).¹¹

One research focus that has been used to learn how to improve prevention and treatment has been to shed light on transitions from putatively lesser to more severe forms of harmful substance use. As mentioned, DSM-IV nomenclature and previous transition studies have assumed that abuse represents a milder form of SUD than dependence and that persons progress from abuse to dependence. Some evidence is consistent with these assumptions (e.g., greater disability and treatment seeking are associated with dependence compared to abuse).³ Other evidence suggests that many individuals qualify for dependence without first qualifying for abuse, indicating that *individuals* frequently do not experience the presumed developmental progression.¹² Perhaps the most important implication of the ubiquitous evidence of the substantial heterogeneity among persons with SUD is that a one-size-fits-all approach to epitomizing etiology or intervention for SUD (and SUD itself) is as insufficient as aspirin would be for treating all forms of pain.

In light of such heterogeneity, one useful research strategy is to investigate a particular subtype (cf. progress made over the last 20 years in research on cancer etiology and treatment). Flórez-Salamanca and colleagues' study focuses on one SUD subtype, persons who qualify for abuse before dependence. Even so, they found large heterogeneity in their participants: 9–27% of persons with abuse later experienced dependence (varying between drugs), some predictors of the transition were common among drugs whereas other predictors were not, and even the predictors consisted of broad categories (e.g., personality disorder) rather than specific factors (e.g., narcissism). This result suggests that even more specific subpopulations may need to be studied based on categories of the predictors of transition reported by Flórez-Salamanca, et al. To illustrate, never-married male alcohol users who qualify for bipolar diagnosis and began substance use after age 14 probably differ in important ways in their SUD development compared to other males who lack bipolar disorder and initiate substance use before age 14.

Within the context of the afore mentioned intersecting issues (to reduce SUD prevalence and costs, characterize SUD, and account for large heterogeneity, perhaps, in terms of severity levels or subtypes), Flórez-Salamanca et al.s' study presents important results regarding how SUD transitions are relevant to diagnosis and treatment. The study itself represents an important contribution to this literature:(1) results generalize to persons of the U.S. who qualify for DSM-IV abuse, (2) data were collected with a widely-used instrument with well-documented psychometric properties, and (3) rigorous analyses were conducted. Several past findings were replicated in the study, thereby documenting their robustness, including rates of transition from abuse to dependence for different drugs, between-drug differences in transition speed, and sociodemographic correlates of transition.

Flórez-Salamanca et al.s' study extends the research literature by demonstrating that psychiatric disorders increase the probability of experiencing abuse as well as subsequent dependence, which few epidemiologic studies have addressed.¹³ The finding that important role(s) are played by psychiatric illness in SUD transitions may be the study's most direct implication for treatment. To illustrate, the negative impact on SUD treatment outcomes associated with co-occurring or prior psychiatric disorders is frequently greater than the difference in treatment outcomes between abuse vs. dependence.^{14–15} High rates of comorbidity of SUD with other psychiatric disorders, certain theoretical models of SUD

etiology (e.g., self-medication model), and efficacy research on SUD outcomes among persons with other comorbid psychiatric disorders provide triangulated evidence that psychopathology can worsen SUD. Collectively, this body of evidence underscores the need to better integrate SUD treatment within medical, and especially mental health, systems.¹⁶

These results additionally illustrate an important, gradual evolution in research on SUD transitions: to determine how knowledge of SUD transitions can be used to improve treatment efficacy.¹⁷ Such research should identify those aspects of heterogeneity in SUD transitions which are most relevant to improving outcomes. Examples of the research questions that are specifically raised by Flórez-Salamanca et al.s' study follow. Within DSM-IV nomenclature, do patients with only abuse differ qualitatively from those with only dependence (or from those with both)? Are transitions associated with treatment outcomes (e.g., perhaps slowing transitions ought to be an important clinical goal)? Does the speed of a transition reflect changes in underlying bio psychological processes of addiction and/or recovery?¹⁸

The answers to these questions can inform both SUD treatment and etiology. For instance, determining how transitions are (or are not) associated with development and treatment of SUD could identify (or rule out) a way to gauge SUD severity, thus informing diagnostic nomenclature, clinical outcomes and intervention strategy. Fortunately, extant datasets including the National Epidemiological Survey on Alcohol and Related Conditions(NESARC)already contain the data needed to test these types of research questions. Perhaps the greatest strength of the DSM nomenclature is its provision of a common metric that is used in a breadth of research. This common metric, coupled with recent methodologies to combine datasets,¹⁹ provides a low-cost approach to obtaining large samples of subtypes of persons with SUD (e.g., those with a particular subset of diagnostic symptoms/criteria). In this manner, the aforementioned research questions could be addressed for specific SUD subtypes using combinations of existing samples, scientific paradigms, research designs and clinical trials (which also are etiology experiments). BecauseDSM-5 SUD nomenclature largely consists of DSM-IV criteria (with certain exceptions, such as the addition of a "craving" symptom/criterion), numerous SUD researchers already possess high quality data to investigate DSM-5-based SUD transitions.

Flórez-Salamanca et al.s' study, therefore, highlights the need to not conceptualize SUD as an endpoint, but rather a milestone of pathological human development. Once an SUD is initiated, it frequently becomes a chronic, albeit dynamic, condition. SUD, as well as all other risky or problem behaviors, inherently reflects a complex array of hetero genetic equifinalities and multifinalities. Thus, scientific understanding of, and impactful intervention against, harmful compulsive substance use will likely require sophisticated ontogenetic (within-person developmental) models²⁰ as well as greater understanding of the mechanisms which bias development toward and away from SUD outcomes (not only identifying what the mechanisms are).²¹

Toward this end, the science of SUD etiology is also gradually reaching an impasse with regard to how well large-sample-oriented methods can address what are inherently idiographic (within-person, ontogenetic) research questions. Past efforts to understand

individual differences within the context of SUD for the purpose of matching individuals to treatments have often led to disappointing results.²² However, the delimiting factors in previous studies may well be not due to the theories or hypotheses driving the research. Rather, the disappointing results may be due to reliance upon nomothetic (sample-level) methodologies, which are designed to aggregate individual differences into population-level estimates. The time has come to supplement SUD transition research by employing extant, rigorous idiographic methodologies to intensively examine the developmental *processes* involved in etiology and treatment.^{23–25} Fortunately, these methods are well-suited for clinical research and particularly for inpatient research with a patient-centered focus.^{24–25} In sum, Flórez-Salamanca et al.s' findings and the SUD transition science within which their study occurs exemplify the need to understand SUD as a developmental and idiographic phenomenon.

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