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Alcohol use disorder conceptualizations and diagnoses reflect their sociopolitical context

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Abstract

The present paper highlights how alcohol use disorder (AUD) conceptualizations and resulting diagnostic criteria have evolved over time in correspondence with interconnected sociopolitical influences in the United States. We highlight four illustrative examples of how DSM-defined alcoholism, abuse/dependence, and AUD have been influenced by sociopolitical factors. In doing so, we emphasize the importance of recognizing and understanding such sociopolitical factors in the application of AUD diagnoses. Last, we offer a roadmap to direct the process of future efforts toward the improved diagnosis of AUD, with an emphasis on pursuing falsifiability, acknowledging researchers' assumptions about human behavior, and collaborating across subfields. Such efforts that center the numerous mechanisms and functions of behavior, rather than signs or symptoms, have the potential to minimize sociopolitical influences in the development of diagnostic criteria and maximize the treatment utility of diagnoses.

Keywords

alcohol use disorder; diagnosis; sociopolitical influences

INTRODUCTION

Since appearing in the first Diagnostic and Statistical Manual of Mental Disorders (DSM; 1952) (American Psychiatric Association [APA] 1952) and the *International Classification of Diseases* (ICD; 1967), the criteria for what is now termed an alcohol use disorder (AUD) have evolved significantly. AUD nosology and classification have been jointly influenced by scientific progress in the understanding of AUD *and* sociopolitical influences and their context. In the DSM and ICD, diagnostic criteria are largely determined by expert consensus. Although decisions about diagnostic criteria, diagnostic thresholds, and related issues may be informed by empirical evidence and experts selected based on varying expertise, such decisions are nonetheless socially constructed and therefore still reflect, at least to some degree, the interests, beliefs, and biases of those individuals defining pathology (Lazaroff 2006; Nathan et al. 2016; Krueger et al. 2018). This leads to the potential for behaviors to be seen as maladaptive when they interfere with expectations and norms of the dominant culture. These behaviors are then assigned labels, such as AUD. As Jellinek said, “a disease is what the medical profession recognizes as such” (Jellinek 1960). Thus, AUD nosology may neglect the perspectives of those not represented as experts, including oppressed groups who hold less power (e.g., people with lived experience of AUD, people of color) and who have historically faced more barriers to higher education and scientific careers (Jordan and Jegede 2020; Stull et al. 2022). What might result from this process are diagnostic criteria that fail to generalize (Room 2006; Caetano 2011) and pathologize non-dominant groups of people (Lazaroff 2006).

In this “think piece,” we offer critical reflections on how sociopolitical processes influence AUD nosology, specifically DSM criteria, through the use of several illustrative examples. We focus on U.S. influences and the DSM, given the DSM remains the dominant classification system in the U.S. and will likely continue to evolve through expert consensus processes. We conclude by highlighting the need for future conceptualizations of alcohol-related problems to overtly consider the potential influence of sociopolitical processes through approaches that are more reflexive and center mechanisms and functions of behavior, rather than signs or symptoms. We recommend recognition that scientific and sociopolitical influences are inherently connected, as sociopolitical influences impact research questions and methods, policies, and treatment priorities. We advocate for a conceptualization of AUD that recognizes cultural and contextual factors to ultimately improve the treatment of AUD.

EXAMPLE 1: DSM-I (1952)

In the DSM-I (APA 1952) “alcoholism” fell under the broader classification of Sociopathic Personality Disorders. Per the DSM-I, “Individuals to be placed in this category are ill primarily in terms of society and of conformity with the prevailing cultural milieu, and

not only in terms of personal discomfort and relations with other individuals" (p. 38). Sociopathic reactions were thought to reflect "underlying personality disorder, neurosis, or psychosis, or occur as the result of organic brain injury or disease" (p. 38). The DSM-I did permit exceptions to the diagnosis of "alcoholism" in cases "in which there is well-established addiction to alcohol without recognizable underlying disorder" (p. 39; APA 1952). Thus, "alcoholism" was an individual illness of inability to drink in ways that adequately conformed to societal expectations (i.e., lack of self-control), and a disturbance of "normal" personality development (Levine 1978; Room 1985; Miller and Kurtz 1994; Valverde 1997; Robinson and Adinoff 2016).[1]

