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Characterizing the Patterns, Predictors, and Processes Involved in Recovery from Substance Use Disorders

Rebecca L. Smith
Virginia Commonwealth University

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By

REBECCA L. SMITH
B.S., University of Mary Washington, May 2013
M.S.W., Virginia Commonwealth University, May 2015
M.S., Virginia Commonwealth University, December 2019

Co-Directors: Danielle Dick, Ph.D.
Director
Rutgers Addiction Research Center
Rutgers University

Wendy Kliewer, Ph.D.
Professor
Department of Psychology
Virginia Commonwealth University

Virginia Commonwealth University
Richmond, Virginia
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Alcohol use disorder: Alcohol use disorder (AUD) is a medical condition diagnosed in accordance with the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association [APA], 2013) and is characterized by an impaired ability to stop or control alcohol use despite aversive consequences.

Genome-wide association study: Genome-wide association studies (GWAS) are an approach to investigate the association between specific genetic variants across the genome with particular phenotypes of interest.

Polygenic risk score: Polygenic risk scores (PRS) are one way to investigate the influence of genetic liabilities on complex phenotypes by using additive genetic effect of allele variants across the genome on an outcome of interest (Bogdan et al., 2018; Choi et al., 2020; Wray et al., 2014).

Recovery Capital: Recovery capital is a continuum of resources that individuals can use to initiate and sustain remission and prevent relapse (Granfield & Cloud, 1999).

Recovery Science: "Recovery science [should be an] independent field of inquiry to create a scientific specialization focused upon the causes, interventions, and practices that initiate and foster lifelong wellness" (Brown & Ashford, 2019, p. 3).

Relapse: Relapse is a return to alcohol or substance use after a period of abstinence.

Remission: Remission refers to situations when an individual who previously met diagnostic criteria for AUD and/or SUD no longer meets criteria within the past year (APA, 2013; Kelly & Hoeppner, 2015). Individuals can be in full remission (no longer meeting diagnostic criteria) or partial remission (still meeting diagnostic criteria but at a lower level of problem severity; APA, 2013).

Substance use disorder: Substance use disorders (SUDs) are medical conditions diagnosed in accordance with the DSM-5 (APA, 2013) and are characterized by an impaired ability to stop or control alcohol or drug use despite aversive consequences.

List of Frequently Used Acronyms

AUD: Alcohol use disorder

COGA: Collaborative Study on the Genetics of Alcoholism

GWAS: Genome-wide association study

PRS: Polygenic risk score

SSAGA: Semi-Structured Assessment for the Genetics of Alcoholism

SUD: Substance use disorder

Abstract

CHARACTERIZING THE PATTERNS, PREDICTORS, AND PROCESSES OF RECOVERY FROM SUBSTANCE USE DISORDERS

Rebecca L. Smith, M.S.W., M.S.

A dissertation submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University

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Co-Directors: Danielle Dick, Ph.D. & Wendy Kliewer, Ph.D.

Alcohol and drug use disorders are associated with significant cost to individuals, families, and society. Approximately 40-75% of affected individuals remit from alcohol use disorders (AUD). Although the *development* of AUD is well studied, *recovery* from AUD and factors that contribute to recovery are not as well characterized. With the definition of recovery evolving towards a process rather than an outcome, there is a need to better understand psychosocial functioning and quality of life among individuals at different points in their recovery journeys. Concurrently, additional research is needed to understand the interplay between sources of recovery capital, individual differences in risk factors for substance use, genetic influences, and psychosocial functioning and quality of life. To address that gap, we used a subsample of young adults who met criteria for AUD in their lifetime but later remitted (N = 323), derived from the Collaborative Study on the Genetics of Alcoholism (COGA) Prospective Study, to investigate profiles of functioning related to quality of life at first remitted assessment. We also examined recovery capital, epidemiological risk factors for substance use, and genome-wide polygenic risk scores as predictors of these profiles. Results suggested that a two-profile solution fit the data best, with 40% of participants categorized into the "infrequent alcohol use,

good health" profile and 60% into the "frequent alcohol use, good to very good health" profile. These findings indicate heterogeneity in functioning related to quality of life among those in AUD remission. Importantly, however, individuals in the "frequent alcohol use, good to very good" profile relative to individuals in the "infrequent alcohol use, good health" profile may represent those individuals who matured out of AUD. Social support for recovery, lifetime maximum depressive and post-traumatic stress disorder symptoms, and lifetime exposure to sexual assaultive trauma were higher in the "infrequent alcohol use, good health" profile than in the "frequent alcohol use, good to very good health" profile, while affiliation with deviant peers during adolescence and sensation seeking was higher in the latter profile. When examining the joint influence of all predictors on assigned profile membership, none had a unique effect. None of our polygenic risk scores were associated with assigned profile membership. Our findings add to the body of literature suggesting that heterogeneity in patterns of quality of life exists among those in AUD remission. Although none of our predictors had a unique effect on assigned profile membership when modeled simultaneously, findings suggest that mechanisms that underlie the development of AUD, such as comorbid internalizing symptoms and externalizing behaviors, may be important to consider with respect to quality of life in AUD remission. Moreover, findings from the present study can inform our understanding of recovery processes. The present findings underscore the importance of measuring individuals' self-reported recovery status and/or recovery identity so as not to conflate recovery processes with maturing out of AUD.

Characterizing the Patterns, Predictors, and Processes of Recovery from Substance Use Disorders

Statement of the Problem

Alcohol and drug use disorders are associated with significant cost to individuals, families, and society (Grant et al., 2015; Hasin et al., 2007; Rehm et al., 2009). Alcohol and drug use disorders (hereafter referred to as substance use disorders [SUDs]) represent a leading cause of disability worldwide (Grant et al., 2015; Griswold et al., 2018; Peacock et al., 2018; Rehm et al., 2009). SUDs are associated with elevated levels of health problems, interpersonal violence, and suicide (Rehm et al., 2009), with alcohol involved in 3.6% and illicit drugs involved in 0.8% of deaths worldwide (Peacock et al., 2018). Epidemiological studies in the United States suggest that 14% and 29% of individuals meet past-year and lifetime criteria for alcohol use disorders, respectively (Hasin et al., 2007), and 3.9% and 9.9% of individuals meet past-year and lifetime criteria for illicit substance use disorders (Substance Abuse and Mental Health Services Administration [SAMHSA], 2017). SUDs cost the United States more than $272 billion in 2017 (National Center for Drug Abuse Statistics, 2022). Although the *development* of SUDs is well studied, *recovery* from SUDs and factors that contribute to recovery are not as well characterized, thereby impeding treatment and underscoring the need to better understand the correlates of recovery. A better understanding of recovery processes can help reduce the pervasive individual, familial, and societal consequences of SUDs and improve treatment efforts.

Our understanding of recovery from SUDs remains limited, in part because of variable operational definitions of recovery (discussed below). Recovery has been reported in terms of remission from SUDs, resolution of an alcohol or drug problem, abstinence from alcohol and/or drugs, lack of high-risk or problematic substance use, and self-reported identification as being

"in recovery." This has contributed to difficulty in estimating prevalence rates of recovery from SUDs. For example, one nationally representative study found that approximately 1 in 10 US adults reported having "resolved" an alcohol or drug problem, of whom approximately 50% identified as being in recovery (Kelly et al., 2018; Kelly et al., 2017). According to a recent nationally representative study using data from the National Survey on Drug Use and Health (SAMHSA, 2020), approximately 12% of US adults perceived themselves as ever having a substance use problem, of whom 73% consider themselves recovered or in recovery. Together, despite variable definitions of recovery, results from extant studies suggest that many individuals recover from SUDs, while also underscoring the need for additional research on recovery processes using a standardized definition of recovery.

Review of Extant Literature on Recovery

Recovery Science

Recovery science is a burgeoning field of study that was previously encompassed by the field of addiction science (Brown & Ashford, 2019; McDaniel et al., 2020). However, recent schools of thought that the study of addiction provides only limited insight into recovery processes have propelled the development of this field. According to Brown and Ashford (2019), recovery science fundamentally differs from addiction science in that it is a strengths-based, process-oriented field of study. Recovery science emphasizes study of recovery-oriented systems of care, recovery capital, and social identity models of recovery, while addiction science typically focuses on the course of and cure for pathology and symptomatology (Brown & Ashford, 2019; McDaniel et al., 2020; Kelly et al., 2018). Recovery science aims to understand, measure, and validate recovery phenomena and trajectories in order to increase the efficacy of recovery-oriented systems of care. Recovery science focuses on understanding milestones,

turning points, and common pathways of different recovery trajectories (Brown & Ashford, 2019; McDaniel et al., 2020). Other defining features of recovery science include a focus on strengths-based orientations and the emphasis on subjective data, or the lived experiences of individuals in recovery, in addition to empirical data (Brown & Ashford, 2019; McDaniel et al., 2020). Put succinctly, "recovery science [should be an] independent field of inquiry to create a scientific specialization focused upon the causes, interventions, and practices that initiate and foster lifelong wellness" (Brown & Ashford, 2019, p. 3).

Defining Recovery

Compared to the study of SUDs and addiction, the field of recovery science is much less characterized. One barrier to advancements in this field is the evolving definition of recovery. Traditionally, recovery was synonymous with remission from an SUD (Kelly & Hoeppner, 2015). Remission refers to situations when an individual who previously met diagnostic criteria for an SUD no longer meets criteria within the past year (American Psychiatric Association [APA], 2013; Kelly & Hoeppner, 2015). Individuals can be in full remission (no longer meeting diagnostic criteria) or partial remission (still meeting diagnostic criteria but at a lower level of problem severity; APA, 2013). Under this traditional definition of recovery, abstinence from alcohol and drugs was considered the main mechanism by which individuals recovered. This emphasis on abstinence as a main mechanism of recovery was due in part to the focus placed on abstinence by multiple pathways to recovery (e.g., Alcoholics Anonymous), the decreased value placed on substances associated with sustained abstinence, and the documented reciprocal associations between abstinence and sustained remission and improvements in psychosocial functioning and health (Kaskutas et al., 2014; Kelly & Hoeppner, 2015; White, 2012; Witkiewitz, 2013; Witkiewitz et al., 2021). As such, abstinence often is the primary goal of

substance use treatment programs (White, 2012). More recently, recovery has been redefined to refer to sustained remission achieved through profound and personal lifestyle changes and experiences (Kelly & Hoeppner, 2015). Nevertheless, the terms "recovery" and "remission" are still frequently used interchangeably in extant literature, though operational definitions often vary by study (White, 2012).

In the past decade, there have been multiple attempts to refine the definition of recovery to incorporate the lived experiences of individuals in recovery (Kelly & Hoeppner, 2015). Attempts to refine and reframe recovery represent a shift from disease- and clinician-focused paradigms to one that is more person-centered, strength-based, and hopeful (Kelly et al., 2017; Kelly & Hoeppner, 2015). This paradigmatic shift involved a definition of recovery that was broader in scope than simply lack of use and associated consequences (i.e., abstinence and remission), though many working definitions still include components of abstinence (Kelly & Hoeppner, 2015). Further, this new paradigm incorporates psychosocial functioning and wellbeing, often describing recovery as a process, experience, state, or lifestyle (Kelly & Hoeppner, 2015).

Recent Refinements to Recovery Definitions

Scholars have made efforts to redefine recovery through empirical studies (Kaskutas et al., 2014, 2015) and theoretical construct development (Kelly & Hoeppner, 2015). In an attempt to specify the personal health and wellbeing aspect of recovery, Kaskutas and colleagues (2014) recruited 9,341 individuals from diverse sites nationwide who self-identified as being in recovery and who endorsed a variety of pathways to recovery (e.g., mutual help groups, formal treatment, natural recovery), and had them rate which elements were central to their personal definitions of recovery. In addition to the key role of abstinence in recovery, three additional defining

components of recovery emerged, all of which were found to be stable over time (Kaskutas et al., 2015): essentials of recovery, enriched recovery, and spirituality of recovery. Essentials of recovery are cognitive, affective, and behavioral changes that are necessary to sustain abstinence/low-risk substance use, including having a supportive social network, healthy coping skills, and positive self-regard. Enriched recovery elements pertain to gaining a sense of fulfillment and purpose that often is associated with recovery processes, and they include giving back to the community, taking responsibility for one's actions, and finding inner peace. Lastly, the spirituality of recovery domain highlights the important role of spiritual and/or religious beliefs and practices in recovery processes. In sum, drawing upon lived experiences of individuals, these findings underscore the centrality of sustained holistic and multifaceted improvements in health and wellbeing in defining recovery. However, it remains to be known whether improvements in health and wellbeing are central components of recovery for all.

Based on these empirical studies and roundtables convening experts in the field of both addiction and recovery science, several working definitions of recovery set forth by government and health agencies in the US have been developed. Convened in 2006, the Betty Ford Institute Consensus Panel (2007) posited three fundamental criteria for recovery: sobriety from drugs and alcohol that is sustained over time, personal health and wellbeing, and citizenship. The inclusion of personal health and wellbeing as a key criterion of recovery represented a growing recognition of the importance of physical, mental, and social wellbeing beyond simply the absence of disease. The addition of citizenship, which refers to a sense of recovery identity and membership within a recovery community, signals the acknowledgement of recovery as a personal process of change. Similarly, SAMHSA (2012) adapted their working definition of recovery to better align with Betty Ford's by emphasizing the importance of improvements of functioning and wellbeing

in health, home, purpose, and community that allow individuals to strive towards their full potential. Most recently, the National Institute on Alcohol Abuse and Alcoholism (NIAAA; 2020) adopted a new definition of recovery that highlights this broad, holistic view of recovery, stating that individuals are recovered if they achieve sustained remission from AUD and cessation from heavy drinking. Although not included in its definition, the NIAAA further goes on to note that recovery is often marked by improvements in psychosocial functioning and wellbeing, which can promote sustained recovery. The new definitions of recovery by the latter two federal-level organizations represent turning points in this paradigmatic shift and have the potential to influence the future directions of recovery science research and practice, and preliminary evidence of this shift are indicated by the increased focus on recovery capital.

Recovery Capital Theory. A common thread in all of these new definitions is consideration of improvements in psychosocial functioning and wellbeing that often accompanies the recovery journey. One increasingly common way by which such improvements in functioning can be conceptualized is through recovery capital theory (Hennessy, 2017; Kelly & Hoeppner, 2015). Recovery capital is a continuum of resources that individuals can use to initiate and sustain remission and prevent relapse (Granfield & Cloud, 1999). The concept of recovery capital was derived and adapted from a large body of extant literature on capital applied to other fields (e.g., human resources, business, economics; Hennessey et al., 2017). Capital was applied to the field of recovery science by Granfield and Cloud (1999) after identifying individual, interpersonal, and structural resources that significantly correlated with sustained recovery (Dawson et al., 2012; Hennessy, 2017; White & Cloud, 2008). Additional researchers have extended this work, with results suggesting that recovery capital is prospectively related to sustained recovery (Kelly & Hoeppner, 2015; Laudet & White, 2008). The growing focus on

recovery capital represents a shift away from emphasizing the psychopathology of addiction and towards targeting and building upon existing strengths in recovery (White & Cloud, 2008).

Recovery capital spans internal and external resources across multiple domains. In its original conception as applied to the field of recovery, domains included personal, social, and community capital (Granfield & Cloud, 1999; Hennessey et al., 2017; White & Cloud, 2008). Through refinements, personal capital was divided into human and physical capital (Granfield & Cloud, 1999; White & Cloud, 2008) and cultural capital was added as a key form of community capital (Cloud & Granfield, 2008; White & Cloud, 2008). These domains mirror the individual-, micro-, meso-, and macro-ecological levels outlined in Brofenbrenner's (1979) ecological framework (Hennessey et al., 2017). There are also forms of negative recovery capital, or the factors that undermine one's recovery motivations and efforts, which span all domains/ecological levels and may include things like stigma, negative social norms, relationships with substance-using peers, trauma exposure, stressful life events, and genetic liability for SUDs (Cloud & Granfield, 2008; Hennessey et al., 2017). According to a systematic review of recovery capital, the specific number and division of domains varies across studies (range = 3-5; Hennessey et al., 2017); however, we will use the following domains: social, human, physical, and community/cultural. Each domain of recovery capital is described in more detail below.

Social Recovery Capital. Social recovery capital includes resources such as social support from friends, family, and partners (White & Cloud, 2008). Positive social capital is gained when one's social network is supportive of recovery and/or sobriety efforts, and can include close other participation in treatment and access to sober-friendly leisure activities (White & Cloud, 2008). Social recovery capital is an extremely important correlate of recovery, emerging as a commonly cited and critical resource in qualitative studies of individuals in

recovery (Hennessy, 2017; Laudet et al., 2006). Quantitative studies also support the importance of this domain, with a robust body of literature indicating that greater recovery-oriented social support is associated with less substance use (e.g., Best et al., 2017; Laudet et al., 2006; McCutcheon et al., 2014, 2016; Moos & Moos, 2007) and higher quality of life (Best et al., 2015). In sum, social recovery capital is an essential component of recovery from SUDs with far-reaching benefits.

Human Recovery Capital. Human recovery capital encompasses the intrinsic resources an individual has relating to their own wellbeing and recovery efforts (Hennessey et al., 2017; White & Cloud, 2008). Examples of human recovery capital include mental and physical health, employment and educational skills, interpersonal skills, self-esteem, self-efficacy, personal values, and sense of purpose in life (White & Cloud, 2008). In addition to emerging as commonly endorsed themes in qualitative studies (Hennessey et al., 2017), research suggests that human recovery capital, particularly sense of purpose in life, prospectively predicts sustained recovery (Laudet & White, 2008). Additionally, extant literature suggests that individuals with higher self-efficacy are better able to cope with high-risk situations and avoid relapse (Marlatt & Gordon, 1985; Moos & Moos, 2007). The beneficial effects of human recovery capital on recovery are amplified during early recovery (Moos & Moos, 2007), but they persist over time (Laudet & White, 2008; Moos & Moos, 2007).