This conceptualization of "alcoholism" as drinking in a way that fails to conform to societal norms is consistent with the belief that excessive alcohol consumption is a sin, moral failing, or failure of willpower (concepts which are themselves socially constructed; Miller and Kurtz 1994; Valverde 1997). These beliefs can be traced back as far as biblical times (Proverbs 20:1; Luke 21:34,12:42; Galatians 5:21) and were a common perspective in the temperance movement. For example, temperance activists argued that *any* drinking was dangerous because people were incapable of drinking in moderation and avoiding drunkenness (Lender and Martin 1987) and that drinking was the root of social ills, particularly among lower social classes (Levine 1983). However, during prohibition, the public began to view alcohol consumption less as a societal problem and more as a circumscribed problem limited to a few with health problems and/or character defects (Miller and Kurtz 1994), such as lack of willpower. Such a conceptualization also aligns with the dispositional disease model of "alcoholism" as an individual "illness." These perspectives influenced—and were influenced by—the institutional "care" of "alcoholics" (Edman 2009) and the tenants of *Alcoholics Anonymous* (Alcoholics Anonymous 1939). Per this model, "alcoholism" is best treated by addressing the underlying problems with morality and increasing self-control (Room 1985). As an example of social influences on perceptions of AUD etiology, the alcohol industry embraced this conceptualization because it suggested one had to be predisposed to be harmed by alcohol use, so "alcoholism" was assumed to be confined to few individuals (see Heather 2013).

The DSM-I reflected this dispositional disease model of "alcoholism". Specifically, manifestations of psychopathology, including "alcoholism," were considered an abnormality within the individual, overlooking who is defining "normality" and failing to consider contributions of systemic factors and contexts that may influence alcohol consumption (Velásquez et al. 1993). Indeed, some authors suggest that the label of "alcoholic" was applied more to individuals from oppressed groups who were thought to lack self-control and willpower, including those of low socioeconomic status and women, when compared to men of upper and middle classes who were assumed to be born with more willpower (Valverde 1997).

[1]Not all explanations of addiction that center on addiction as a breakdown in self-regulation are moralizing. For example, behavioral economics is consistent with the idea of irrational or disordered choice (e.g., Rachlin et al. 1981) but fails to hold the same moralizing undertones that assume disordered choice is a product of sin or moral failing.

EXAMPLE 2: DSM-III (1980)

Several advances in psychiatric classification with a particular focus on the increased role of empiricism presaged DSM-III, likely undergirded partially by neo-Kraepelinian attempts to move away from psychological and social (including psychoanalytic) conceptualizations of psychiatric disorders and towards more empirical conceptualizations of psychopathology to gain legitimacy as a medical discipline (Compton and Guze 1995; Wakefield 2022). The National Council on Alcoholism (1972) offered criteria for "alcoholism" that focused mainly on the adverse consequences associated with drinking, alcoholism's progressive nature, its physical course, and its neuroadaptations (i.e., tolerance, withdrawal). This was consistent with the conceptualization of addiction as a disease process and the medicalization of addiction (Campbell 2011). After, Edwards and Gross (1976) put forth their model of the alcohol dependence syndrome, which included mostly behaviorally-based criteria to index dependence, including narrowing of the drinking repertoire, increased tolerance, withdrawal, and compulsion.

The alcohol dependence syndrome and neo-Kraepelinian perspectives on the biology of mental illness laid the foundation for DSM-III (APA 1980), which replaced the term "alcoholism" with abuse and dependence, alongside specific diagnostic criteria. Dependence was presumed to reflect a more severe physiological manifestation than abuse, which was related more to social and occupational problems in the absence of tolerance or withdrawal (Robinson and Adinoff 2016). For the first time, substance use disorders were separated into their own diagnostic category and no longer classified as a personality disorder. Still, the increasing influence of psychiatry and related neo-Kraepelinian medicalization of AUD came with different sociopolitical and economic influences including managed healthcare and pharmaceutical companies having high stakes in research outcomes, such as medication trials. Such research was likely a powerful motivator for psychiatrists to describe AUD as a medical disease because psychiatrists rely primarily on medications in their treatment approaches (Lazaroff 2006). As stated by Zur and Nordmarken (n.d.), "Without medications, psychiatrists stand to lose their place in the treatment hierarchy, and the DSM would lose its legitimacy as a necessary biological-medical tool" (p. 4). The neo-Kraepelinian, medical model has continued to influence DSM diagnostic categories to date, likely due, in part, to the continued influence of pharmaceutical companies on treatment trials for many psychiatric disorders (Sedler 2016).

EXAMPLE 3: DSM-IV (1994)

In response to the limitations of previous AUD criteria, namely the limited treatment utility (Kawa and Giordano 2012) and poor reliability and validity (Millon and Klerman 1986) of DSM-III, the Institute of Medicine of the National Academy of Sciences (1990) proposed a continuum-based model that emphasized the severity of consumption, dependence, alcohol-related problems (including health problems), and the need for improved prevention and intervention. This model was influenced by an emerging public health perspective that was eager to replace the neo-Kraepelinian dispositional disease model with a conceptualization of AUD as the combination of numerous factors, including intrapersonal (e.g., susceptibility to drinking) and environmental (e.g., taxation, supply) factors (Miller 1993; Roizen). DSM-

IV's (American Psychiatric Association 1994) criteria were revised with the public health perspective in mind, and were based on a comprehensive re-analysis of epidemiological data, collection of new field data, and empirical literature reviews. Changes to the DSM-IV reflect the public health perspective insofar as abuse criteria were broadened to capture consequences more aligned with a continuum of problems, adding criteria on role interference (e.g., work/school), as well as intrapersonal, interpersonal, and legal problems. The consumption continuum was also emphasized to a greater degree, as abuse was thought to reflect less severe "problematic" use consistent with the lower end of the AUD severity continuum and alcohol dependence was thought to reflect compulsive use and the more severe end of the AUD continuum. Although this conceptualization of AUD was more empirically based than previous conceptualizations, the DSM-IV did not entirely move away from its neo-Kraepelinian roots, given a continued focus on descriptive signs and symptoms rather than etiological and sociocontextual processes (Compton and Guze 1995).

Further, to address growing concerns about the lack of consideration of diversity and culture, the DSM-IV Task Force consulted with the NIMH Culture and Diagnosis Work Group, who sought to contextualize human suffering within cultural and sociopolitical situations and suggested framing the DSM as a cultural document influenced by certain implicit values and perspectives (Kirmayer 1998). These groups had a clash between their somewhat incompatible goals. The resulting product was a compromise that was frustrating for both groups (Kirmayer 1998). The DSM-IV Task Force framed the diagnostic criteria as "universal," "atheoretical," and "culture-free," and because of this, chose to exclude any suggested wording that challenged that view. The Cultural Formulation and Glossary of Culture-Bound Syndromes developed by the NIMH Culture and Diagnosis Work Group was placed in the DSM-IV Appendix (Mezzich et al. 1999). Ultimately, the AUD description and criteria included limited information about cultural considerations, including population statistics and medical differences (e.g., differential alcohol metabolism) (APA 1994). Although this example is not specific to AUD, it illustrates the sociopolitical processes at play in developing psychiatric diagnoses and their criteria, and a lack of explicit examination of and acknowledgement regarding who is determining something "culture-free" in the determination of DSM diagnostic criteria.

EXAMPLE 4: DSM-5 (2013)

The DSM-5 Task Force envisioned the new edition as representing a paradigm shift from the descriptive level of diagnosis seen in the previous editions to using biologically-based etiology to define diagnostic categories (Zachar et al. 2019). However, other experts suggested the biological etiology of psychiatric disorders was in its infancy and basing diagnostic criteria on it was premature (Frances 2009). The DSM-5 ultimately retained its descriptive diagnoses (although many of the retained criteria were historically included to capture AUD as a "mental disease" and thus still have a biological basis) (Zachar et al. 2019), collapsing the abuse and dependence categories and, in turn, conceptualizing AUD as a unitary dimension composed of 11 criteria that span impaired control, social impairment, risky use, and pharmacological criteria along a severity continuum (APA 2013). DSM-5 dropped the legal problems criterion, given low prevalence, poor fit with other criteria, low discrimination, and differential item functioning (Hasin et al. 2013). Dropping the legal

problems criterion also addressed concerns regarding racial and ethnic inequities, given that American Indian/Alaska Native and Black people are more likely than White people to experience alcohol and drug related arrests that were not explained by differences in consumption (Camplain et al. 2020). DSM-5 also added a craving criterion given evidence for craving as a treatment target across substances as well as overlap with ICD alcohol dependence criteria (Hasin et al. 2013).