Physical Recovery Capital Physical recovery capital refers to the extrinsic and tangible resources an individual has that influences their recovery journeys (Hennessey et al., 2017; White & Cloud, 2008). For example, stable employment, financial assets, stable housing, food security, access to transportation, and health insurance and access to treatment are sources of physical recovery capital (White & Cloud, 2008). According to a systematic review of recovery

capital, the role of physical capital in recovery, particularly financial assets, was an especially prevalent theme that emerged from the qualitative studies reviewed (Hennessey et al., 2017). Quantitative studies also lend evidence supporting the conclusion that financial resources predict less problematic substance use and fewer consequences (Moos & Moos, 2007). This is also echoed in SAMHSA's (2012) working definition of recovery, which includes home as a key component to underscore the importance of housing and food security in resolving substance use problems. In sum, physical capital corresponds to individuals' basic physiological and safety needs (Maslow, 1943) that must be addressed before they can focus on other, higher-order aspects of wellbeing in recovery.

Community / Cultural Recovery Capital. Community recovery capital includes the resources, policies, and attitudes surrounding substance use disorders and recovery (Hennessey et al., 2017; White & Cloud, 2008). Relevant resources and policies that support recovery-oriented systems of care and community-based recovery support services promote recovery capital (White & Cloud, 2008). For example, availability of recovery community-based supports and mutual help groups within a given community serves to increase community recovery capital (White, 2012). Cultural recovery capital, while similar to community capital, incorporates culture-specific attitudes surrounding substance use disorders and recovery (Hennessey et al., 2017) and resources that resonate with individuals' cultural and faith-based identities (White & Cloud, 2008). For example, faith-based recovery services that cater to individuals of various faiths serve to increase cultural recovery capital. Some sources of recovery capital may span both community and cultural domains, such as policies and attitudes towards individuals with histories of criminal charges related to their substance use. In sum, community and cultural

recovery capital are the meso- and macro-level structural and organizational factors that influence the types and availability of resources for individuals in recovery.

Interrelationships Between Sources of Recovery Capital. Although recovery capital is partitioned into different domains, there is significant overlap and connectedness amongst them. First, many treatment programs and recovery-oriented support services aim to increase recovery capital across all domains (Hennessey et al., 2017; White & Cloud, 2008). For example, affiliation with Alcoholics Anonymous (AA), as 12-step based mutual help group, can contribute to accrual of social, human, physical, community, and cultural sources of capital. Individuals in AA may gain a social network of others in recovery (social), efficacy and skills to maintain their abstinence (human), free and unlimited access to a form of treatment (physical), a sense of recovery identity (cultural) and belonging (community). Moreover, extant research suggests a momentum-building phenomenon in which increases in recovery capital in one domain contribute to increases in other domains (Hennessey et al., 2017). Conversely, negative recovery capital can span any domain and can interact with capital across any other domain to detract from one's ability to initiate and sustain recovery (Cloud & Granfield, 2008; Hennessey et al., 2017). This is evidenced by the prevalence of relapse rates even among individuals with substantial recovery capital. These interrelationships make logical sense when considered through the ecological framework, as each domain spans the micro-, meso-, or macro-levels and these levels are encompassed by/encompass the others. This framework highlights the multidimensional and multidirectional aspects of recovery capital, all of which should be considered by treatment providers, policy makers, and other recovery-oriented systems of care.

Biaxial Formulation of Recovery

In an effort to explicitly demonstrate the associations between recovery and improvements in psychosocial functioning and wellbeing, conceptualized as recovery capital, we present the biaxial formulation of recovery. Kelly and Hoeppner (2015) developed a biaxial formulation of recovery in order to develop a clear operational definition of recovery that can be more concretely measured while also clearly distinguishing between remission and recovery. The authors define recovery as "a dynamic process characterized by increasingly stable remission resulting in and supported by increased recovery capital and enhanced quality of life" (p. 9). In this model, remission from SUDs is placed on one axis and recovery capital is placed on the other axis. There is a linear and reciprocal relationship, such that longer time in remission is associated with greater recovery capital, and greater recovery capital is associated with sustained remission. Together, sustained remission and recovery capital contribute to an improved quality of life. Further, this suggests that a particular focus on improving individuals' recovery capital can have far-reaching implications for their abstinence, remission, and functioning in recovery.

For the purposes of this dissertation, we will be using this biaxial formulation of recovery as our guiding framework of recovery. We will refer to recovery as an overarching construct composed of remission and improvements in psychosocial functioning and wellbeing, with the latter measured via recovery capital.

Ongoing Debate Surrounding Recovery Definitions

With the recent refinements to the definition of recovery, there is an ongoing debate about the relative importance of its component features (i.e., psychosocial functioning and wellbeing, abstinence, remission). Some scholars (e.g., Witkiewitz et al., 2019, 2020) assert that the components of holistic improvements in health and wellbeing are (or should be) more central

to defining recovery than abstinence. However, this prioritization of functioning and wellbeing above and beyond abstinence is not without controversy and has sparked debate among scholars in the recovery science field. Scholars who oppose this view (e.g., Kelly & Bergman, 2021) assert that remission status and abstinence/low-risk use should be prioritized. The arguments for each side are outlined below.

In a recent commentary, Witkiewitz and Tucker (2020) stated that abstinence is not an essential component of self-reported recovery for all individuals, and "positive changes in functioning and wellbeing often are more fundamental elements" (p. 36). These claims are supported by extant research suggesting that individuals who reduce their substance use, but are not abstinent, also demonstrate high levels of psychosocial functioning and improvements in wellbeing (Witkiewitz et al., 2019). Such assertions are important to consider given that findings using nationally representative, community-based, and high-risk samples indicate that individuals are more likely to be non-abstinent in remission than abstinent (Dawson et al., 2005, 2012; Mann et al., 2005; McCutcheon et al., 2014). Epidemiological studies estimate that approximately 17-18% of individuals achieve non-abstinent remission from AUD (Dawson et al., 2005; Fan et al., 2019). Together, these findings underscore that non-abstinence is a viable path for many people who consider themselves in recovery.

Moreover, some scholars assert that many individuals who reduce their drinking but continue to engage in occasional heavy drinking exhibit high levels of functioning (Witkiewitz et al., 2019). In a letter to the editor, (Witkiewitz and colleagues (2021) emphasize that sustained abstinence is a high standard that many individuals do not achieve. They further highlight the arbitrary nature of low- and high-risk drinking thresholds as it pertains to AUD and recovery broadly. The focus solely on remission status and abstinence can serve to trivialize the strides

that individuals who consider themselves in recovery make to reduce their alcohol and drug consumption, and can foster a sense of shame and stigma that impedes the recovery process. In sum, the authors claim that such a myopic focus on abstinence has limited utility in public health and on improving the lives of individuals who are trying to reduce risky patterns of alcohol use.

Other scholars push back against the acceptance of substance use in recovery, by any definition, in favor of using remission and abstinence/low-risk use as the defining features (Kelly & Bergman, 2021). In a commentary, Kelly and Bergman (2020) warn of the potential detrimental consequences of deprioritizing abstinence. First, continual engagement in heavy substance use is associated with a variety of health risks, including liver disease, cancers, dementia, and premature death. The authors thus argue that even if psychosocial functioning in recovery, by any definition, is largely unaffected by these health risks, individuals that continue to use substances in recovery still face health-related consequences. Second, the focus on psychosocial functioning of individuals without regard to ongoing substance use fails to recognize the collateral social harms that impact close others. For example, the authors highlight the frequent co-occurrence of negative affect, anger, depression, irritability, and insomnia with heavy substance use, all of which can impact friends, family, and children. Lastly, the primary focus on functioning raises questions about the recovery status of individuals who may be low functioning despite being abstinent and remitted. Given that individuals often exhibit high stress levels and low functioning during the early stages of self-reported recovery (Kelly, Greene, et al., 2018), this may create an unnecessarily high standard for achieving recovery status and perpetuate stigma among those in early recovery. In sum, the authors claim that the focus on functioning and wellbeing regardless of continued substance use is harmful to individuals in recovery as well as close others in their lives.

While there is currently no consensus on how to best resolve this scholarly debate, two things are worth noting. First, reactions to this debate from individuals in self-reported recovery largely center on the lack of lived experience incorporated into these arguments (Coon, 2021). Namely, many of the arguments posited by scholars on both sides of the debate are supported by research, and are removed from clinical and lived experiences of individuals in self-reported recovery (Coon, 2021). Second, although research suggests that individuals who are abstinent and those who reduce their substance use demonstrate improvements in functioning, a robust body of literature suggests that abstainers fare better than non-abstainers (Dawson et al., 2007; Mann et al., 2005; McCutcheon et al., 2014; Subbaraman & Witbrodt, 2014). Compared to non-abstinent individuals in self-reported recovery, abstinent individuals tend to report greater quality of life, even when controlling for time in recovery (Subbaraman & Witbrodt, 2014). Such evidence points to the continued importance of abstinence and remission in recovery, in addition to indicators of wellbeing.

Why These Changes in Definitions Matter

The frequent changes and discrepancies in recovery definitions over time have contributed to a lack of consistent prevalence rate estimates, thereby impeding our global understanding of recovery. These variable operationalizations and measurements of recovery have led to difficulty in estimating prevalence rates of remission and recovery from SUDs (Kelly et al., 2017, 2018; SAMHSA, 2017; Witkiewitz & Tucker, 2020). Results from a recent systematic review of remission from SUDs, defined as not meeting criteria for an SUD in the prior 12 months, suggest that approximately 35% of individuals remit (Fleury et al., 2016). Specific to AUD, in US population-based studies AUD past-year remission rates ranged from 54% to 72% (Dawson et al., 2005; Fan et al., 2019; Hasin et al., 2007). In contrast, using a

broader definition of self-reported recovery yields slightly higher prevalence rates. Estimates of US adults with a history of SUDs who consider themselves to be in recovery range from 50% (Kelly et al., 2018; Kelly et al., 2017) to 75% (SAMHSA, 2017). The lack of consistent prevalence rate estimates can lead to either wasteful allocations or shortages of treatment resources, depending on whether we overestimate or underestimate the number of people in recovery. Although the newly expanded conceptualization of recovery to include a focus on psychosocial functioning and wellbeing represents an improvement over previous operationalizations, there is still no standardized definition of recovery. Establishing a standardized, consistently used definition that draws upon this expanded understanding of recovery and upon which stakeholders (e.g., funding organizations, healthcare providers, and individuals with lived experience) can agree will facilitate the understanding of the scope of who recovers from SUDs and significantly advance the field of recovery science.

Correlates of Recovery

Despite ongoing refinements to the definition of recovery, research on correlates of this process has continued. The extant research on correlates presented below are broken down by axiom of recovery: remission and recovery capital (Kelly & Hoeppner, 2015). First, it is important to note that many studies examining the correlates of recovery have done so without a clear operational definition of recovery, following the school of thought that someone is in recovery if they say they are in recovery (Valentine, 2018). Well-established positive correlates of remission status and self-reported recovery include ever receiving substance use treatment (Dawson et al., 2012; Jones et al., 2020) and higher spirituality (Dawson et al., 2012; Laudet et al., 2006). Other established positive correlates of remission in particular include being female, being married (Dawson et al., 2005, 2012; McCutcheon et al., 2012, 2014), later age of SUD

onset (Dawson et al., 2005), and being employed (Dawson et al., 2012; McCutcheon et al., 2012; Moos & Moos, 2007). In contrast, cigarette use, illicit drug use (Dawson et al., 2012), deviant peer affiliation (Brown et al., 1989), negative affective states (Marlatt, 1996), history of childhood trauma (McCutcheon et al., 2012), and greater severity of SUD problems was negatively correlated with remission status (Dawson et al., 2005). These correlates provide foundational knowledge of who is likely to remit or recover from SUDs.

With the recent emphasis on recovery capital, research on correlates of recovery capital has increased. As might be expected, recovery capital is associated with abstinence and remission from SUDs (Laudet & White, 2008). Research suggests that individuals with greater recovery capital exhibit more self-efficacy than those with less recovery capital (Gilbert & Kurz, 2018), suggesting that one mechanism by which recovery capital influences abstinence from alcohol and drugs is through self-efficacy. Specifically, spirituality and AA affiliation were associated with higher self-efficacy related to alcohol abstinence, and social support was associated with higher self-efficacy related to drug abstinence (Gilbert & Kurz, 2018). Beyond abstinence and remission status, greater recovery capital was associated with greater psychosocial functioning and wellbeing among individuals in recovery (Cano et al., 2017; Laudet et al., 2006; Laudet & White, 2008; White & Cloud, 2008). Other correlates of recovery capital include longer time spent living in recovery housing, engaging in meaningful activities (e.g., employment, volunteering, educational pursuits; Cano et al., 2017), and decreasing the salience of barriers to recovery (Best et al., 2021; Cano et al., 2017). In sum, these correlates of recovery capital provide insight into the second axiom of recovery, or what factors can help individuals to sustain remission and contribute to enhanced functioning and quality of life.

Pathways to Recovery

Pathways to recovery follow the principle of equifinality, in that multiple different paths can lead to the same outcome (Kelly et al., 2017). Pathways generally fall into two categories, assisted and unassisted, with assisted pathways further divided into clinical and non-clinical pathways (Kelly et al., 2017). Assisted clinical pathways to recovery include substance use treatment, such as clinical treatments, pharmacotherapy, behavior therapy, and other holistic therapies (Kelly et al., 2017; Moos & Moos, 2007; Pennelle, 2019; Sobell et al., 2000). Assisted non-clinical pathways to recovery include mutual help groups, recovery community centers, collegiate recovery programs, and recovery housing (Kelly et al., 2017; Moos & Moos, 2007; Pennelle, 2019; Sobell et al., 2000). In contrast, unassisted pathways to recovery include natural means of problem resolution, such as "maturing out" of use because of competing developmental demands (e.g., work, marriage, children, caring for elderly parents; Kelly et al., 2017; Lee et al., 2013) or other self-managed pathways (Pennelle, 2019). It is common for individuals to utilize multiple pathways to recovery (Kelly et al., 2017), and some studies suggest that individuals who endorse multiple pathways exhibit better outcomes than those who endorse only one pathway to recovery (Kelly et al., 2017; Laudet et al., 2002). Overall, pathways to recovery should bolster one's recovery capital - the resources that help them achieve and maintain remission - and promote positive recovery outcomes.

Assisted Pathways

According to a study of recovery pathways using a national sample of individuals who reported resolving a problem with alcohol and/or other drugs (Kelly et al., 2017), approximately half (53.9%) of individuals in self-reported recovery reported lifetime use of an assisted pathway. Use of assisted pathways was higher among individuals in midlife and those with

younger age of initiation, polysubstance use, more severe substance use problems, comorbid mental health conditions, and criminal justice involvement. The most commonly endorsed assisted pathways included mutual help groups (45%), followed by formal substance use treatment (28%) and community-based recovery supports (22%). Involvement in mutual help groups is not only frequently endorsed, but also prospectively predictive of sustained recovery and lower stress levels over time (Laudet & White, 2008; Moos & Moos, 2007). We discuss mutual help groups, the most common pathway to recovery, below.

Mutual help groups are peer-run organizations that aim to help individuals achieve and maintain abstinence from alcohol and/or other drugs (Moos, 2008). The most well-known mutual help group is Alcoholics Anonymous (AA), which follows a 12-step philosophy to achieve abstinence (Donovan et al., 2013). Other examples of mutual help groups, some but not all of which follow 12-step philosophies, include Narcotics Anonymous (NA), SMART Recovery (Rettie et al., 2021), Recovery Dharma, Refuge Recovery, and Secular Organizations for Sobriety (Pennelle, 2019). Across both 12-step and non-12-step mutual help groups, several components have been identified as useful to recovery processes: perspective-taking, connection to others, skill development, the value of group activities, and a change in self (Rettie et al., 2021). Each of these components serve to increase positive recovery capital across one or more domains by helping individuals to improve their self-efficacy and self-esteem, providing sober social supports and leisure activities, improving coping skills, establishing new recovery-promotive social norms, and creating a sense of meaning (Moos, 2008; Rettie et al., 2021). Spirituality, which is frequently emphasized within non-secular mutual help group organizations, also predicts greater human recovery capital, measured via higher quality of life and lower stress levels (Laudet & White, 2008). Together, this contributes to the superior effectiveness rates of

mutual help groups (Kelly et al., 2020; Moos, 2008) relative to other forms of substance use treatment (Kelly et al., 2020).

Unassisted Pathways

Estimates from the National Recovery Survey suggest that nearly half of individuals who self-identify as being in recovery resolve their alcohol and/or drug problems through unassisted pathways, often referred to as spontaneous or natural recovery (Kelly et al., 2017). The use of unassisted pathways was higher among individuals with less severe substance use problems and less complex mental health troubles (Kelly et al., 2017). Individuals who achieve and maintain recovery through unassisted pathways may have especially high levels of social recovery capital, as prior research found that individuals who naturally recovered, defined via self-reported recovery status, from AUD reported high levels of spousal support (Sobell et al., 1993). Further, individuals may be more likely to opt for unassisted pathways to recovery as a means to moderate their own substance use, rather than quit entirely (Slutske, 2010). This speculation is supported by extant research suggesting that individuals who use unassisted pathways are more likely to continue to use substances in moderation compared to those who use assisted pathways (Fan et al., 2019). Lastly, some individuals may avoid assisted pathways because of the stigma surrounding SUDs and addiction and/or the belief that they should be able to resolve the problem on their own (Cunningham et al., 1993). In sum, despite the varied reasons for choosing unassisted pathways, many individuals are able to achieve natural self-reported recovery from SUDs (Fan et al., 2019; Kelly et al., 2017; Sobell et al., 1993).