Despite an incomplete shift to biologically-based etiology to define diagnostic categories, the influence of the Brain Disease Model of Addiction (BDM; Koob and Moal 1997; Leshner 1997; Koob and Volkow 2010; Volkow et al. 2016) is reflected in DSM-5 AUD diagnostic criteria. The BDM claims that addiction is a chronic, relapsing brain disease that is characterized by compulsive substance seeking and use despite negative consequences. Accordingly, changes to the DSM-5, such as the addition of the craving criterion, as well as criteria added in prior versions of the DSM, such as compulsive use (loss of control) and drinking despite physical and psychological consequences of alcohol use reflect the BDM. The BDM is not a new influence on conceptualizations of alcohol-related problems, but it has been increasingly endorsed by organizations with significant power, including the National Institute on Alcohol Abuse and Alcoholism and the National Institute on Drug Abuse since publication of DSM-IV. Various neurobiological models of addiction argue that the transition from substance use to addiction is determined by structural and functional changes in the brain with repeated use (Koob and Moal 1997; Koob and Volkow 2010). Yet, chronic addiction is the exception rather than the rule: most people with a substance use disorder diagnosis recover, and most recover without treatment (Tucker et al. 2020). Thus, a chronic, relapsing form of AUD may represent an important, severe phenotype, but it does not necessarily characterize most of the population that experiences difficulties with alcohol and does not acknowledge that systemic factors influence AUD (Pickard 2022a). Still, the BDM has influenced the DSM-5 AUD diagnosis, likely due to sociopolitical and economic factors (e.g., providing more research funding to explore addiction as a brain disease which then informs iterations in future DSM criteria).

DISCUSSION AND CONCLUSIONS

Public and scientific perceptions of alcohol use and related problems – and, accordingly, diagnostic criteria for AUD – have evolved substantially over time. Conceptualizations of AUD are socially constructed insofar as they are directly influenced by prevailing discourse regarding psychological dysfunction among scientists and the broader public and the positionality of those who develop DSM criteria. Conceptualizations are also influenced indirectly given sociopolitical pressures on research, treatment, and policy priorities. Sociopolitical influences on AUD conceptualizations may limit the utility of diagnostic categories for identification and treatment planning, which may be particularly problematic when such influences go unacknowledged. For example, if severe manifestations of AUD such as compulsion predominate the conceptualization of AUD and resulting diagnostic criteria, people with less severe manifestations of AUD that may still benefit from treatment could go overlooked and not be served by treatments that are designed to treat more severe manifestations.

In future DSM Work Groups, we call for increased reflexivity, at a minimum. That is, we encourage the work group to reflect on their positionality, including their beliefs about what causes and maintains addiction and how that may be influenced by their individual identities, such as their racial identity, gender, or socioeconomic status, as well as other factors, such as beliefs about addiction and potential conflicts of interest that may influence such thinking. Increased reflexivity may help bring transparency to the Work Group process and allow more explicit consideration of the sociopolitical factors at play in the construction of the diagnostic criteria. This is important because it may be impossible to fully reduce or eliminate sociopolitical influences on our nosologic frameworks. Relatedly, we call for those applying AUD criteria to consider how the intersectional identities of individuals with lived experience of AUD may be similar or different from those who developed the AUD criteria. Through increased awareness and explicit consideration of these factors, AUD diagnoses may become more useful to a broader population of individuals with AUD, both in their construction and application.

Additionally, a greater focus on dimensional etiological mechanisms and the function of drinking behavior (e.g., availability of alternative, non-alcohol reinforcers) in AUD diagnosis has the potential to minimize sociopolitical influences in the development of diagnostic criteria. For example, moving away from reductionistic conceptualizations of AUD may reduce the pressure for experts to decide on the “correct” conceptualization of what causes and maintains AUD and instead offer a more holistic profile of an individual’s functioning. That is not to say that a mechanistic focus will free AUD conceptualizations of sociopolitical influence, but ensuring a consideration of a broader range of explanations for AUD (e.g., neurobiological, social, psychological, environmental) and not just those considered to be associated with the “brain disease” of addiction, may more precisely identify alcohol-related problems (Boness et al. 2021) and personalized, multi-faceted treatment targets beyond pharmacological approaches. Of note, several dimensional frameworks for classifying alcohol-related problems that prioritize etiological mechanisms have recently been proposed, including the Alcohol Addiction Research Domain Criteria (Litten et al., 2015) and associated Addictions Neuroclinical Assessment (Kwako et al., 2016), the NIDA Phenotyping Battery (NIDA PhAB; Keyser-Marcus et al., 2021; Watts et al., 2022), and the Etiologic, Theory-based, Ontogenetic Hierarchical Framework (ETOH Framework; Boness et al., 2021) might hold promise for informing future classification of AUD based on mechanisms and functions of behavior. As an alternative to the DSM process, some experts have suggested a complete jettison of diagnosis in favor of an approach focused on heavy drinking over time (e.g., Rehm 2016), but this many introduce new challenges such as the pathologizing of consumption itself.

To increase the utility of future AUD conceptualizations, regardless of specific approach, we advocate for a transparent and inclusive process that includes a willingness to pursue falsifiable hypotheses and respond accordingly as well as decreased allegiance to unitary or reductionistic models of addiction. Without such guiding values in place, mechanistic conceptualizations of AUD are at risk of being prone to the same criticisms as the BDM, including limited empirical support, inadequate attention to alternative scientific accounts of addiction, and the potential for increased stigmatization and pessimism toward those with

AUD (e.g., Heather 2013; Heather 2017; Satel and Lilienfeld 2017; Heather et al. 2018; Pickard 2022).

Considering contextual factors in parallel to mechanisms may also encourage more explicit treatment of AUD as a "culturally bound syndrome" influenced by sociopolitical factors (Room 1985), at least in part. In this way, AUD criteria could be more aligned with etiologic and maintenance processes in AUD, and contextual factors may serve as important clinical indicators for choosing the best treatment. For example, the DSM-5 Cultural Formulation Interview is intended to provide information about the impact of culture on an individual's clinical presentation (e.g., cultural definition of the problem and its cause) and treatment (e.g., cultural impacts on current treatment expectations). This could be used to better understand how the etiologic and maintenance processes for a given person with AUD are influenced by their cultural values. Such changes in AUD diagnosis might help connect individuals to treatment or prevention programming that matches their unique biological, psychological, environmental, cultural characteristics, and sociocultural contexts. This procedure is consistent with precision medicine efforts which, to date, have not used DSM or ICD diagnostic criteria of AUD as specific targets, and have instead more often focused on treatment responses based on psychological characteristics (e.g., incentive salience/reward drinking; Mann et al. 2018; Witkiewitz et al. 2019) and biological characteristics (e.g., specific genotypes or brain activation patterns; Gelernter et al. 2007; Kranzler et al. 2014; Schacht et al. 2017). Nonetheless, future work is needed to elucidate how to best incorporate cultural and environmental factors into idiographic assessment and treatment planning, given prior efforts have focused on psychological and biological characteristics.

We recognize that the scientific process cannot be disentangled from broad sociopolitical influences as well as interpersonal dynamics of scientific task forces, given funding priorities and the influence of a researcher's identity on the questions they ask and methods they use. As such, we acknowledge that the backgrounds of our authorship team (who were trained in social work, psychology, and psychiatry, and who each have varied clinical and personal lived experience with addiction), and the sociopolitical factors at play in our own lives, may influence the perspectives we describe here. Future progress will require scientists to pursue falsifiability in our research, acknowledge our assumptions about human behavior, and collaborate across subfields to capture the numerous influences on AUD etiology, development, and maintenance. It will also require engaging community partners (e.g., "frontline" treatment providers, policy makers, payors) and people with lived experience to incorporate their invaluable expertise on their own experiences alongside empirical research, moving towards shared power and equitable diagnostic classification. A focus on broad etiologic mechanisms, empirical classification, and contextual factors is needed to ensure continued progress towards addressing the significant public health impacts of AUD.

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REFERENCES

Alcoholics Anonymous. 1939. Alcoholics Anonymous: the story of how more than one hundred men have recovered from alcoholism. New York, NY: Alcoholics Anonymous World Services.