Gaps in the Literature and Introduction to the Dissertation

Gaps in the Literature

Despite the growing number of studies in the field of recovery science, critical limitations exist. First, there are only a small number of studies that have focused on variability in individuals' journey to recovery (Kelly et al., 2019; Kelly, Greene, et al., 2018; Witkiewitz et al., 2019, 2020). In a recent study investigating the number of attempts required to successfully resolve an alcohol or drug problem, Kelly and colleagues (2019) found evidence suggesting there are subgroups of individuals with different pathways to recovery and who require a different number of attempts to resolve their substance problem, providing preliminary evidence of heterogeneity in recovery journeys. Another study underscoring the variability in recovery journeys found that the association between recovery capital and resolution of alcohol/drug problems was not linear, such that the biggest increases in positive measures were during the first five years post-resolution, followed by gradual increases out until 40 years post-resolution (Kelly et al., 2018). Lastly, recent research found evidence to suggest that individuals exhibit heterogeneous patterns of psychosocial functioning following post-AUD treatment, with some individuals demonstrating high levels of psychosocial functioning despite engaging in heavy drinking, and others exhibiting low levels of functioning despite infrequent heavy drinking (Witkiewitz et al., 2019, 2020). In sum, these findings are indicative of variability in recovery journeys that may depend on individual differences, time in recovery, and recovery capital.

Importantly, however, the authors used self-reported resolution of an alcohol or drug problem (Kelly et al., 2018, 2019) or substance use treatment history (Witkiewitz et al., 2019, 2020) as sample inclusion criteria. Neither self-reported problem resolution, treatment participation, nor prior treatment completion necessitates sustained remission status, meaning

some individuals included in those studies may have met criteria for an active alcohol or substance use disorder at the time of assessment. Research that uses diagnostic remission criteria as sample inclusion criteria, instead of self-reported resolution and/or treatment history, may capture different samples and yield different patterns of results. It is currently unclear whether patterns and processes associated with recovery may differ between remitted and non-remitted individuals, regardless of self-reported resolution and/or treatment history. Thus, additional research is needed to better characterize this heterogeneity in recovery journeys, including replicating findings across remitted samples and investigating correlates that discriminate between observed subgroups.

A second critical limitation is that, although it is well-established that genetic influences play a key role in substance use behaviors (Deak & Johnson, 2021; Turkheimer, 2000), very few studies have incorporated genetic influences into their studies of recovery processes. Of the existing studies at the intersection of these two fields, most have focused specifically on AUD. Among those studies, there are mixed findings on the associations between genetic liability for AUD and abstinence in AUD remission, with some studies finding that individuals with a family history of AUD were more likely to be in non-abstinent remission (Dawson et al., 2005, 2012; Mann et al., 2005; McCutcheon et al., 2014) and others finding no associations between family history of AUD and abstinent or non-abstinent remission (Knop et al., 2007). Taken together with extant studies suggesting that individuals with a family history of AUD are more likely to relapse (Farmer et al., 2022), the discrepant findings depending on abstinent or non-abstinent remission are important to resolve because continued alcohol use can influence recovery capital (Mann et al., 2005; McCutcheon et al., 2014; Subbaraman & Witbrodt, 2014), psychosocial

functioning and quality of life (Subbaraman & Witbrodt, 2014), and risk of relapse (Sliedrecht et al., 2019).

More broadly, genetic influences can influence a range of behavioral outcomes (Turkheimer, 2000), many of which may correspond to individuals' recovery capital and quality of life in remission. Specifically, extant research suggests that a substantial proportion of genetic variance for AUD is accounted for by a shared, heritable liability towards externalizing behaviors (Barr & Dick, 2019; Dick et al., 2010; Krueger et al., 2002). This is relevant because a predisposition towards externalizing may influence psychosocial functioning and quality of life among those in remission. For example, one study found that family history of substance misuse was associated with greater delayed discounting (an indicator of impulsivity) in offspring, which was in turn related to offspring's substance use later in life (VanderBroek et al., 2016). Thus, delayed discounting may be one mechanism by which family history influences substance use. This is important to consider given that delayed discounting is associated with treatment outcomes, risk of relapse, and quality of life among individuals in remission (Athamneh et al., 2019, 2022). Substance use disorders may also share an underlying heritability towards internalizing behaviors, as substance use is highly comorbid with mood and anxiety disorders (Edwards et al., 2012; Hesselbrock & Hesselbrock, 2006; Kessler et al., 2003; Tully & Iacono, 2016). Negative affective states, like depression and anxiety, are a potent form of negative recovery capital (Cloud & White, 2008; Hennessey, 2017) and are one of the most common predictors of relapse (Marlatt, 1996). Together, parallel lines of research suggest that genetic influences are critical to understand and contextualize recovery capital and quality of life among individuals in remission.

Lastly, no studies to our knowledge have used polygenic risk scores, a state-of-the-science index of genetic liability that involves additive genetic effects across the genome, when examining the associations between genetics and recovery processes. Studies at the intersection of genetics and recovery science have used family history as an index of genetic liability, which makes it challenging to disentangle genetic and environmental effects. Complex disorders, such as substance use and recovery, are influenced by many genes of small effects, and measures of family history may not reflect this. In a recent study, Lai and colleagues (2022) found that polygenic risk scores significantly predicted AUD even after adjusting for family history, suggesting that polygenic risk scores account for additional variance in AUD. Thus, additional research on the role of genetic influences on recovery processes, particularly that which uses state-of-the-science approaches to measure genetic liability, is necessary to advance the field of recovery science and to provide preliminary insights into personalized alcohol and substance use treatments.

Introduction to the Dissertation

With the definition of recovery evolving towards a process rather than an outcome, there is a need to better understand psychosocial functioning and quality of life among individuals at different points in their recovery journeys. Concurrently, additional research is needed to understand the interplay between sources of recovery capital, individual differences in risk factors for substance use, genetic influences, and psychosocial functioning and quality of life. The goal of this dissertation was to bring together the literatures on recovery capital, epidemiological risk factors, and genetic risk factors to elucidate the patterns, predictors, and processes of recovery from SUDs. The current study conducted to achieve this goal is briefly described below.

In the current study, we used a family-based, genetically informed dataset of adults that was enriched for risk of AUD, the Collaborative Study on the Genetics of Alcoholism (COGA) Prospective Study (N = 3,129). Based on the recent emphasis on understanding functioning and wellbeing among individuals beyond their remission status, we characterized and predicted patterns of functioning related to quality of life in a sample of individuals who reduced their symptoms such that they no longer met AUD criteria. We first investigated profiles of functioning related to quality of life among a sample of individuals in remission from AUD. Next, we examined predicters of profile membership. We tested whether proximal sources of recovery capital (social support for recovery, attendance at religious services, treatment history) and distal sources of negative recovery capital (deviant peer affiliation during adolescence, interpersonal trauma) predicted profile membership. We also tested the extent to which epidemiological risk factors (substance use history, internalizing characteristics, and externalizing behaviors), and genetic liabilities towards alcohol problems, externalizing behaviors, and internalizing behaviors influenced patterns of quality of life. Findings from this study can elucidate the role of a diversity of distal and proximal influences on patterns of quality of life, and may lead to an improved understanding of AUD recovery trajectories.

Profiles and Predictors of Functioning Among COGA Prospective Study Adults in Recovery from Alcohol Use Disorders

Background

Alcohol use disorders (AUD) are associated with high personal and societal costs (Grant et al., 2015; Hasin et al., 2007; Rehm et al., 2009), and are one of the leading causes of premature death and disability (Griswold et al., 2018; Peacock et al., 2018; Rehm et al., 2009). Approximately 40-75% of affected individuals remit from AUD (Dawson et al., 2005, 2013; Dennis et al., 2005; Kelly et al., 2017); however, compared to the study of the development of alcohol problems, the factors that contribute to AUD recovery are not as well characterized.

Traditionally, recovery was defined simply as remission from AUD. Under this traditional definition, abstinence from alcohol and drugs was considered the main mechanism by which individuals recovered (Kaskutas et al., 2014; Kelly & Hoeppner, 2015; White, 2012). More recently, recovery has been redefined to capture broader improvements in quality of life (Brown & Ashford, 2019; Kelly & Hoeppner, 2015; Laudet, 2007; Neale et al., 2016). Recovery from AUD is now understood to be a dynamic process involving both remission and improvements in psychosocial functioning and wellbeing, with the latter measured via recovery capital (i.e., the continuum of resources available to sustain remission; Brown & Ashford, 2019; Cloud & Granfield, 2008; Kelly & Hoeppner, 2015; Laudet, 2007; Neale et al., 2016).

According to the biaxial formulation of recovery (Kelly & Hoeppner, 2015), remission and recovery capital linearly and reciprocally related. Accordingly, longer time in remission is associated with accrual of greater recovery capital, and greater recovery capital is associated with sustained remission (Dennis et al., 2007; Kelly & Hoeppner, 2015; Marlatt, 1996). Together, these contribute to improvements in psychosocial functioning and quality of life (Kelly &

Hoeppner, 2015; Laudet et al., 2006; Laudet & White, 2008; White & Cloud, 2008). Throughout this paper, we refer to recovery as the overarching process of remitting from AUD and accruing recovery capital in pursuit of improved quality of life, with specific references to recovery components (i.e., remission or recovery capital) as appropriate.

Recovery and Quality of Life

The path to improved quality of life in AUD remission is increasingly recognized as heterogeneous, with new studies indicating that individuals may experience more or less improvement in quality of life compared to others (Witkiewitz et al., 2019). However, few studies have systematically examined patterns and predictors of quality of life among this population. Another gap in the field is that although it has become clear that understanding AUD necessitates a genetically informed perspective (Turkheimer, 2000), this has not been widely integrated into the field of recovery science. We know from twin and family studies that the development of AUD is heritable (Hart & Kranzler, 2015; Verhulst et al., 2015). Further, the influence of genetic factors on diverse behavioral outcomes (Turkheimer, 2000) suggests differential improvements in quality of life among those in AUD remission may also be genetically influenced. Improved understanding of quality of life among individuals in AUD remission will improve personalized treatment, facilitate precision medicine, and promote recovery-oriented systems of care.

Key Areas of Quality of Life

Quality of life among individuals with SUDs is generally poor (Laudet et al., 2006), such that one frequently given reason for initiating recovery is the hope for a better life (Granfield & Cloud, 1999; Laudet et al., 2002). Previous studies have highlighted key areas of quality of life correlated with sustained remission: life satisfaction, mental and physical health, and abstinence

from alcohol and drugs (Cloud & Granfield, 2008; Kaskutas et al., 2014, 2015; Neale et al., 2016). We briefly highlight the relevant research on each area below.

First, life satisfaction can refer to individuals' subjective satisfaction, experiences, and level of functioning with their life generally or with specific facets of their life, including social domains, vocational, romantic, familial (Christie et al., 2021; Laudet, 2011). Individuals with SUDs generally report lower life satisfaction than those without SUDs (Donovan et al., 2005; Laudet, 2011), in part because of the deleterious effects and consequences of chronic substance use on one's health and psychosocial functioning (Laudet, 2011). Recovery from these effects and consequences, as well as the hopes of living a better, more satisfying life are frequently identified goals among individuals who want to resolve their substance use problems (Granfield & Cloud, 1999; Laudet et al., 2002). As expected, longer time in remission is positively and reciprocally related to greater life satisfaction (Christie et al., 2021; Kelly & Hoeppner, 2015; Laudet et al., 2006; Laudet & White, 2008). Moreover, recent research suggests that general life satisfaction post-problematic substance use is comparable to general satisfaction levels before the onset of problematic use (Christie et al., 2021). This extant research underscores the importance of life satisfaction as a critical component associated with sustained remission.

Individuals with SUDs generally have poor health, and another frequently given reason for initiating recovery is to avoid negative consequences associated with ongoing substance use (Moos & Moos, 2007), including adverse health consequences from continued use. As expected, improvement in health is strongly correlated with sustained remission (Laudet et al., 2006), and time in remission is positively correlated with better health (Kelly & Hoeppner, 2015; Laudet et al., 2006; Laudet & White, 2008). Indeed, previous research has found that the most common cause of relapse was related to negative physical, psychological, and social states (Marlatt,

1996). This thus indicates that quality of life and mental and physical health are domains of psychosocial functioning that are key to sustained remission.

Lastly, abstinence is strongly associated with sustained remission and improvements in quality of life (Kelly & Bergman, 2021; Subbaraman & Witbrodt, 2014). Research suggests that individuals who are abstinent and those who reduce their substance use demonstrate improvements in functioning, with abstainers showing greater improvements relative to non-abstainers (Donovan et al., 2005; Kline-Simon et al., 2013; Mann et al., 2005; McCutcheon et al., 2014; Subbaraman & Witbrodt, 2014). Moreover, compared to non-abstinent individuals in remission, abstinent individuals tend to report greater quality of life, even when controlling for time in remission (Subbaraman & Witbrodt, 2014). Together, this suggests that individuals in abstinent remission, compared to those in non-abstinent remission, demonstrate greater gains in recovery capital and quality of life that can help sustain their remission.

Heterogeneity in Patterns of Functioning Related to Quality of Life

Despite extant literature suggesting that improvements in psychosocial functioning and quality of life are associated with sustained remission, recent research by one group of researchers indicates that individuals post-AUD treatment exhibit heterogeneous patterns of psychosocial functioning (Witkiewitz et al., 2019, 2020). Witkiewitz and colleagues (2019) examined a sample of individuals at three years post-AUD treatment, and they found evidence of four profiles: 1) low functioning, frequent heavy drinking; 2) low functioning, infrequent heavy drinking; 3) high functioning, occasional heavy drinking; and 4) high functioning, infrequent non-heavy drinking. The authors replicated this work, again finding evidence for four profiles of functioning, among another sample of individuals post-AUD treatment (Witkiewitz et al., 2020). These findings suggest individuals may demonstrate high levels of psychosocial functioning

despite engaging in heavy drinking, or low levels of functioning despite infrequent heavy drinking (Witkiewitz et al., 2019, 2020). This extant research thus suggests that examination of AUD recovery based solely on remission from AUD fails to capture the variation in psychosocial functioning and quality of life critical to a holistic understanding of recovery (Cloud & Granfield, 2008; Granfield & Cloud, 1999).

One limitation of the existing research on functioning in AUD recovery is that prior work in this area was not limited strictly to samples in current remission, meaning some individuals may have had active AUD at the time of functioning assessments. Specifically, the research in this area conducted by Witkiewitz and colleagues (2019) used participants from the outpatient treatment arm of Project MATCH who met criteria for DSM-III-R alcohol abuse or dependence. Psychosocial functioning was measured at baseline, during treatment, and four times post-treatment. Importantly, it is unclear to readers whether study participants had active AUD at each of the assessments, as treatment participation and/or prior treatment completion does not necessitate sustained remission status. Further, approximately 60% of the sample engaged in heavy drinking at three years post-treatment (Witkiewitz et al., 2019). Although considerations beyond remission are important, patterns of functioning may differ between remitted and non-remitted individuals.

A second limitation of the existing research in this area is that the majority of extant studies are limited to samples with a lifetime history of treatment. Epidemiological studies suggest that less than one-quarter of US adults with prior to past year AUD ever received treatment (Fan et al., 2019), while other studies indicate that many individuals resolve their AUD problems naturally (Fan et al., 2019; Grant et al., 2015; Slutske, 2010), so studies that exclude non-treatment samples fail to capture a large proportion of remitted individuals. Further,

previous studies suggest that individuals with severe AUD are more likely to seek treatment than those with less severe AUD (Dawson et al., 2005, 2012). It is possible that differences in AUD severity and treatment-seeking behaviors may result in differential functioning among remitted individuals. Together, these limitations point to the need for additional research on functioning among individuals in AUD remission, unconstrained by treatment history.

Recovery Capital as Predictors of Heterogeneous Improvements in Quality of Life

To date, there is limited understanding of what factors impact patterns of functioning related to quality of life in AUD remission. However, recovery capital serves as a foundational framework through which heterogeneity in quality of life can be examined. Prior work in this area, conducted by Laudet and colleagues (2006), focused on the role of social support, spirituality and religiousness, and 12-step affiliation as recovery capital that can buffer against stress to improve quality of life among a sample of individuals who previously met criteria for SUD and reported one or more months of current abstinence. The authors found that social support, spirituality and religiousness, and 12-step affiliation significantly buffered the pathogenic effects of stress on quality of life. These results highlight the importance of these specific sources of recovery capital on quality of life among individuals on the path to recovery. Importantly, however, variation in patterns of quality of life were not examined. Thus, in the present study, we focused on these same proximal sources of recovery capital (i.e., social support, spirituality and religiousness, and 12-step affiliation) as predictors of heterogeneous patterns of quality of life. We also examined distal sources of negative recovery capital (i.e., history of interpersonal trauma, deviant peer affiliation during adolescence). We discuss extant research on each in greater detail below.

Social Support. Social support is one robust form of recovery capital that is associated with sustained remission and improved quality of life (Laudet et al., 2006; Moos & Moos, 2007). First, social support is directly associated with improvements in multiple aspects of psychosocial functioning, reductions in stress levels, and greater engagement in health behaviors (Cohen & Wills, 1985; Ditzen & Heinrichs, 2014; Umberson et al., 2010). Moreover, research suggests that social support buffers against the pathogenic effects of stressful life events to enhance quality of life (Laudet et al., 2006) and mitigate alcohol use (Smith et al., 2021), underscoring the importance of social recovery capital. In particular, social support for recovery is essential to making the lifestyle changes necessary to sustain remission and avoid relapse, as greater social recovery capital can improve access to recovery supports, sober leisure activities, and foster a sense of recovery identity (Clifford & Longabaugh, 1992; Laudet et al., 2006; Mawson et al., 2015; White & Cloud, 2008). In sum, greater social support is associated with improved quality of life among individuals in remission because social support allows individuals to maintain their recovery while living a meaningful, connected life. As such, social support is a critical domain to understanding differential patterns of quality of life among those in AUD remission.