American Psychiatric Association. 1952. Mental disorders: Diagnostic and statistical manual. Mental Hospital Service.

American Psychiatric Association. 1980. Diagnostic and statistical manual of mental disorders. 3rd ed. Washington D.C.

American Psychiatric Association. 1994. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association.

American Psychiatric Association. 2013. Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Association.

Boness CL, Watts AL, Moeller KN, Sher KJ. 2021. The Etiologic, Theory-Based, Ontogenetic Hierarchical Framework of Alcohol Use Disorder: A Translational Systematic Review of Reviews. Psychol Bull. doi:10.31219/osf.io/bscuh.

Caetano R 2011. THERE IS POTENTIAL FOR CULTURAL AND SOCIAL BIAS IN DSM-V: Commentaries. Addiction. 106(5):885–887. doi:10.1111/j.1360-0443.2010.03308.x.

Campbell. 2011. The Metapharmacology of the “Addicted Brain.” Hist Present. 1(2):194. doi:10.5406/historypresent.1.2.0194.

Camplain R, Camplain C, Trotter Ii RT, Pro G, Sabo S, Eaves E, Peoples M, Baldwin JA. 2020. Racial/Ethnic Differences in Drug-and Alcohol-Related Arrest Outcomes in a Southwest County From 2009 to 2018. Am J Public Health. 110:S85–S92. doi:10.2105/AJPH.2019.305409.

Compton WM, Guze SB. 1995. The neo-Kraepelinian revolution in psychiatric diagnosis. Eur Arch Psychiatry Clin Neurosci. 245(4–5):196–201. doi:10.1007/BF02191797.

Edman J 2009. What’s in a Name?: Alcohol and Drug Treatment and the Politics of Confusion. Nord Stud Alcohol Drugs. 26(4):339–353. doi:10.1177/145507250902600402.

Edwards G 1986. The Alcohol Dependence Syndrome: a concept as stimulus to enquiry. Addiction. 81(2):171–183. doi:10.1111/j.1360-0443.1986.tb00313.x.

Edwards G, Gross MM. 1976. Alcohol dependence: provisional description of a clinical syndrome. BMJ. 1(6017):1058–1061. doi:10.1136/bmj.1.6017.1058.

Frances AJ. 2009. A warning sign on the road to DSM-V: Beware of its unintended consequences. Psychiatr Times. 26(8):1, 4–5, 8–9.

Gelernter J, Gueorguieva R, Kranzler HR, Zhang H, Cramer J, Rosenheck R, Krystal JH. 2007. Opioid Receptor Gene (OPRM1, OPRK1, and OPRD1) Variants and Response to Naltrexone Treatment for Alcohol Dependence: Results From the VA Cooperative Study. Alcohol Clin Exp Res. 31(4):555–563. doi:10.1111/j.1530-0277.2007.00339.x.

Hanson DJ. 1995. Preventing Alcohol Abuse: Alcohol, Culture, and Control. Greenwood Publishing Group.

Hasin DS, O’Brien CP, Auriacombe M, Borges G, Bucholz K, Budney A, Compton WM, Crowley T, Ling W, Petry NM, et al. 2013. DSM-5 criteria for substance use disorders: Recommendations and rationale. Am J Psychiatry. 170(8):834–851. doi:10.1176/appi.ajp.2013.12060782.

Heather N 2013. Is Alcohol Addiction Usefully Called a Disease? Philos Psychiatry Psychol. 20(4):321–324. doi:10.1353/ppp.2013.0050.

Heather N 2017. Q: Is Addiction a Brain Disease or a Moral Failing? A: Neither. Neuroethics. 10(1):115–124. doi:10.1007/s12152-016-9289-0.

Heather N, Best D, Kawalek A, Field M, Lewis M, Rotgers F, Wiers RW, Heim D. 2018. Challenging the brain disease model of addiction: European launch of the addiction theory network. Addict Res Theory. 26(4):249–255. doi:10.1080/16066359.2017.1399659.

Institute of Medicine. 1990. National Priorities for the Assessment of Clinical Conditions and Medical Technologies: Report of a Pilot Study. Washington, DC: The National Academies Press. https://www.nap.edu/catalog/1529/national-priorities-for-the-assessment-of-clinical-conditions-and-medical-technologies.