Spirituality and Religiosity. Spirituality and religiosity are overlapping but distinct forms of human and community recovery capital that can play key roles in many individuals' journeys to sustained remission (Laudet et al., 2006). Indeed, the vast majority of individuals who self-identify as being in recovery consider spiritual and/or religious beliefs and practices essential to their personal definitions of recovery (Kaskutas et al., 2015). Spirituality and religiosity often serve as a way to make meaning out of life events, facilitate finding one's purpose in life, and can foster a sense of group membership (Villani et al., 2019). A robust body of literature suggests that spirituality and religiosity is positively associated with better greater

quality of life via better psychosocial adjustment and higher life satisfaction (Koenig et al., 2012; Laudet et al., 2006; Laudet & White, 2008; Villani et al., 2019). Further, higher spirituality and religiosity can buffer against stress and lead to enhanced quality of life (Laudet et al., 2006). Lastly, spirituality and religiosity are inversely related to frequency of substance use (Koenig et al., 2012). In light of research highlighting the psychosocial benefits of spirituality and religiosity and the central role of spiritual/religious practices emphasized by individuals who self-identify as in recovery, this form of recovery capital is a key area to include in understanding patterns of quality of life among those in remission.

12-Step Affiliation. Affiliation with 12-step mutual help groups is one of the most common pathways to remission (Kelly et al., 2017) and serve as a critical form of community recovery capital. Affiliation with 12-step groups is associated with sustained abstinence from substances (Kelly et al., 2020; Subbaraman & Witbrodt, 2014) and elevated levels of recovery-oriented supports (Rettie et al., 2021), all of which can contribute to sustained remission and greater quality of life. Moreover, 12-step groups are frequently faith-based and/or integrate spiritual practices, which can facilitate finding a sense of higher power and purpose in life. Lastly, there is some evidence suggesting that 12-step affiliation is associated with reductions in stress and enhanced quality of life (Laudet et al., 2006). To that end, affiliation with 12-step groups is a foundational form of recovery capital that may contribute to differential patterns of quality of life among individuals in remission.

Interpersonal Trauma. Interpersonal trauma is a form of negative recovery capital that is associated with mental, physical, and social distress (Khantzian, 2004; Overstreet et al., 2017; Read et al., 2012). Interpersonal trauma refers to a traumatic event in which another person is responsible for perpetrating the event (as opposed to a natural disaster or trauma resulting from

combat or war), including physical and sexual assault or abuse (Kessler, 1995; McLaughlin et al., 2013). Exposure to interpersonal trauma is associated with elevated levels of alcohol and substance use in adolescence and adulthood (Begle et al., 2011; Berenz et al., 2016; Breslau, 2009; Keyes et al., 2011; Overstreet et al., 2017; Smith et al., 2021), and is prospectively associated with the development of SUDs (Cicchetti & Handley, 2019; Norman et al., 2012). For example, those exposed to interpersonal trauma during childhood tend to initiate substance use earlier (Dube et al., 2003) and exhibit more severe substance-related problems (Shin et al., 2013), relative to those without childhood interpersonal trauma. This is important to note given that individuals with more severe SUDs tend to require more treatment and a greater number of recovery attempts than those with less severe problems (Kelly et al., 2019), which may be in part because they have accrued more negative recovery capital and less cumulative capital to help them sustain remission (Cloud & Granfield, 2008). Specifically, interpersonal trauma exposure is associated with negative sense of self (Kouvelis & Kangas, 2021), poorer self-esteem, poorer emotional regulation (Cicchetti & Toth, 2015; Toth & Cicchetti, 2013), and higher stress reactivity (McEwen, 2004). Together, these consequences contribute to a lack of coping skills to successfully manage adverse events, which is related to an increased likelihood of relapse (Moos & Moos, 2007; Sliedrecht et al., 2019). Drawing on these parallel lines of research, we can speculate that interpersonal trauma exposure may negatively impact one's quality of life and ability to sustain remission.

Deviant Peer Affiliation. Deviant peer affiliation is another form of negative recovery capital that may detract from one's quality of life in remission. Deviant peer affiliation is associated with an increased likelihood that individuals will engage in risky activities (Hawkins et al., 1992; Kendler et al., 2018; Smith et al., 2019), which may jeopardize individuals' recovery

journeys and wellbeing. Individuals who affiliate primarily with deviant peers tend to engage in risky alcohol use that persists over time, often creating a cycle whereby individuals select into similar peer groups over time which influences their risky alcohol use, and vice versa (Hawkins et al., 1992; Kendler et al., 2018). In contrast, lower peer group substance use is associated with greater recovery capital for the group's individual members and better quality of life (Mawson et al., 2015). Thus, deviant peer affiliation may negatively impact individuals who are seeking to establish recovery-supportive social networks, which in turn can impede their ability to foster a recovery identity, limit their access to recovery supports, and detract from one's social wellbeing that is critical for sustained recovery (Gregoire & Snively, 2001; Laudet et al., 2006; Mawson et al., 2015).

Epidemiological Substance Use Risk Factors as Predictors of Heterogeneous Improvements in Quality of Life

There is a robust body of literature on the epidemiological risk factors for substance use. Well-established risk factors include one's alcohol and substance use history, internalizing characteristics, and externalizing behaviors (Dick, 2011; Hart & Kranzler, 2015; Prom-Wormley et al., 2017). Some researchers have incorporated these risk factors into their studies on recovery, remission, and quality of life (e.g., Daeppen et al., 2014; Tuithof et al., 2014), and we briefly discuss relevant findings from this body of literature below. Importantly, however, relatively few studies have used these risk factors as predictors of heterogeneous patterns of quality of life among those in remission, and fewer studies have considered these risk factors in conjunction with recovery capital.

Alcohol and Substance Use History. Epidemiological risk factors related to alcohol and substance use histories are important to consider as they may influence one's quality of life in

remission. Specifically, prior research suggests that individuals' alcohol use behaviors are associated with their path to recovery, such that earlier onset of alcohol use and more severe AUD are associated with poorer treatment outcomes and functioning (Dawson et al., 2007, 2008; Grant & Dawson, 1997; Kelly et al., 2019). Individuals who initiate alcohol use earlier, relative to those who are older at the age of initiation, tend to develop more severe AUD (Hingson et al., 2006). Further, individuals with a history of severe AUD are at elevated risk of relapse (Fleury et al., 2016; Hingson et al., 2006; Tuithof et al., 2014). Together, these provide preliminary evidence that one's substance use history can influence the course and trajectory of remission, which may include quality of life in remission.

Externalizing Behaviors. Externalizing behaviors represent individual differences that may influence one's recovery capital and differentially influence one's quality of life in remission. Externalizing behaviors, such as sensation seeking and impulsivity, are associated with increased likelihood that individuals will engage in risky activities (Dick et al., 2010; Kramer et al., 2008; Slutske et al., 1998), which may jeopardize individuals' recovery journeys and wellbeing. Delay discounting rates, a measure of the subjective decline in value of a reward based on the delay that is associated with externalizing behaviors and SUDs (Madden & Bickel, 2010), is negatively associated with quality of life and AUD remission status (Athamneh et al., 2022). Further, externalizing disorders are associated with poor substance use treatment outcomes (Winters et al., 2008), including higher number of relapses after treatment (Robbins et al., 2011).

Internalizing Characteristics. Internalizing characteristics, like depression and anxiety symptoms (Edwards et al., 2016; Hesselbrock & Hesselbrock, 2006) can also negatively influence individuals' mental, physical, and social wellbeing. Longer duration of abstinence is

associated with enhanced quality of life, which is inversely related to psychological distress (Hagen et al., 2017). Moreover, individuals may turn to substance use as a means to cope with depression and anxiety symptoms (Hawn et al., 2020; Khantzian, 1997), and comorbid depression and SUDs is associated with an enhanced risk of relapse (Flynn & Brown, 2008). Together, these findings suggest that externalizing and internalizing behaviors may impede individuals' ability to sustain their remission and enhance their quality of life.

Post-Traumatic Stress Disorder. Beyond internalizing behaviors, such as depression and anxiety, post-traumatic stress disorder (PTSD) represents a severe reaction to a traumatic event (Breslau & Davis, 1992; Kessler et al., 1995). This is significant because individuals whose post-traumatic stress symptoms are severe and pervasive enough to meet the clinical threshold for PTSD are 1.6 times more likely be to diagnosed with alcohol use disorders compared to individuals without PTSD (Stewart, 1996). Estimates suggest that among individuals with SUD, approximately 36-52% experience co-occurring PTSD (Breslau & Davis, 1992; Kessler et al., 1995). Given the substantial comorbidity between PTSD and alcohol or substance use disorders, individuals in recovery with a history of PTSD may respond less favorably to treatment, be less able to sustain their remission, and be more likely to relapse (Read et al., 2004). In addition to its association with the development and escalation of problem drinking, PTSD is also related to a poorer quality of life (Blakey et al., 2021), including impairments in physical functioning, general health, mental health (Evren et al., 2011), and interpersonal functioning (Najavits et al., 1997). In sum, extant research indicates that PTSD may influence one's ability to sustain their remission, as well as impact their quality of life in remission.

Genetic Risk Factors as Predictors of Heterogeneous Improvements in Quality of Life

It is well-established that genetic influences play a key role in a range of behavioral outcomes (Turkheimer, 2000), so the role of individual differences in genetic liabilities on quality of life are important to consider. Twin and family studies suggest that the development of AUD is 50-60% heritable (Hart & Kranzler, 2015; Verhulst et al., 2015). However, there are mixed findings on the associations between genetic liability for AUD and remission from AUD, with some studies finding that individuals with a family history of AUD were more likely to be in non-abstinent remission (Dawson et al., 2005; Dawson et al., 2012; Mann et al., 2005) and others finding no associations between family history of AUD and abstinent or non-abstinent remission (Knop et al., 2007). The discrepant findings depending on abstinent or non-abstinent remission are important to resolve because continued alcohol use has implications on one's recovery capital (Mann et al., 2005; McCutcheon et al., 2014; Subbaraman & Witbrodt, 2014) and risk of relapse (Sliedrecht et al., 2019). Specifically, abstinent remission is associated with greater quality of life relative to those in non-abstinent remission (Subbaraman & Witbrodt, 2014). In sum, these mixed findings underscore the need for additional research to clarify the underlying associations, and to elucidate the relationships between genetic liability and quality of life among those in AUD remission.

Evidence from extant research suggests that substance use is part of a larger taxonomy of psychopathology, whereby substance use is a subfactor of higher-order dimensions of externalizing and internalizing characteristics (Kotov et al., 2017). This claim is supported by findings that a large proportion of genetic variance for AUD is accounted for by a shared, heritable liability towards externalizing behaviors (Barr & Dick, 2019; Dick et al., 2010; Krueger et al., 2002). Further, research suggests that SUDs are highly comorbid with mood and anxiety

disorders (Edwards et al., 2012; Hesselbrock & Hesselbrock, 2006; Kessler et al., 2003; Tully & Iacono, 2016), indicating an underlying shared genetic liability. Taken together, the profound and ubiquitous influence of genetic factors on diverse behavioral outcomes (Turkheimer, 2000) suggests differential patterns of quality of life in remission may also be genetically influenced.

Drawing on these converging lines of evidence suggesting the importance of genetic predisposition towards AUD, externalizing characteristics, and internalizing characteristics, these represent ideal starting points to investigate the role of specific genetic risk into models of recovery. Polygenic risk scores (PRS) are one way to investigate the influence of genetic liabilities on complex phenotypes by using additive genetic effect of allele variants across the genome on an outcome of interest (Bogdan et al., 2018; Choi et al., 2020; Wray et al., 2014), here being differential patterns of functioning related to quality of life. To calculate PRS, we create aggregate scores by summing all of the trait-associated alleles each individual carries and weighting that sum by trait-associated allele effect sizes derived from an independent discovery sample (Bogdan et al., 2018; Choi et al., 2020; Wray et al., 2014). The PRS are then carried forward into subsequent analyses to investigate the associations between genetic liabilities and the outcome of interest. In sum, investigating the role of genetic liability towards AUD and externalizing and internalizing characteristics using PRS methods can provide important preliminary insights into the role of genetic influences on patterns of quality of life among those in AUD remission.

Current Study

Recovery is said to be self-evident (Brown & Ashford, 2019; McDaniel et al., 2020); however, individuals post-AUD diagnosis may exhibit heterogeneous patterns of psychosocial functioning and quality of life. To date, only a few studies have examined differential patterns of

quality of life among individuals post-AUD diagnosis (Witkiewitz et al., 2019, 2020). Further, none has incorporated a genetically informative approach. The overall goal of this study was to characterize and predict patterns of functioning related to quality of life among a sample of individuals who remit from AUD using a genetically informed, longitudinal design.

This project had three aims: 1) investigate profiles of functioning related to quality of life among a sample of individuals who remit from AUD; 2a) test whether proximal sources of recovery capital (social support for recovery, attendance at religious services, pathway to recovery) and distal sources of negative recovery capital (deviant peer affiliation during adolescence, interpersonal trauma), associated with profile membership; 2b) test whether epidemiological risk factors for substance use (substance use history, externalizing behaviors, and internalizing symptoms) are associated with profile membership; and 3) test whether genetic liabilities towards alcohol problems, externalizing behaviors, and internalizing behaviors (measured via genome-wide polygenic risk scores) are associated with profile membership. Our hypotheses are outlined below.

1. Based on the small number of studies in this area (e.g., Witkiewitz et al., 2019), we hypothesized that three profiles would emerge. We hypothesized that some individuals in AUD remission would demonstrate high levels of functioning across domains related to quality of life (life satisfaction, health, alcohol and substance use); some would demonstrate low levels of functioning across all domains; and others would demonstrate clustering of improvement across only certain domains.
2. We hypothesized that greater social support, greater frequency of attendance at religious services, 12-step affiliation, and history of professional treatment would be associated with a greater likelihood of membership in profiles characterized by higher functioning

related to quality of life relative to all other profiles. We hypothesized that exposure to interpersonal trauma and more affiliation with deviant peers would be associated with greater likelihood of membership in profiles characterized by lower functioning related to quality of life relative to other profiles.

3. We hypothesized that a history of more alcohol and substance use disorder symptoms, a younger age at first drink, and more externalizing behaviors and internalizing symptoms would be associated with greater likelihood of membership in profiles characterized by lower functioning related to quality of life relative to other profiles.
4. We hypothesized that greater genetic risk for alcohol problems, externalizing behaviors, and internalizing symptoms would be associated with greater likelihood of membership in profiles characterized by lower functioning quality of life, and lower genetic risk would be associated with greater likelihood of membership in profiles characterized by higher functioning related to quality of life.

Method

Sample

Data for the current study came from secondary analysis of the Collaborative Study on the Genetics of Alcoholism (COGA) Prospective Study dataset (N = 3,129). COGA is an interdisciplinary, multi-site project whose overarching goals are to understand the genetic, neurobiological, and environmental factors that contribute to the developmental course of AUD using a well-characterized family-based sample (Begleiter, 1995). COGA ascertained high-risk families through alcohol dependent probands in treatment for AUD, as well as unascertained comparison families across six study sites in the US. In the first 10 years, probands along with all willing first-degree relatives were assessed; recruitment was extended to include additional

relatives in families that contained two or more first-degree relatives with AUD (N = 16,848). Family members from both groups received extensive clinical, behavioral, neuropsychological, neurophysiological, and socio-environmental assessments, providing a rich phenotypic dataset.

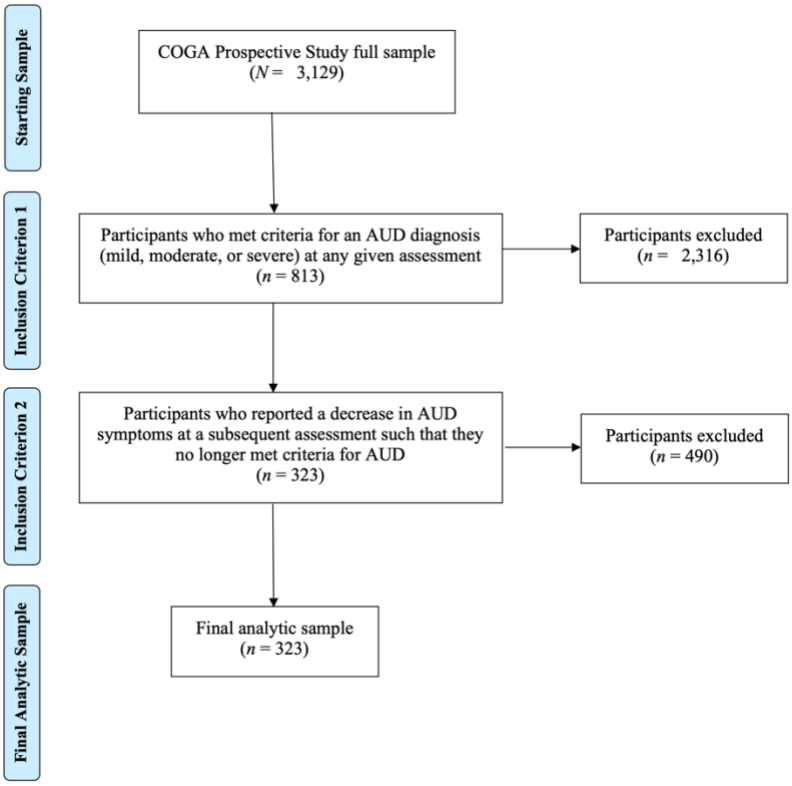
The COGA Prospective Study launched in 2004 with the goal of examining how genetic and environmental risk unfolds across development. As part of the Prospective Study, offspring between ages 12-22 with at least one parent in either the clinically ascertained or unascertained comparison group who had previously completed an interview were recruited to participate and assessed every two years (Bucholz et al., 2017). The COGA Prospective Study sample is thus enriched for risk, with many participants having at least one AUD-affected family member (Bucholz et al., 2017). At each assessment, participants were administered structured interviews, surveys, and other protocols to assess psychiatric and substance use histories, personality measures, and indicators of neurocognitive functioning. Of the Prospective Study sample, 89% completed two or more interviews and 84% have genome-wide association data as of April 2020. Data used in this study were collected between 2005 and 2019. Currently, over 15,000 interviews have been conducted with 3,129 individuals of diverse racial/ethnic backgrounds (primarily European Americans and African Americans).