Jellinek EM. 1960. The disease concept of alcoholism. New Haven: Hillhouse Press. [accessed 2021 May 5]. http://content.apa.org/books/14090-000.

Jordan A, Jegede O. 2020. Building Outreach and Diversity in the Field of Addictions: Building Outreach and Diversity. Am J Addict. 29(5):413–417. doi:10.1111/ajad.13097.

Kawa S, Giordano J. 2012. A brief historicity of the Diagnostic and Statistical Manual of Mental Disorders: Issues and implications for the future of psychiatric canon and practice. Philos Ethics Humanit Med. 7(1):2. doi:10.1186/1747-5341-7-2.

Kirmayer LJ. 1998. The Fate of Culture in DSM-IV. Transcult Psychiatry. 35(3):339–342. doi:10.1177/136346159803500301.

Koob GF, Moal ML. 1997. Drug Abuse: Hedonic Homeostatic Dysregulation. Science. 278(5335):52–58. doi:10.1126/science.278.5335.52.

Koob GF, Volkow ND. 2010. Neurocircuitry of Addiction. Neuropsychopharmacology. 35(1):217–238. doi:10.1038/npp.2009.110.

Kranzler HR, Covault J, Feinn R, Armeli S, Tennen H, Arias AJ, Gelernter J, Pond T, Oncken C, Kampman KM. 2014. Topiramate treatment for heavy drinkers: moderation by a GRIK1 polymorphism. Am J Psychiatry. 171(4):445–52. doi:10.1176/appi.ajp.2013.13081014.

Krueger RF, Kotov R, Watson D, Forbes MK, Eaton NR, Ruggero CJ, Simms LJ, Widiger TA, Achenbach TM, Bach B, et al. 2018. Progress in achieving quantitative classification of psychopathology. World Psychiatry. 17(3):282–293. doi:10.1002/wps.20566.

Lazaroff AM. 2006. The Role of the Diagnostic and Statistical Manual of Mental Disorders in the Maintenance of the Subjugation of Women: Implications for the Training of Future Mental Health Professionals.

Lender ME, Martin JK. 1987. Drinking In America: A History. New York, NY: The Free Press.

Leshner AI. 1997. Addiction Is a Brain Disease, and It Matters. Science. 278(5335):45–47. doi:10.1126/science.278.5335.45.

Levine H 1983. The Good Creature of God and the demon rum: Colonial American and 19th century ideas about alcohol, crime and accidents. In: Alcohol and Disinhibition: Nature and Meaning of the Link. Washington, D.C: U.S.G.P.O. (NIAAA Monograph 12).

Levine HG. 1978. THE DISCOVERY OF ADDICTION. J Stud Alcohol.:143–174.

Mann K, Roos CR, Hoffmann S, Nakovics H, Leménager T, Heinz A, Witkiewitz K. 2018. Precision medicine in alcohol dependence: A controlled trial testing pharmacotherapy response among reward and relief drinking phenotypes. Neuropsychopharmacology. 43(4):891–899. doi:10.1038/npp.2017.282.

Mezzich JE, Kirmayer LJ, Kleinman A, Fabrega HJ, Parron DL, Good BJ, Lin K-M, Manson SM. 1999. The Place of Culture in DSM-IV. J Nerv Ment Dis. 187(8):457–464.

Miller WR. 1993. Alcoholism: Toward a Better Disease Model. Psychol Addict Behav. 7(2):129–136.

Miller WR, Kurtz E. 1994. Models of alcoholism used in treatment: contrasting AA and other perspectives with which it is often confused. J Stud Alcohol. 55(2):159–166. doi:10.15288/jsa.1994.55.159.

Millon T, Klerman GL, editors. 1986. Contemporary directions in psychopathology: toward the DSM-IV. New York: Guilford Press.

Nathan PE, Conrad M, Skinstad AH. 2016. History of the Concept of Addiction. Annu Rev Clin Psychol. 12(1):29–51. doi:10.1146/annurev-clinpsy-021815-093546.

National Council on Alcoholism. 1972. Criteria for the Diagnosis of Alcoholism. Am J Psychiatry. 129(2):127–135. doi:10.1176/ajp.129.2.127.

Pickard H 2022a. Is addiction a brain disease? A plea for agnosticism and heterogeneity. Psychopharmacology (Berl). 239(4):993–1007. doi:10.1007/S00213-021-06013-4/FIGURES/4.

Pickard H 2022b. Is addiction a brain disease? A plea for agnosticism and heterogeneity. Psychopharmacology (Berl). 239(4):993–1007. doi:10.1007/s00213-021-06013-4.

Rachlin H, Battalio R, Kagel J, Green L. 1981. Maximization theory in behavioral psychology. Behav Brain Sci. 4(3):371–388. doi:10.1017/S0140525X00009407.

Rehm J 2016. How should prevalence of alcohol use disorders be assessed globally? Alcohol Use Disord Mortal Syst Rev Meta-Anal. 25(2). doi:10.1002/mpr.1508.

Robinson S, Adinoff B. 2016. The Classification of Substance Use Disorders: Historical, Contextual, and Conceptual Considerations. Behav Sci. 6(3):18. doi:10.3390/bs6030018.

Roizen How Does the Nation's "Alcohol Problem" Change from Era to Era? [accessed 2022 Aug 12]. http://www.roizen.com/ron/postrepeal.htm.

Room R 1985. Dependence and society. Br J Addict. 80(2):133–139. doi:10.1111/j.1360-0443.1985.tb03263.x.

Room R 2006. Taking account of cultural and societal influences on substance use diagnoses and criteria. Addiction. 101:31–39. doi:10.1111/j.1360-0443.2006.01597.x.

Satel SL, Lilienfeld SO. 2017. If Addiction is not Best Conceptualized a Brain Disease, then What Kind of Disease is it? Neuroethics. 10(1):19–24. doi:10.1007/s12152-016-9287-2.

Schacht JP, Randall PK, Latham PK, Voronin KE, Book SW, Myrick H, Anton RF. 2017. Predictors of naltrexone response in a randomized trial: Reward-related brain activation, OPRM1 genotype, and smoking status. Neuropsychopharmacology. 42(13):2640–2653. doi:10.1038/npp.2017.74.

Sedler MJ. 2016. Medicalization in psychiatry: the medical model, descriptive diagnosis, and lost knowledge. Med Health Care Philos. 19(2):247–252. doi:10.1007/s11019-015-9670-5.

Stull SW, Smith KE, Vest NA, Effinger DP, Epstein DH. 2022. Potential Value of the Insights and Lived Experiences of Addiction Researchers With Addiction. J Addict Med. 16(2):135–137. doi:10.1097/ADM.0000000000000867.

Tucker J, Chandler S, Witkiewitz K. 2020. Epidemiology of Recovery From Alcohol Use Disorder. Alcohol Res Curr Rev. 40(3):02. doi:10.35946/arcr.v40.3.02.

Valverde M 1997. 'Slavery from within': The invention of alcoholism and the question of free will*. Soc Hist. 22(3):251–268. doi:10.1080/03071029708568008.

Velásquez RJ, Johnson R, Brown-Cheatham M. 1993. Teaching Counselors to Use the DSM-III-R With Ethnic Minority Clients: A Paradigm. Couns Educ Superv. 32(4):323–331. doi:10.1002/j.1556-6978.1993.tb00259.x.

Volkow ND, Koob GF, McLellan AT. 2016. Neurobiologic Advances from the Brain Disease Model of Addiction. Longo DL, editor. N Engl J Med. 374(4):363–371. doi:10.1056/NEJMra1511480.

Wakefield JC. 2022. Klerman's "credo" reconsidered: neo-Kraepelinianism, Spitzer's views, and what we can learn from the past. World Psychiatry. 21(1):4–25. doi:10.1002/wps.20942.

Witkiewitz K, Roos CR, Mann K, Kranzler HR. 2019. Advancing precision medicine for alcohol use disorder: Replication and extension of reward drinking as a predictor of naltrexone response. Alcohol Clin Exp Res. 43(11):2395–2405. doi:10.1111/acer.14183.

Zachar P, Regier DA, Kendler KS. 2019. The Aspirations for a Paradigm Shift in DSM-5: An Oral History. J Nerv Ment Dis. 207(9):778–784. doi:10.1097/NMD.0000000000001063.

Zur O, Nordmarken N. DSM: Diagnosing for Status and Money, Summary of the Critique of the DSM. https://www.zurinstitute.com/resources/dsm-critique/.