The analytic sample included individuals from the COGA Prospective Study if they (1) met criteria for an AUD diagnosis (mild, moderate, or severe) at any given assessment and (2) reported a decrease in AUD symptoms at a subsequent assessment such that they no longer met criteria for AUD. Thus, times between assessments included in the present study could vary across participants. Figure 1 shows the process to derive the analytic sample, including the number of participants included and excluded at each stage. Preliminary analyses indicated that

323 individuals met these criteria, 86.69% of whom had genome-wide association data, and were used in analyses.

Figure 1

Analytic Sample Derivation Flow Chart



Note. Abbreviations: COGA = Collaborative Study on the Genetics of Alcoholism; AUD = alcohol use disorder.

Measures

As noted above, participants were enrolled on an ongoing basis and reassessed approximately every two years, meaning participants had varying numbers of follow-up assessments available. The average number of completed assessments for each participant in the COGA Prospective Study sample was 3.54 (*SD* = 1.31, range = 2 - 7). Each measure is described in greater detail below.

Aim 1 Measures

Aim 1 measures came from the first assessment at which each participant no longer met criteria for AUD (i.e., remitted) in order to capture their quality of life in early remission across key areas of psychosocial functioning (life satisfaction, mental and physical health, alcohol and illicit substance use).

Life Satisfaction.

Life Satisfaction and Functioning. Life satisfaction and functioning was measured using the Daily Hassles and Uplifts scale (DeLongis et al., 1982; Kanner et al., 1981). The Daily Hassles and Uplifts scale measures the frequency and intensity of daily positive (uplifts) and negative (hassles) experiences. The measure consists of 53 items, with example items such as spouse, family-related obligations, your friends, fellow workers, your health, and social commitments. Participants were asked to indicate the extent to which each item served as both a hassle and as an uplift over the past week. Reponses for both hassles and uplifts were on a four-point scale, ranging from (0) none to (3) a great deal. Sum scores were calculated separately for daily hassles and daily uplifts. A sum score was calculated for participants who responded to at least 75% of the items; participants who answered fewer than 75% of the items were coded as missing. (Additional details on missing data are below.)

Health.

Physical Health. Physical health was measured using the Semi-Structured Assessment for the Genetics of Alcoholism IV (SSAGA-IV; Bucholz et al., 1994). The SSAGA is a poly-diagnostic psychiatric interview developed for COGA that assesses psychiatric disorders, personality traits, and life experiences (Bucholz et al., 1994). Overall physical health was

assessed via one item. Participants were asked to rate their current physical health on a scale from 5 = *poor* to 1 = *excellent* (Ware & Sherbourne, 1992).

Recent Depressive Episode. Recent depressive episode was measured using the SSAGA (Bucholz et al., 1994). Participants were asked to indicate the most recent age at which they experienced an episode of depression lasting two weeks or longer. Those individuals who indicated they recently experienced (i.e., within the past year) a depressive episode were coded as 1, and those who did not were coded as 0.

Any Recent Anxiety-Related Disorders. The presence of any recent anxiety-related disorder was calculated by creating an index of whether participants recently experienced any of the following anxiety disorders, assessed via the SSAGA (Bucholz et al., 1994): obsessive-compulsive disorder, panic disorder, social phobia, and agoraphobia. For each, participants were asked to indicate the most recent age at which they experienced disorder-related symptoms. Those who indicated they recently experienced (i.e., within the past year) an anxiety-related disorder were coded as 1, and those who did not were coded as 0.

Recent Post-Traumatic Stress Disorder Symptoms. Post-traumatic stress disorder (PTSD) was assessed via the SSAGA (Bucholz et al., 1994). Participants were asked to indicate the most recent age at which they experienced symptoms of PTSD resulting from a traumatic event. Those who indicated they recently experienced (i.e., within the past year) PTSD symptoms were coded as 1, and those who did not were coded as 0.

PTSD was examined separately from other anxiety-related disorders because the DSM-5 re-classified PTSD as a trauma and stressor-related disorder, instead of an anxiety-related disorder (APA, 2013; Miller et al., 2014). Nevertheless, PTSD shares symptomology and features with other anxiety-related disorders, supporting the notion that the two disorders are

related to one another through shared loading onto a higher-order internalizing factor (Krueger et al., 2002). Thus, we are examining PTSD and anxiety-related disorders as distinct dimensions of internalizing characteristics at the time of participants' first AUD remission.

Alcohol and Drug Use.

Frequency of Heavy Episodic Drinking. Frequency of heavy episodic drinking was measured using the SSAGA (Bucholz et al., 1994). Participants reported how frequently they engaged in heavy episodic drinking (i.e., consumed five or more drinks in a 24-hour period) over the past 12 months. Response options ranged from 0 = *never* to 12 = *every day*.

Recent Illicit Substance Use Disorder. A count of the number of illicit substance use disorders for which participants met recent criteria was calculated. In the SSAGA (Bucholz et al., 1994), participants were asked to indicate the most recent age at which they experienced symptoms related to each of the following substance use disorders: cannabis, cocaine, stimulant, sedative, opioid, or other use disorder. Those who indicated they recently experienced (i.e., within the past year) symptoms related to a substance use disorder were coded as 1, and those who did not were coded as 0. The number of disorders for which each participant was coded as 1 was then summed to create a count.

Aim 2A Measures

Proximal Sources of Recovery Capital.

Social Support for Recovery. Social network support for recovery was measured by the Important People and Activities (IPA; Clifford & Longabaugh, 1991, 1992). The IPA was developed to measure social network support of drinking and abstinence (Clifford & Longabaugh, 1991, 1992). This measure was adapted to reflect a continuous measure of social recovery capital, with higher scores indicating higher social recovery capital (SRC-IPA; Francis

et al., 2022). The adapted SRC-IPA is a 7-item measure, with two underlying factors: network abstinence behaviors and basic network structure. Network abstinence behaviors includes items that ask about respondents about their social network's alcohol use (quantity, frequency, drinking status) and support for drinking or abstinence. Basic network structure includes items that ask about respondents' basic social network structure or social connectivity (network size, average contact with network, network diversity). Ordinal response options from the original IPA (e.g., (0) not at all to (7) daily) were recoded to exist on a continuum (e.g., -2 to 2), such that higher scores reflect higher levels of social recovery capital and vice versa. Total scores were calculated by summing across all responses and taking the z-score (Francis et al., 2022). The SRC-IPA had low but acceptable internal consistency ($\alpha = 0.60$), and it showed predictive validity with self-report of peer alcohol and substance use and development of AUD (Francis et al., 2022). Because we conceptualized social support for recovery as a proximal measure of recovery capital, data for this measure came from each participant's first remitted assessment.

Frequency of Attendance at Religious Services. Attendance at religious services was measured using one item from the SSAGA (Bucholz et al., 1994). Participants were asked to indicate the number of times they attended religious services in the past 12 months (Chartier et al., 2016; Meyers et al., 2019). Responses ranged from 0 to 365. Data for this measure came from each participant's first remitted assessment, as we conceptualized this as a proximal source of recovery capital. We acknowledge that attendance at religious services only captures one dimension of religiosity and does not necessarily reflect spirituality; however, there is evidence that attending religious services is associated with improved health, enhanced quality of life, and decreased substance use (Laudet et al., 2006; White & Cloud, 2008).

Treatment. History of treatment was measured by the SSAGA (Bucholz et al., 1994). Participants reported if they were ever treated for a drinking problem. Participants who endorsed receiving treatment were asked to indicate the type of treatment from the following options: "AA or another self-help group," "an outpatient alcohol program," "an outpatient program for something other than alcohol," "an inpatient alcohol program," "when you were an inpatient for medical complications due to alcohol," or "any other place or program." Because participants may have participated in both AA groups and professional treatment, we calculated lifetime (all assessments up to each participant's first remitted assessment) measures for each type of treatment as follows:

Professional Treatment. Participants who endorsed any outpatient, inpatient, or other place/program at any assessment through their first remitted assessment were collapsed into one "professional treatment" category and coded as 1, and those who never endorsed any form of professional treatment were coded as 0. Participants who never received treatment for a drinking problem in the initial screener question were coded as 0 for treatment history.

12-Step Affiliation. Participants who endorsed treatment via AA or another self-help group at any assessment through their first remitted assessment were coded as 1, and those who never endorsed AA treatment were coded as 0. Participants who never received treatment for a drinking problem in the initial screener question were coded as 0 for AA affiliation. Professional treatment was measured by the SSAGA (Bucholz et al., 1994). Participants reported if they were ever treated for a drinking problem. Participants who endorsed receiving treatment were asked to indicate the type of treatment from the following options: "AA or another self-help group," "an outpatient alcohol program," "an outpatient program for something other than alcohol," "an inpatient alcohol program," "when you were an inpatient for medical complications due to

alcohol," or "any other place or program." Because participants may have participated in both AA groups and professional treatment, we calculated lifetime measures for each type of treatment. Participants who endorsed any other form of treatment at any assessment through their first remitted assessment inclusively were collapsed into one "professional treatment" category and coded as 1, and those who never endorsed any form of professional treatment were coded as 0. Participants who never received treatment for a drinking problem in the initial screener question were coded as 0 for treatment history.

Distal Sources of Negative Recovery Capital.

Deviant Peer Affiliation During Adolescence. Affiliation with deviant peers during adolescence was measured by the SSAGA (Bucholz et al., 1994). Participants reported how many of their childhood friends engaged in deviant behaviors, including smoking, drinking alcohol, and using drugs. Response options ranged from (1) *none of them* to (4) *all of them*. Participants over age 18 were asked to think about experiences between ages 12-17 to answer these items. For participants aged 18 or older at the time of their first assessment, data from each participant's first assessment was used to reduce the length of time between the timeframe probed (i.e., ages 12-17) and the time of assessment; for participants under age 18 at the time of their first assessment, data was used from the assessment at which each participant was closest to age 17 to ensure the entire probed timeframe is reflected. Items were summed to create a composite score of peer deviance, with higher scores indicating affiliation with more deviant peers (Kendler et al., 2015; Smith et al., 2019). Participants who responded to at least 50% of the items had calculated scores; the remainder were coded as missing.

Interpersonal Trauma Exposure. Interpersonal trauma exposure was measured by the SSAGA (Bucholz et al., 1994). Participants reported if they were ever exposed to physical or

sexual assault or abuse, which was categorized into nonsexual assaultive trauma and sexual assaultive trauma (Meyers et al., 2019; Subbie-Saenz de Viteri et al., 2020). Nonsexual assaultive trauma was defined as ever having been shot; stabbed; mugged or threated with a weapon, or experienced a break-in or robbery; held captive and/or tortured, and kidnapped. Sexual assaultive trauma was defined as ever having been raped or sexually assaulted. Each category of interpersonal trauma exposure was measured cumulatively, such that the number of different types of nonsexual assaultive traumas ever experienced was summed and the number of different types of sexual assaultive traumas ever experienced was summed to create respective measures of lifetime cumulative interpersonal trauma exposures. The lifetime timeframe was defined as using data from all assessments before each participant's first remitted assessment.

Aim 2B Measures

Substance Use History.

Age at first drink. Age at first drink was measured using the SSAGA (Bucholz et al., 1994). Participants reported the age at which they consumed their first whole drink of alcohol.

Lifetime maximum AUD severity. Lifetime maximum AUD severity was calculated as the maximum number of AUD symptoms endorsed across all assessments before each participant's first remitted assessment, measured via the SSAGA (Bucholz et al., 1994).

Lifetime maximum SUD severity. Lifetime maximum SUD severity was calculated as the maximum number of SUD symptoms across all illicit substances endorsed across all assessments before each participant's first remitted assessment, measured via the SSAGA (Bucholz et al., 1994).

Internalizing Characteristics.

Lifetime maximum depressive symptoms. Lifetime maximum depressive symptoms was assessed via the SSAGA (Bucholz et al., 1994). Lifetime maximum depressive symptoms were calculated as the maximum number of depressive symptoms endorsed across all assessments before each participant's first remitted assessment.

Lifetime maximum anxiety-related disorders. Lifetime maximum anxiety-related disorders, assessed using the SSAGA (Bucholz et al., 1994), was calculated as the maximum number of anxiety-related disorders endorsed across all assessments before each participant's first remitted assessment. Anxiety-related disorders include the following: obsessive-compulsive disorder, panic disorder, social phobia, and agoraphobia.

Lifetime maximum PTSD symptoms. Lifetime maximum PTSD symptoms, assessed via the SSAGA, were measured as the maximum number of symptoms endorsed across all assessments before each participant's first remitted assessment. PTSD was examined separately from other anxiety-related disorders because the DSM-5 re-classified PTSD as a trauma and stressor-related disorder, instead of an anxiety-related disorder (APA, 2013; Miller et al., 2014). Nevertheless, PTSD shares symptomology and features with other anxiety-related disorders, supporting the notion that the two disorders are related to one another through shared loading onto a higher-order internalizing factor (Krueger et al., 2005). Thus, we examined PTSD and anxiety-related disorders as distinct dimensions of internalizing characteristics.

Externalizing Behaviors.

All externalizing behaviors mentioned below were factor analyzed to reduce the data and account for the shared variance in externalizing behaviors accounted for by each of these measures (Dick et al., 2008). The derived factor score was then used as the predictor in Aim 2b analyses.

Sensation seeking. Sensation seeking was assessed via the Sensation Seeking Scale (SSS; Zuckerman, 1994), a 40-item measure with four subscales designed to capture the extent to which individuals seek out stimulation and arousal: thrill and adventure seeking (TAS), experience seeking (ES), disinhibition (Dis), and boredom susceptibility (BS). Items are presented as 40 pairs of statements, such as "I like wild uninhibited parties" and "I prefer quiet parties with good conversation." For each pair, respondents are instructed to circle the statement that best describes their likes or the way they feel. Sensation seeking total scores were calculated by summing respondents' scores across all subscales. The SSS total scores have demonstrated good internal reliability (α range = 0.83 - 0.86) and have good predictive validity (Zuckerman, 1994). Because this measure was only administered to participants once, data came from each participant's first available assessment before their first remitted assessment.

Attention Deficit Hyperactivity Disorder. Attention Deficit Hyperactivity Disorder (ADHD) symptoms were measured using the SSAGA (Bucholz et al., 1994). Participants over age 18 were asked to think their experiences between ages 6-10 to answer items related to ADHD symptoms. As these were retrospective measures for some participants, data from each participant's first assessment was used to reduce the length of time between the timeframe probed and the time of assessment.

Oppositional Defiant Disorder. Oppositional Defiant Disorder (ODD) symptoms were measured using the SSAGA (Bucholz et al., 1994). Participants were instructed to think about experiences as a child or adolescent to answer items related to ODD. As these were retrospective measures for some participants, data from each participant's first assessment was used to reduce the length of time between the timeframe probed and the time of assessment.

Conduct Disorder. Conduct Disorder (CD) symptoms were measured using the SSAGA (Bucholz et al., 1994). Participants were instructed to think about experiences as a child or adolescent to answer items related to CD. As these were retrospective measures for some participants, data from each participant's first assessment was used to reduce the length of time between the timeframe probed and the time of assessment.

Antisocial Personality Disorder. Antisocial Personality Disorder (ASPD) symptoms were measured using the SSAGA (Bucholz et al., 1994). ASPD was measured as the number of symptoms participants endorsed after their 15th birthday. ASPD symptoms were calculated as the maximum number of symptoms endorsed across all assessments before each participant's first remitted assessment.

Aim 3 Measures

DNA samples were genotyped using the Illumina 1M and Illumina OmniExpress (Illumina, San Diego, CA), and Smokescreen (BioRelm, Walnut, CA) arrays. Genotyping was conducted at the Center for Inherited Disease Research, with QC performed locally following standard procedures (Auton et al., 2015). Single nucleotide polymorphisms (SNPs) were imputed to the 1000 genomes phase 3 (1KDGP) reference panel. Genetic data were used to calculate genome-wide polygenic risk scores (PRS) in individuals of European and African genetic ancestry to broadly index genetic liability across the following dimensions: alcohol problems, internalizing, and externalizing. The large-scale, publicly available genome-wide association studies (GWAS) and the methods used for calculating PRS are outlined below. The phenotype domains, discovery GWAS, and GWAS sample characteristics are also summarized in Table 1.

Table 1

Phenotype, Genome-Wide Association Study Discovery Sample, and Sample Size by Ancestry

Domain	Phenotype	Discovery Sample (Reference)	Sample Ancestral Composition (Sample Size)
Alcohol problems	Alcohol use disorder, alcohol problems	MVP, UK Biobank, and PGC meta-analysis GWAS of AUD (Zhou et al., 2020)	European (435,563)
		MVP GWAS of AUD (Kranzler et al., 2019)	African (56,648)
Internalizing	Broad depression, including self-reported depression or probable depression based on depressive symptoms endorsed, clinically diagnosed MDD from self-report and from hospital records, and self-reported help-seeking for problems with nerves, anxiety, tension or depression	PGC, UK Biobank, and 23andme MDD meta-analysis GWAS (excluding the 23andme sample; Howard et al., 2019) and MVP sample from MVP, UK Biobank, and FinnGen MDD meta-analysis GWAS (Levey et al., 2021)	European (750,414)
		MVP, UK Biobank, and FinnGen MDD meta-analysis GWAS (Levey et al., 2021)	African (59,600)
Externalizing	Multivariate externalizing factor including ADHD, problem alcohol use, life time cannabis use, age of first sex, number of lifetime sexual partners, general risk tolerance, and lifetime smoking	Externalizing Consortium GWAS of externalizing behaviors (Karlsson Linnér et al., 2021)	European (1,492,085)
		Pan-UK Biobank risk tolerance GWAS (*Pan UKBB*, 2021)	African (6,636)

Note. Abbreviations: GWAS = genome-wide association study; AUD = alcohol use disorder; MVP = Million Veteran Program; PGC = Psychiatric Genomics Consortium; MDD = Major Depressive Disorder.

Alcohol Problems. For our alcohol problems PRS, we used prioritized SNPs identified in the Million Veteran Program (MVP), UK Biobank, and Psychiatric Genomics Consortium (PGC) meta-analysis GWAS of AUD (Zhou et al., 2020), and the MVP GWAS of AUD (Kranzler et al., 2019). The MVP, UK Biobank, and PGC meta-analysis GWAS of AUD was composed of 435,563 individuals of European ancestry (Zhou et al., 2020). This meta-analysis GWAS was performed using the alcohol use disorder and alcohol problem phenotypes (Zhou et al., 2020). The MVP GWAS of AUD was composed of 56,648 individuals of African ancestry and used the alcohol consumption and alcohol use disorder phenotypes (Kranzler et al., 2019).

Internalizing. For our internalizing PRS, we used prioritized SNPs identified in the Psychiatric Genomics Consortium, UK Biobank, and 23andme major depressive disorder (MDD) meta-analysis GWAS (Howard et al., 2019) and the MVP, UK Biobank, and FinnGen MDD meta-analysis GWAS (Levey et al., 2021). The MDD GWAS was performed using a broad depression phenotype, which included self-reported depression or probable depression based on depressive symptoms endorsed, clinically diagnosed MDD from self-report and from hospital records, and self-reported help-seeking for problems with nerves, anxiety, tension or depression (Levey et al, 2021; Howard et al., 2019). Because the full summary statistics from the MVP, UK Biobank, and FinnGen MDD meta-analysis GWAS (Levey et al., 2021) were not made publicly available as of the time of data analysis, we used METAL to meta-analyze the PGC and UK Biobank MDD meta-analysis GWAS (excluding the 23andme sample; Howard et al., 2019) and the MVP sample from the Levey et al., 2021 MDD meta-analysis GWAS. This resulted in a sample of 750,414 individuals of European ancestry and 59,600 individuals of African ancestry (Levey et al, 2021).

Externalizing. For our externalizing PRS, we used prioritized SNPs identified in the Externalizing Consortium GWAS of externalizing behaviors (Karlsson Linnér et al., 2021) and the Pan-UK Biobank GWAS of risk tolerance (*Pan UKBB*, 2021). The Externalizing Consortium GWAS of externalizing was composed of 1,492,085 individuals of European ancestry (Karlsson Linnér et al., 2021). Genomic structural equation modeling was applied to summary statistics from GWAS on seven externalizing disorders and behaviors: ADHD, problem alcohol use, life time cannabis use, age of first sex, number of lifetime sexual partners, general risk tolerance, and lifetime smoking (Karlsson Linnér et al., 2021). A single latent externalizing factor was identified, and GWAS was performed on this underlying latent externalizing factor. For

individuals of African ancestry, we also incorporated results from the Pan UK Biobank GWAS of risk tolerance, which included 6,636 individuals of African ancestry (*Pan UKBB*, 2021).

PRS Calculation. Each PRS was calculated using the same procedure outlined here. We created the European ancestry PRS using PRS-CS (Ge et al., 2019) and the African ancestry PRS using the PRS-CSx approach (Ruan et al., 2021), which is a variation of the PRS-CS approach (Ge et al., 2019) that has enhanced predictive power in ancestrally diverse populations with under-powered non-European GWAS (Ruan et al., 2021). Instead of specified *p*-value thresholds for the inclusion of SNPs, PRS-CS uses a Bayesian approach to account for LD using information on the correlated SNPs based on an external LD reference panel and summary statistics from a discovery GWAS, which is then used to estimate the posterior effect sizes of genetic variants to the phenotype of interest. PRS-CSx expands upon this approach with its explicit assumption that causal variants for a given phenotype are largely shared across ancestry groups, but the effect sizes of causal variants may vary across groups (Ruan et al., 2021). This latter approach combines the GWAS summary statistics from multiple ancestries the large and mostly European ancestry samples with population-specific allele frequencies and LD patterns to calculate meta-analyzed posterior effect sizes which are then used in PLINK to calculate individual-level polygenic scores in the African ancestry subset of COGA. In both European and African genetic ancestry analyses we allowed PRS-CS to learn the global shrinkage parameter from the data and increased the total number of MCMC iterations and burn-in settings to 10,000 and 5,000 respectively, as doing so shows prediction improvement (Schultz et al., 2022). Genetic analyses were stratified by ancestral group (European and African).

Covariates

Covariates included sex, age at first remitted interview, self-reported race/ethnicity, and time in remission. Sex was coded as male (1) or female (2). Age was measured in years and came from participants' first remitted assessment. The self-identified racial/ethnic composition of our analytic sample was White (n = 225), Black (n = 65), other (n = 30), or Asian (n = 3). Because of the smaller sample sizes of the latter two groups, participants who identified as any other race/ethnicity or as Asian were collapsed into one group. (For genetic analyses, we used genetic principal components to derive each participant's ancestral group. Participants who were of European or African descent, regardless of self-reported race/ethnicity, were included in the genetic analyses. Participants from any other ancestral group were excluded from the Aim 3 analyses.) Time since last AUD diagnosis was measured as the number of years between participants' first remitted assessment and the last assessment at which they met criteria for an AUD diagnosis. Genetic principal components, derived from the genetic data, were included in genetic analyses to control for population stratification. Covariates were examined for association with profile membership, and were included in all generalized linear models (i.e., logistic and linear regressions for Aims 2 and 3).

Analytic Plan

Study hypotheses were pre-registered at https://osf.io/rxznt. Data analysis began with general data cleaning. Variables were examined for expected range and violations from normality (for continuous variables), with transformations being conducted when appropriate; outliers were determined and excluded as necessary (e.g., unreasonably extreme levels of alcohol consumption). Continuous variables that were three or more standard deviations above or below the mean were considered univariate outliers. Multivariate outliers for continuous variables were

determined by calculating Mahalanobis distance, with a threshold of $D^2 < .001$ indicating a multivariate outlier. Data cleaning and preparation was conducted using R (R Core Team, 2019); inferential analyses were conducted using MPlus version 8.4 (Muthén & Muthén, 2000), R, and SPSS version 28 (IBM Corp., 2017).

Aim 1 Analytic Plan

We used finite mixture modeling to characterize differential profiles of functioning related to quality of life among individuals who reduced their symptoms such that they no longer met criteria for AUD. Profiles were defined by Aim 1 indicators described above, which correspond to aspects of quality of life (life satisfaction, health, and alcohol and illicit substance use), to empirically determine the patterns of shared response between them. We fit one- through five-class solutions with maximum likelihood estimation with robust standard errors to account for missing and non-normal data. The number of classes that provide the best fit to the data were determined based on indices of model fit (e.g., Lo Mendell Rubin [LMR] Likelihood Ratio test, Akaike's Information Criteria [AIC], Bayesian Information Criterion [BIC], and sample-size-adjusted BIC [ssBIC]), entropy, and interpretability of the final solution (Muthén & Muthén, 2000; Nylund et al., 2007). The AIC, BIC, and ssBIC are indicators of goodness of fit of the models to the data that account for the parsimony of the model (i.e., number of estimated parameters). The LMR likelihood ratio test assesses the hypothesis that a *k*-1 class model fits the data better than a *k*-class model, with a non-significant *p*-value (> .05) suggesting that the model with one fewer class fits the data better. Entropy provides a measure of the degree to which the classes are clustered to each other, with values > .80 indicating good separation distinguishing the classes (Celeux & Soromenho, 1996). (We note that we use the terms class and profiles interchangeably.)

After identification of the best-fitting solution, each profile was examined and described, including the number of people per class and general demographics (i.e., age, sex, race/ethnicity). We used the resulting profile structure from these analyses to compare differences between profiles in average scores across domains of quality of life used to determine these profiles. Additionally, posterior probabilities of profile membership were derived from the solution and used to assign participants to profiles. Importantly, assigned profile membership is probabilistic, so using a hard partition of assigned profile membership can result in loss of power and/or biased estimates resulting from misclassification in profile assignment. This concern poses less of an issue when entropy is high (i.e., > .80; Clark and Muthen, 2009; entropy for the selected profile solution in the present study was .76). To address possible difference between results based on assigned versus probabilistic profile membership, we examined both assigned and probabilistic profile membership as outcomes in subsequent analyses.

Aims 2A and 2B Analytic Plan

After classifying participants into profiles for Aim 1 we conducted chi-square tests (for binary indicators) and *t*-tests (for continuous indicators) to examine whether the two profiles significantly differed on each predictor. Next, we conducted two sets of analyses to examine the joint influence of covariates and predictors on profile membership when modeled simultaneously. In the first set of analyses, we conducted a linear regression to test whether the covariates and predictors were associated with probability of profile membership. In the second set of analyses, all covariates and predictors were entered into a binary logistic regression model to test their joint influence on assigned profile membership (e.g., profile 1 versus 2).

Aim 3 Analytic Plan

We used analyses similar to those described above to test whether genetic liability towards alcohol problems, externalizing characteristics, and internalizing symptoms, measured via PRS, predicted assigned and probabilistic profile membership. Probability of membership in each profile identified in Aim 1 was regressed onto each PRS in a one set of analyses, and assigned membership in each of the profiles was regressed onto each PRS in a second set of analyses. Due to variation in allele frequency for different ancestry groups (Márquez-Luna et al., 2017; Peterson et al., 2017), all analyses involving genetic information were analyzed separately for European and African ancestral groups, which reflect the two largest groups in COGA. Genetic principal components were included to control for population stratification. The association between each PRS (alcohol problems, internalizing symptoms, and externalizing characteristics) and profile membership (both assigned and probabilistic) initially was tested in separate models, with significant PRS entered simultaneously into a final model to examine their joint influence on profile membership.

Missing Data

We note that there were substantial levels of missingness (36.84%) on the Daily Hassles and Uplifts scale, an indicator of life satisfaction and functioning in our latent profile analysis. (All other variables demonstrated low to adequate missingness [i.e., < 12.08%].) The level of missingness on this measure is likely attributable to the fact that participants were asked to complete the survey and mail it back in, whereas most other variables used in the current study were assessed via the SSAGA, a semi-structured assessment administered by an interviewer. To determine whether any of our study variables were associated with missing data on the Daily Hassles and Uplifts scale, we conducted a preliminary binary logistic regression. Completion

status was regressed onto all other Aim 1 indicators and Aim 2 predictors (or comparable variables), as well as sex and race/ethnicity. We reverse coded completion status, such that 0 = *completed* and 1 = *missing*, so odds ratios from the model were interpreted as participants' likelihood of having missing data on this measure. Results from this model are presented in Table 2. None of the variables were associated with the likelihood of missing data on the Daily Hassles and Uplifts scale. To further ensure that our data were missing completely at random (MCAR), we conducted Little's MCAR test on all LPA indicators included in Aim 1 analyses. Little's MCAR was not statistically significant ($\chi^2(24) = 19.10$, $p = .749$), indicating that our indicators were likely MCAR.

Table 2

Associations Between Key Constructs and Completion Status of Daily Hassles and Uplifts Measure

	OR	*Beta*	SE	*p*
Intercept	**0.57**	**-0.56**	**0.26**	**0.034**
Sex (0 = Male)	0.88	-0.12	0.30	0.681
Race (0 = White)				
Black	1.15	0.14	0.38	0.723
Other	1.29	0.26	0.44	0.561
Interpersonal trauma exposure	0.84	-0.17	0.31	0.575
Professional treatment history	1.08	0.08	0.90	0.933
12-step affiliation	0.65	-0.43	1.00	0.667
Any anxiety-related disorder (at remittance)	0.99	-0.01	0.52	0.987
Any anxiety-related disorder (at max AUD)	1.19	0.18	0.56	0.755
Any illicit substance use disorder	0.68	-0.39	0.43	0.375
Age at first drink	0.89	-0.12	0.15	0.441
Deviant peer affiliation during adolescence	1.01	0.01	0.15	0.953
Depression symptoms (at max AUD)	0.89	-0.11	0.17	0.502
Depression symptoms (at remittance)	0.99	-0.01	0.17	0.955
Physical health	0.93	-0.07	0.14	0.604
Frequency of church attendance	1.26	0.23	0.16	0.163
Frequency of heavy episodic drinking	1.03	0.03	0.14	0.852

Sensation seeking	0.88	-0.13	0.15	0.398
Social support for recovery	0.82	-0.20	0.15	0.184
Maximum AUD symptoms	0.85	-0.16	0.17	0.342
Observations		266		

Note. **Bold type** indicates $p < .05$. Completion status of the Daily Hassles and Uplifts measure was reverse coded, such that 0 = *completed* and 1 = *missing*, so odds ratios from the model can be interpreted as participants' likelihood of having missing data on this measure. Analyses were conducted using non-imputed data.

In the current study, we addressed missing data in two ways: full information maximum likelihood estimation (FIML) and multiple imputation. FIML is one of the most commonly used methods for treating missing data in latent profile analyses, particularly when data are not missing completely at random (Lanza & Cooper, 2016; Spurk et al., 2020). Thus, we used this approach to address missing data when conducting our latent profile analysis (Aim 1). However, FIML is not suitable to address missingness among exogenous variables (Lanza & Cooper, 2016), so multiple imputation was used prior to address missing data in our predictor variables from Aims 2A and 2B.

Prior to multiple imputation of the exogenous variables (i.e., predictor variables for Aims 2A and 2B), we conducted Little's MCAR test. Little's MCAR was not statistically significant ($\chi^2(50) = 64.04$, $p = .088$), indicating that our predictors were likely MCAR. The only Aim 2 predictor variables with missing data were sensation seeking (2.79%) and social support for recovery (12.07%). Multiple imputation with five imputed datasets were created, and pooled estimates are reported for inferential analyses involving the imputed variables.

Sensitivity Analyses

We ran a series of sensitivity analyses in order to examine the robustness of our results. First, we ran a set of sensitivity analyses comparing our pattern of findings to those observed when excluding individuals who met criteria for any SUD at the time of their first remitted assessment. This allowed us to investigate whether patterns of functioning related to quality of

life differ when using a more stringent threshold of remission from all alcohol and substance use disorders. Next, as multiple imputation is based on pooled parameter estimates from multiple copies of the dataset which are then combined to produce a final dataset with no missing data, we ran a set of sensitivity analyses to determine whether the data imputation process affected our pattern of results. After running Aim 2 analyses using the imputed dataset, we reran our analyses using the unimputed dataset and compared our results. This allowed us to investigate the robustness of our results and ensure that our pattern of findings was not significantly altered by the inclusion of imputed data.

Results

Preliminary Analyses

Descriptive Statistics, Zero-Order Correlations, and Data Cleaning

The analytic sample was 45.20% female and 54.80% male. The sample was majority White (69.66%), followed by 20.12% Black/African American and 10.22% other racial/ethnic group. Participants' average age at their first remitted assessment was 24.72 years (*SD* = 3.28), and they had an average of 2.84 years (*SD* = 1.75) in remission. Descriptive statistics for all study constructs are presented in Table 3, and zero-order correlations are presented in Table 4. These analyses were conducted using the raw (i.e., non-transformed), non-imputed data. We examined variables for expected range and violations from normality (for continuous variables), with transformations being conducted when appropriate; outliers were determined and excluded as necessary (e.g., unreasonably extreme levels of alcohol consumption). Based on the descriptive statistics, we transformed/collapsed the following variables: recent illicit substance use disorder (Aim 1 measure), both cumulative trauma variables, and religious service attendance (Aim 2A measures).

First, we noted limited variance for our recent illicit substance use disorder indicator and our cumulative trauma predictors. For recent illicit substance use disorders, we noted that only two participants (0.6%) indicated they recently experienced symptoms related to two or more disorders (92.3% experienced none, and 7.1% experienced recent symptoms for one disorder). Thus, we collapsed this count variable into a dichotomous one where 0 = *no recent illicit substance use disorder* and 1 = *recent symptoms for at least one illicit substance use disorder*. Approximately 7.7% reported experiencing recent symptoms of an illicit SUD at the time of their first AUD remission. Similarly, only 3.7% of participants reported more than one type of sexual assaultive trauma, and 5.6% of participants endorsed more than one type of nonsexual assaultive trauma. Since the majority of the variance for these variables could be captured in a binary variable, we dichotomized both trauma variables, such that 0 = *no history of sexual/nonsexual trauma* and 1 = *history of sexual/nonsexual trauma*. Approximately 15.8% of individuals had a history of sexual assaultive trauma, and 26.0% of participants had a history of nonsexual assaultive trauma (4.95% had a history of both). Lastly, frequency of religious service attendance was highly skewed and kurtotic (8.35 and 88.04, respectively), so we log-transformed this variable after adding a constant of 1 for individuals who attended religious services zero times (log +1). (We note that we attempted to transform lifetime cumulative anxiety-related disorders, but the skew worsened after transforming. Thus, we retained our original variable.) All transformed variables were used in inferential analyses.

Next, we examined univariate and multivariate outliers. We identified the following outliers, defined as three standard deviations above or below the mean: two cases with outliers on age of first drink (< 8 years old), two cases with outliers on lifetime maximum PTSD symptoms (> 15 symptoms), 8 cases with outliers on lifetime maximum AUD symptoms (> 8

symptoms), and 7 cases with outliers for maximum CD symptoms (> 6 symptoms). Because none of these values seemed like coding errors or unreasonable values for the analytic sample, we opted to retain these values (Aguinis et al., 2013)[1]. Using Mahalinobis' distance with a threshold of $D^2 < .001$, we identified five multivariate outliers (1.55% of the sample). Multivariate outliers are sensitive to univariate outliers, and our theory-driven decision to retain the univariate outliers above may have contributed to the multivariate outliers. Further, we felt it was defensible to retain the multivariate outliers given 1) our sample size of 323, 2) the fact that the outliers were not very extreme and constituted less than 2% of the sample, and 3) the fact that multivariate analyses tend to be more robust to violations of normality (Cohen et al., 2003).

Power Analyses

Given our small sample size of individuals of racial/ethnic backgrounds other than White, we conducted a priori power analyses using G*Power 3.1 (Faul et al., 2009) to estimate statistical power for the genetic analyses. Table 5 presents results for varying effect sizes expressed as R^2, or the explained variance in the outcome due to the varying effect size of a PRS predictor. This range of values shows that when analyzing the model separated by ancestral group, there was adequate power to estimate genetic effects explaining >3% of variance (R^2 = .03) for the EA group. PRS based on the largest GWAS for alcohol problems, internalizing, and externalizing phenotypes, detailed above, currently account for 1-10% of the variance in independent samples. Although AA samples remain underpowered (Martin et al., 2017), the implementation of the PRS-CSx may help improve predictive power (Ruan et al., 2021). Importantly, we believe it is an ethical imperative to include underrepresented groups in research

[1] Although we decided to retain all univariate and multivariate outliers, we ran sensitivity analyses in which we winsorized all univariate outliers and reran all *t*-tests and binary logistic regressions. The results observed from analyses using the winsorized variables paralleled the results we obtained in our primary analyses using the non-winsorized variables.

Table 3

Descriptive Statistics for Study Constructs

Variable	*M (SD)* or %	Range	*N*	Skew, Kurtosis
Aim 1				
Daily hassles	39.91 (20.98)	0 – 117	204	0.90, 0.91
Daily uplifts	60.98 (27.03)	6 – 152	204	0.78, 0.50
Physical health (5 = poor, 1 = excellent)	2.27 (0.96)	1 – 5	323	0.39, -0.42
Frequency of heavy episodic drinking	5.18 (3.03)	0 – 12	323	-0.16, -1.08
Recent depressive episode	3.40%	0 – 1	323	-
Any recent anxiety-related disorder symptoms	5.30%	0 – 1	323	-
Recent Post-Traumatic Stress Disorder symptoms	2.50%	0 – 1	323	-
Any recent illicit SUD	7.70%	0 – 1	323	-
Aim 2A				
Social support for recovery	-1.51 (3.65)	-20.12 – 11.08	287	0.01, 2.56
Frequency of religious service attendance	10.22 (29.92)	0 – 365	323	8.27, 86.11
12-Step affiliation	4.00%	0 – 1	323	-
Professional treatment history	4.30%	0 – 1	323	-
Deviant peer affiliation during adolescence	4.06 (2.58)	0 – 12	323	0.64, 0.23
Cumulative sexual assaultive traumas	0.23 (0.65)	0 – 4	323	3.88, 17.54
Cumulative nonsexual assaultive traumas	0.32 (0.60)	0 – 3	323	1.87, 3.13
Aim 2B				
Age at first drink	14.99 (2.36)	6 – 21	323	-0.33, 1.11
Lifetime maximum AUD symptoms	3.20 (1.60)	2 – 10	323	1.86, 3.67
Lifetime maximum SUD symptoms	1.36 (2.23)	0 – 7	323	1.42, 0.55
Lifetime maximum Major Depressive Disorder symptoms	3.58 (3.62)	0 – 9	323	0.22, -1.69
Cumulative lifetime anxiety-related disorders	3.87 (0.50)	1 – 4	323	-4.44, 20.50
Lifetime maximum Post-Traumatic Stress Disorder symptoms	2.28 (4.35)	0 – 16	323	1.76, 1.75
Sensation seeking score	20.28 (6.24)	5 – 36	314	-0.10, 0.35
Attention Deficit Hyperactivity Disorder symptoms	4.84 (5.35)	0 – 18	323	1.06, -0.05
Oppositional Defiant Disorder symptoms	1.85 (2.09)	0 – 8	323	0.95, -0.10
Conduct Disorder symptoms	1.51 (1.62)	0 – 9	323	1.55, 2.92
Antisocial Personality Disorder symptoms	2.67 (1.85)	0 – 7	323	0.52, -0.62

Note. Abbreviations: *M* = Mean; *SD* = Standard deviation; AUD = alcohol use disorder; SUD = substance use disorder. Means and standard deviations are presented for continuous variables, and percentages endorsed are presented for binary variables. Data presented here were based on non-imputed data. “At remitted assessment” indicates the measure was taken from the assessment at

which each participant first did not meet diagnostic criteria for alcohol use disorder (i.e., remitted). A sum score was calculated for participants who answered at least 75% of items on the Daily Hassles and Uplifts scale, and for those who answered at least 50% of the peer deviance items.

Table 4

Zero-Order Correlations for Study Constructs

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1. Sex	--																											
2. Race/ Ethnicity	-0.04	--																										
3. Age at first remitted assessment	0.06	0.05	--																									
4. Time in remission	0.05	0.00	***0.44***	--																								
5. Difference in Hassles - Uplifts	0.04	***-0.18***	-0.08	-0.01	--																							
6. Frequency of heavy episodic drinking	***-0.15***	-0.05	-0.10	-0.09	0.04	--																						
7. Physical health (5 = poor, 1 = excellent)	**0.14**	***0.17***	***0.19***	***0.15***	0.06	***-0.17***	--																					
8. Recent Post-Traumatic Stress Disorder symptoms	0.10	-0.04	0.07	0.02	-0.04	0.02	**0.12**	--																				
9. Any recent anxiety-related disorder symptoms	***0.23***	-0.04	0.01	0.01	0.04	-0.07	**0.14**	**0.14**	--																			
10. Recent depressive episode	0.07	-0.06	0.08	0.08	0.00	-0.05	0.02	0.08	0.11	--																		
11. Any recent illicit SUD	-0.03	-0.05	-0.06	-0.09	0.05	0.02	0.05	-0.05	0.04	0.07	--																	
12. Social support for recovery	0.06	**0.12**	-0.03	0.04	-0.01	-0.09	0.07	**-0.14**	-0.01	0.06	0.09	--																
13. Frequency of religious service attendance	-0.04	0.07	0.00	-0.01	***-0.19***	-0.02	-0.05	0.09	-0.01	-0.03	**-0.13**	**0.12**	--															
14. 12-Step affiliation	0.000	-0.01	**0.14**	**0.11**	-0.05	-0.03	0.09	***0.17***	0.02	-0.04	-0.06	0.04	0.07	--														
15. Professional treatment history	-0.07	0.01	0.05	0.07	-0.09	-0.08	0.08	***0.16***	0.02	0.04	-0.01	0.03	0.10	***0.65***	--													
16. Deviant peer affiliation during adolescence	-0.02	0.03	0.02	0.09	-0.01	0.10	0.07	0.06	0.01	0.00	0.10	**-0.14**	-0.03	***0.18***	***0.24***	--												
17. Cumulative sexual assaultive traumas	***0.26***	-0.01	0.08	0.05	0.04	**-0.12**	0.11	0.10	***0.22***	***0.20***	-0.01	0.05	-0.03	-0.05	-0.03	-0.02	--											
18. Cumulative nonsexual assaultive traumas	***-0.24***	**0.13**	0.02	0.03	-0.11	0.01	0.10	***0.15***	-0.06	-0.02	-0.04	-0.06	**-0.12**	**0.13**	0.06	***0.16***	0.06	--										
19. Age at first drink	-0.01	0.10	***0.15***	-0.05	-0.09	-0.07	-0.01	-0.01	0.01	0.02	-0.10	0.04	-0.03	**-0.11**	***-0.20***	***-0.36***	-0.08	**-0.11**	--									
20. Lifetime maximum AUD symptoms	0.02	0.00	0.05	-0.02	0.00	0.00	0.02	-0.02	-0.03	-0.01	**0.12**	0.00	0.01	**0.14**	***0.21***	***0.26***	0.00	0.04	***-0.25***	--								
21. Lifetime maximum SUD symptoms	-0.10	0.03	***0.27***	***0.18***	0.02	-0.01	0.11	0.07	0.03	***0.15***	0.09	-0.02	-0.01	***0.17***	***0.18***	***0.33***	0.09	***0.23***	**-0.13**	***0.19***	--							
22. Lifetime maximum Major Depressive Disorder symptoms	***0.29***	0.00	***0.17***	**0.12**	**0.17**	***-0.21***	***0.26***	***0.16***	**0.14**	***0.17***	-0.07	-0.04	-0.08	0.10	0.08	***0.15***	***0.28***	0.08	-0.09	***0.15***	***0.27***	--						
23. Cumulative lifetime anxiety-related disorders	***0.19***	0.03	***0.24***	0.09	-0.03	**-0.14**	***0.22***	***0.31***	***0.21***	0.05	-0.08	-0.05	-0.01	0.07	0.02	0.06	***0.31***	***0.18***	-0.01	0.09	***0.15***	***0.36***	--					
24. Lifetime maximum Post-Traumatic Stress Disorder symptoms	***-0.26***	***-0.22***	-0.05	***0.15***	0.11	0.11	-0.05	-0.04	-0.07	-0.05	0.02	***-0.18***	***-0.17***	0.07	0.07	***0.16***	-0.03	**0.11**	***-0.18***	**0.13**	0.05	-0.01	**-0.13**	--				
25. Sensation seeking	-0.08	0.04	0.03	0.04	0.03	-0.05	**0.13**	0.05	0.10	0.00	-0.02	0.01	-0.08	0.06	0.01	***0.14***	0.08	***0.25***	-0.05	***0.18***	**0.13**	***0.30***	***0.29***	**0.12**	--			
26. Attention Deficit Hyperactivity Disorder symptoms	0.05	0.02	0.06	-0.02	0.00	-0.04	***0.16***	0.11	0.05	**0.12**	-0.04	0.08	-0.06	0.03	0.06	***0.15***	***0.15***	***0.25***	-0.11	**0.14**	***0.27***	***0.40***	***0.29***	-0.02	***0.60***	--		
27. Oppositional Defiant Disorder symptoms	***-0.18***	0.06	0.11	0.06	0.03	-0.03	0.07	0.05	-0.10	-0.02	0.10	0.01	-0.03	0.01	0.06	***0.34***	-0.03	***0.33***	***-0.26***	***0.27***	***0.38***	***0.22***	***0.20***	**0.12**	***0.38***	***0.45***	--	
28. Conduct Disorder symptoms	***-0.22***	***0.15***	***0.26***	***0.16***	-0.02	-0.05	***0.15***	0.07	-0.05	0.03	0.05	-0.02	-0.06	***0.20***	***0.21***	***0.38***	0.06	***0.40***	***-0.23***	***0.36***	***0.47***	***0.28***	***0.23***	***0.15***	***0.38***	***0.48***	***0.66***	--

Note. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

to establish a foundation of research in diverse populations. Further, since no studies have examined whether genetic influences contribute to heterogeneity in quality of life among individuals who reduce their AUD symptoms, the potential contribution to the literature is significant.

Table 5

Estimated Power by Sample Size and R^2 for Genetic Analyses

	European American Ancestry	**African American Ancestry**
R^2	**Power**	**Power**
.005	0.18	0.09
.01	0.32	0.12
.02	0.56	0.20
.03	0.73	0.28
.04	0.85	0.35
.05	0.92	0.42
	N = 225	N = 65

Note. Estimated power by sample size and R^2 was determined using G*Power 3.1. Sample size for ancestral groups was estimated using participants' self-reported race/ethnicity and does not reflect their genetically derived ancestral background.

Aim 1: To Investigate Profiles of Functioning Related to Quality of Life Among a Sample of Individuals Who Remit From AUD

Latent Profile Analysis and Class Enumeration

Latent profile models were fit to the data using the indicators outlined above. Of note, we determined that the average hassles and average uplifts scores were difficult to meaningfully interpret when entered separately in the model. To that end, we calculated the difference between hassles and uplifts for each participant and used the resulting score as our indicator of life satisfaction and functioning. A higher score (i.e., larger difference) can be understood as poorer functioning whereby participants endorsed each item as a more hassling (negative) experience than an uplifting (positive) one. As part of the class enumeration process, we fit the model with one- through five-class models and compared which solution best fit the data (see Table 6). We

compared model fit for each solution and determined the best fitting model as the most parsimonious, most well-separated, and most interpretable.

Table 6

Model Fit Comparisons for One- Through Five-Class Models

		Parsimony Criteria			Clustering Criteria			
Classes	LL	AIC	BIC	ssBIC	Entropy	Likelihood ratio test	LMR *p*-value	BLRT *p*-value
1	-2479.50	4979.01	5016.78	4985.07	-	-	-	-
2	**-2446.84**	**4929.67**	**4997.67**	**4940.57**	**0.761**	2 vs. 1 class	< **.001**	< **.001**
3	-2438.17	4928.33	5026.55	4944.08	0.715	3 vs. 2 classes	0.1239	0.192
4	Model did not converge					4 vs. 3 classes	-	-
5	Model did not converge					5 vs. 4 classes	-	-

Note. Abbreviations. LL = loglikelihood; BIC = Bayesian information criterion; ssBIC = sample size adjusted BIC; BLRT *p*-value = *p*-value of the bootstrapped likelihood ratio test; LMR *p*-value = *p*-value of the adjusted Lo–Mendell–Rubin likelihood ratio test. **Bold** type indicates selected solution.

Fit indices indicated that a 2-class solution fit the data best, given the lowest values for BIC and ssBIC, as well as the higher entropy of the model. Further, the LMR likelihood ratio test and the BLRT comparing a 2-class to a 1-class solution was significant, suggesting that a 2-class model fit the data significantly better than a 1-class model. Neither the LMR likelihood ratio test nor the BLRT comparing a 3-class to a 2-class solution was significant, suggesting that a 3-class model did not represent a substantial improvement in fit. Despite increasing the number of random starts (up to 5,000 random starts), neither a 4-class nor a 5-class solution converged on a loglikelihood value. The results from these solutions are thus not trustworthy due to potential issues surrounding spurious local maxima or local solutions and were thus not considered further. Moreover, failure to converge on a loglikelihood value despite a high number of random starts indicates that the specified model cannot be identified and the maximum number of

profiles have been estimated (Lanza, 2016). Importantly, simulation studies suggest that the ssBIC, LMR, BLRT are the best information criteria for identifying the true number of classes (Nylund et al., 2007; Tofighi & Enders, 2007), and these information criteria all supported a 2-class solution.

Class Interpretation

Mean values for the continuous indicators and endorsement rates for the dichotomous indicators are shown in Table 7. Figure 1 shows scores on the continuous indicators by profile, and Figure 2 shows the probability of endorsing dichotomous indicators by profile. Raw values are presented in the table for interpretation and contextualization, while standardized values are shown in the figure because of scale differences between the indicators. The first profile included 39.9% (n = 129) of the sample, and the second profile included 60.1% (n = 194) of the sample.

Primary differences between the profiles included sex, frequency of HED, and physical health. Profile 1 had a greater proportion of females compared to profile 2 (54.3% vs. 39.2%; $\chi^2(1) = 6.53$, $p = .011$). Profile 1 reported lower HED than profile 2 ($M_{diff} = -5.08$, $t(321) = -19.19$, $p < .001$). The mean frequency of HED for profile 1 was 2.21 (SD = 0.20), where 2 corresponds to a response option of engaging in HED between 3-5 days per year and 6-11 days per year over the past 12 months. The mean frequency of HED for profile 2 was 7.29 (SD = 0.17), where 7 corresponds to engaging in HED 1-2 days per week over the past 12 months. Lastly, although individuals in both profiles reported good physical health, individuals in profile 1 reported slightly worse physical health than those in profile 2 ($M_{diff} = 0.39$, $t(321) = 3.30$, $p = .001$). On average, profile 1 reported a score of 2.50 (SD = 0.10) and profile 2 reported a score of 2.11 (SD = 0.07), where a score of 2 corresponds to very good health and a score of 3 corresponds to good health. Although not statistically significant, a greater proportion of

individuals in profile 1 relative to profile 2 reported recent anxiety-related disorder symptoms and recent depressive episodes. We thus characterized profile 1 as the "infrequent alcohol use group, good health" group, and profile 2 as the "frequent alcohol use, good to very good health" group. There were no other statistically significant differences between the profiles in terms of demographics (race, age at first remission, or time since last AUD diagnosis [measured as time between participants' last affected and first remitted assessments]) or profile indicators (all *p*s > .334; see Table 7).

Sensitivity Analyses

Because the focus of this project was on individuals in remission from AUD and to retain the largest sample size possible, we did not exclude individuals who met recent criteria for an illicit substance use disorder from the analytic sample. However, there is some debate as to whether individuals who reduce their use of one substance may substitute with another (Blanco et al., 2014; Sussman & Black, 2008). To that end, we ran a set of sensitivity analyses comparing our pattern of findings to those observed when excluding individuals who met criteria for any illicit SUD at the time of their first remitted assessment. In sensitivity analyses, we excluded 25 individuals who endorsed criteria for one (n = 23) or more (n = 2; two individuals endorsed criteria for two SUDs) SUDs at the time of their first AUD remission.

We then refit one- through five-class solutions. In these models, we dropped recent illicit substance use as an indicator because we limited the sensitivity subsample to exclude anyone who endorsed this variable. All other indicators and model parameters were unchanged. The pattern of results paralleled that which we observed using the full analytic sample, suggesting that a 2-class model fit the data best. Results from these sensitivity analyses are shown in Table 8. Because we largely observed the same pattern of results, we retained the model and class

Table 7

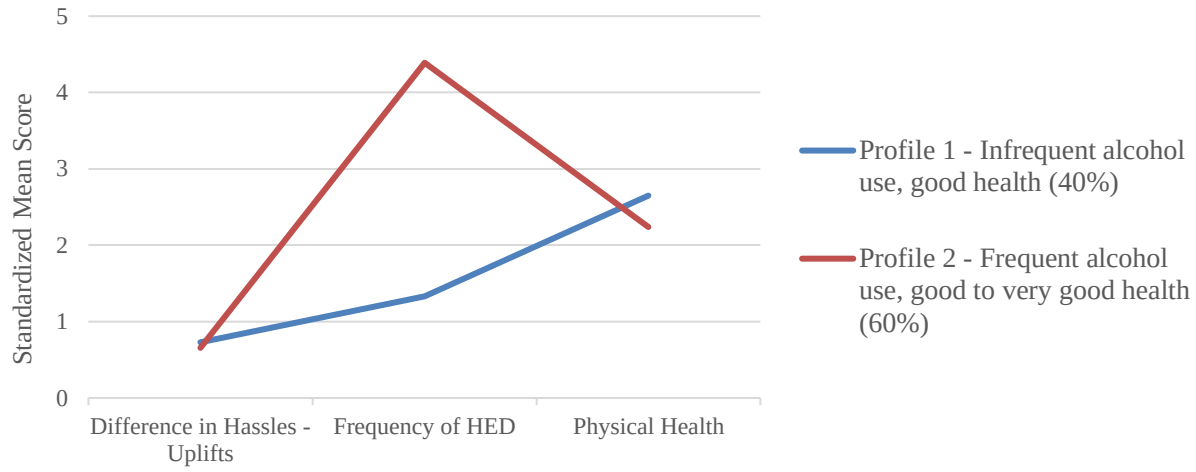
Descriptive Statistics and Differences Across Identified Profiles

	Profile 1 – infrequent alcohol use, good health	**Profile 2 – frequent alcohol use, good to very good health**	χ^2 / ***t*-test statistic**
	***M* (*SD*)**	***M* (*SD*)**	
Demographics			
Sex (% female)	54.26%	39.18%	**χ^2(1) = 6.53, *p* = .011**
Race (% White)	63.57%	73.71%	χ^2(1) = 5.30, *p* = .071
% Black/African American	26.36%	15.98%	
% Other	10.08%	10.31%	
Age at first remit	25.12 (3.64)	24.44 (3.00)	*t*(237.32) = 1.83, *p* = .068
Time since last AUD diagnosis	3.04 (2.15)	2.70 (1.42)	*t*(202.14) = 1.57, *p* = .118
Profile Indicators			
Difference between hassles and uplifts	-22.42 (3.78)	-20.18 (2.86)	*t*(321) = -.480, *p* = .632
Frequency of heavy episodic drinking (0 = never, 2 = 3-5 days/year, …, 7 = 1 day/week, 8 = 2 days/week, …, 12 = ever day)	***2.21 (0.20)***	***7.29 (0.17)***	***t(321) = -19.19, p < .001***
Physical health (1 = excellent, 2 = very good, 3 = good, 4 = fair, 5 = poor)	***2.50 (0.10)***	***2.11 (0.07)***	***t(321) = 3.30, p = .001***
Recent PTSD symptoms	2.5% (1.4)	2.5% (1.2)	*t*(321) = 0.013, *p* = .990
Recent anxiety-related disorder symptoms	7.6% (2.4)	3.7% (1.4)	*t*(321) = -1.32, *p* = .186
Recent depressive episode	4.6% (1.9)	2.6% (1.2)	*t*(321) = -0.92, *p* = .357
Recent illicit SUD	7.5% (2.6)	7.9% (2.1)	*t*(321) = 0.15, *p* = .882
Observations (%)	129 (39.9%)	194 (60.1%)	-

Note. Abbreviations: PTSD = Post-Traumatic Stress Disorder; SUD = Substance Use Disorder. Mean and standard error values shown for the profile indicators are from the data using FIML to handle missing values. Raw values (unstandardized) are presented in the table for interpretation and contextualization. *T*-tests for dichotomous indicators were conducted using logit values. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

Figure 2

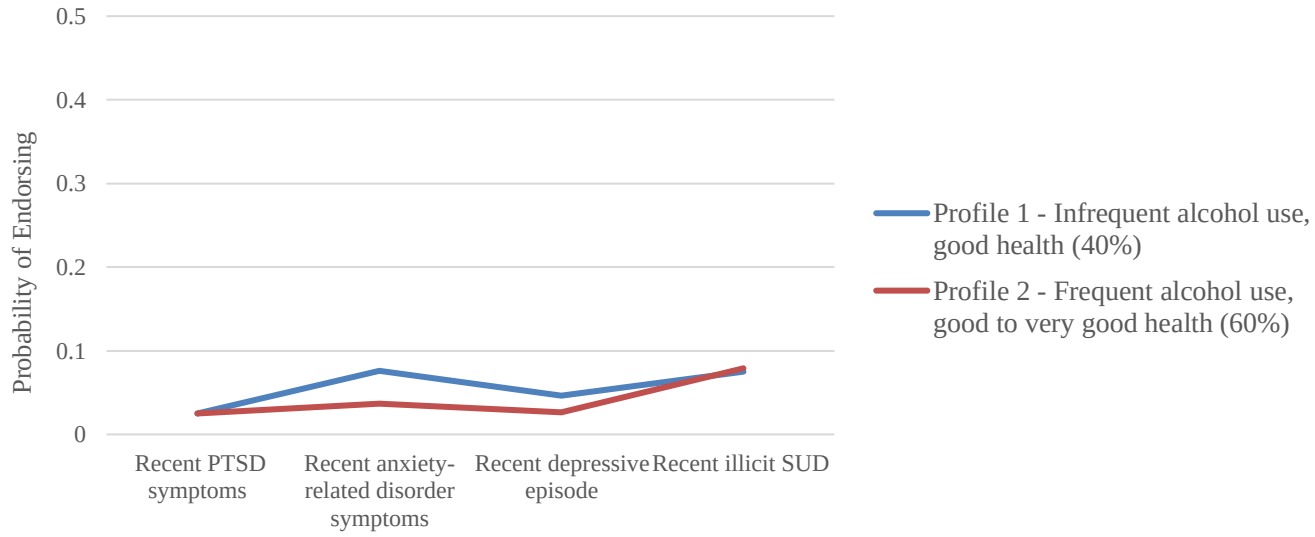
Standardized Scores for the Continuous Quality of Life Indicators by Profile Membership



Note. Abbreviations: HED = heavy episodic drinking. The variables are scaled such that higher mean scores indicate worse functioning.

Figure 3

Probability of Endorsing Each of the Dichotomous Quality of Life Indicators by Profile Membership



Note. Abbreviations: PTSD = Post-Traumatic Stress Disorder; SUD = Substance Use Disorder. Probability of endorsing ranges from 0 to 1; the scale shown here is truncated to better show the (nonsignificant) differences between the profiles.

Table 8

Sensitivity Analyses Excluding Participants Who Endorsed Any Recent Symptoms of an Illicit Substance Use Disorder

		Parsimony Criteria			Clustering Criteria			
Classes	LL	AIC	BIC	ssBIC	Entropy	Likelihood ratio test	LMR *p*-value	BLRT *p*-value
1	-2163.62	4345.24	4378.51	4349.97	-	-	-	-
2	**-2133.39**	**4298.78**	**4357.93**	**4307.19**	**0.775**	**2 vs. 1 class**	**< .001**	**< .001**
3	-2126.14	4298.29	4383.32	4310.38	0.800	3 vs. 2 classes	0.155	0.333
4	-2119.04	4298.08	4408.99	4313.85	0.713	4 vs. 3 classes	0.552	0.500
5	Model did not converge					5 vs. 4 classes	-	-

Note. Abbreviations. LL = loglikelihood; BIC = Bayesian information criterion; ssBIC = sample size adjusted BIC; BLRT *p*-value = *p*-value of the bootstrapped likelihood ratio test; LMR *p*-value = *p*-value of the adjusted Lo–Mendell–Rubin likelihood ratio test. The sample used for these analyses represents 298 individuals who did not endorse any recent symptoms of an illicit substance use disorder at the time of their first alcohol use disorder remission ($n_{excluded}$ = 25). The loglikelihood was unable to be replicated for the 5-class model, despite using 1000 random starts. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

probabilities for the full analytic sample (including the 25 individuals who endorsed recent symptoms for one or more SUDs at the time of first AUD remission) for all subsequent analyses.

Aim 2

Aim 2A: Test Whether Sources of Recovery Capital are Associated with Profile Membership

First, we examined proximal sources of recovery capital and distal sources of negative recovery capital as a function of assigned profile membership by examining descriptive statistics and conducting *t*-tests for continuous variables and chi-square tests for categorical variables.

Results from these univariate models are shown in Table 9. Individuals in profile 2 ("frequent alcohol use, good to very good health" profile) reported significantly lower levels of social support for their recovery (M_{diff} = 0.94; $t(281) = 2.11$, $p = .036$, Cohen's $d = 0.26$) and were less likely to report a history of sexual assaultive trauma ($\chi^2(1) = 4.27$, $p = .044$, Cohen's $d = 0.23$) relative to those in profile 1 ("infrequent alcohol use, good health" profile). Those in profile 2 also reported significantly higher levels of affiliation with deviant peers during adolescence compared to those in the profile 1 (M_{diff} = 0.27; $t(321) = -2.00$, $p = .047$, Cohen's $d = 0.23$). The two profiles did not differ in terms of frequency of religious service attendance, 12-step affiliation, history of professional treatment, or lifetime physical assault (all *p*s > .257).

Aim 2B: Test Whether Epidemiological Risk Factors for Substance Use are Associated with Profile Membership

We also examined epidemiological risk factors for substance use as a function of assigned profile membership by examining descriptive statistics and conducting *t*-tests for continuous variables and chi-square tests for categorical variables. Results from these univariate models are shown in Table 9. Individuals in profile 1 ("infrequent alcohol use, good health" profile) had higher lifetime maximum depressive (M_{diff} = 1.16; $t(321) = 2.85$, $p = .005$, Cohen's $d = 0.32$) and PTSD (M_{diff} = 1.09; $t(230.02) = 2.12$, $p = .035$, Cohen's $d = 0.25$) symptoms compared to profile 2 ("frequent alcohol use, good to very good health" profile). None of the substance use history variables (age at first drink, maximum AUD symptoms, maximum SUD symptoms) or the other internalizing variables (anxiety-related disorders) varied by profile (all *p*s > .288).

For our externalizing variables (ADHD, ODD, CD, ASPD, sensation seeking), we first tested for differences in the observed variables between assigned profiles. Individuals in profile 2

scored higher on sensation seeking than those in profile 1 (M_{diff} = -1.46; $t(8581)$ = -2.03, p = .042, Cohen's d = 0.23). None of the other observed externalizing variables were significantly different between profiles (all ps > .260; see Table 9).

Table 9

Comparison of Predictor Variables Across the Identified Profiles

Predictor Variable	**Profile 1 – infrequent alcohol use, good health**	**Profile 2 – frequent alcohol use, good to very good health**	χ^2 / ***t*-test statistic**	**Cohen's *d***
	M (*SD*) or %	*M* (*SD*) or %		
Aim 2A Predictors				
Social support for recovery	**-0.96 (0.34)**	**-1.90 (0.27)**	***t*(281) = 2.11, *p* = .036**	**0.26**
Religious service attendance (log+1)	0.55 (0.63)	0.54 (0.67)	$t(321) = 0.10$, $p = .922$	
12-step affiliation	2.33%	5.15%	$\chi^2(1) = 1.61$, $p = .257$	
History of professional treatment	3.88%	4.64%	$\chi^2(1) = 0.11$, $p = 1.00$	
Peer deviance during adolescence	**3.71 (2.59)**	**4.29 (2.56)**	***t*(321) = -2.00, *p* = .047**	**0.23**
Lifetime sexual assault	**20.93%**	**12.37%**	**$\chi^2(1) = 4.27$, *p* = .044**	**0.23**
Lifetime physical assault	24.81%	26.80%	$\chi^2(1) = 0.16$, $p = .700$	
Aim 2B Predictors				
Age at first drink	15.16 (2.77)	14.88 (2.03)	$t(271.43) = 0.96$, $p = .337$	
Lifetime maximum AUD symptoms	3.09 (1.61)	3.28 (1.59)	$t(321) = -1.07$, $p = .288$	
Lifetime maximum SUD symptoms	1.36 (2.34)	1.36 (2.15)	$t(321) = -0.02$, $p = .987$	
Lifetime maximum depressive symptoms	**4.28 (3.62)**	**3.12 (3.55)**	***t*(321) = 2.85, *p* = .005**	**0.32**
Lifetime maximum PTSD symptoms	**2.94 (4.92)**	**1.85 (3.89)**	***t*(230.02) = 2.12, *p* = .035**	**0.25**
Cumulative lifetime anxiety-related disorders	3.84 (0.53)	3.89 (0.48)	$t(321) = -0.96$, $p = .338$	
Observed externalizing variables				
Lifetime maximum ADHD symptoms	4.91 (5.40)	4.79 (5.33)	$t(321) = 0.20$, $p = .843$	
Lifetime maximum ODD symptoms	1.92 (2.16)	1.80 (2.04)	$t(321) = 0.50$, $p = .618$	
Lifetime maximum CD symptoms	1.64 (1.77)	1.43 (1.52)	$t(321) = 1.13$, $p = .260$	
Lifetime maximum ASPD symptoms	2.74 (1.91)	2.61 (1.80)	$t(321) = 0.62$, $p = .533$	
Latent externalizing factor	0.05 (0.94)	-0.04 (0.87)	$t(321) = 0.86$, $p = .388$	
Sensation seeking	**19.40 (0.59)**	**20.86 (0.44)**	***t*(8581) = -2.03, *p* = .042**	**0.23**

Note. Sensation seeking and social support for recovery used data from the pooled multiple imputations because those variables had missing data. The standard error mean, not the standard deviation, is shown for the imputed variables. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

Next, we used factor analysis with maximum likelihood estimation to reduce the data and account for the shared variance in externalizing behaviors accounted for by each of our observed externalizing variables (see Table 10; Dick et al., 2008). The latent factor was standardized. The initial model demonstrated fair to adequate fit, $\chi^2(5) = 74.44$, SRMR = 0.08, TLI = 0.67, CFI = 0.84. All of the observed externalizing variables, except sensation seeking (.14), significantly loaded onto the underlying latent factor (all other loadings > .56). Cronbach's alpha was adequate ($\alpha = .71$). We thus cut sensation seeking from the factor model based on poor factor loading (sensation seeking was entered into the logistic regression model as an observed variable) and refit the model. This resulted in 4-item model, yielding improved model fit, $\chi^2(2) = 61.97$, SRMR = 0.07, TLI = 0.58, CFI = 0.86. All retained items demonstrated good factor loadings (> .57) and the trimmed scale demonstrated good reliability ($\alpha = .79$). The latent factor accounted for 49.13% of the shared variance. We then conducted a *t*-test to determine whether the profiles differed on their externalizing factor score, but did not find evidence to suggest a significant difference ($p = .725$).

Table 10

Factor Loadings for Externalizing Latent Factor

	Model 1		**Model 2**	
Variable	Beta	SE	Beta	SE
Externalizing Factor				
Lifetime maximum ADHD symptoms	***0.56***	***0.31***	***0.58***	***0.33***
Lifetime maximum ODD symptoms	***0.65***	***0.12***	***0.67***	***0.12***
Lifetime maximum CD symptoms	***0.76***	***0.09***	***0.76***	***0.09***
Lifetime maximum ASPD symptoms	***0.79***	***0.10***	***0.78***	***0.10***
Sensation seeking	**0.14**	**0.39**	-	-

Note. Abbreviations: ADHD = Attention Deficit Hyperactivity Disorder; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; ASPD = Antisocial Personality Disorder. ***Bold italic*** type indicates $p < .001$. **Bold** type indicates $p < .05$.

Finally, we used linear regression to examine the joint influence of all covariates and predictors on probability of membership in each profile. Analyses were conducted using the pooled imputed datasets, and we examined the joint association of all covariates and predictors modeled simultaneously. Results are shown in Table 11. In this model, longer time since last AUD diagnosis (b = 0.03, 95% CI: [0.00, 0.06]) and higher lifetime maximum MDD symptoms (b = 0.02, 95% CI: [0.00, 0.03]) were associated with a higher probability of being in profile 1 (and associated with a lower probability of being in profile 2, with parameters being the inverse of those for profile 1). This model accounted for 7.0% of the variance in probability of profile membership (adjusted R^2 = 0.07). No other predictors were significantly associated with probability of profile membership.

We also conducted binary logistic regressions in which we examined the joint associations between predictors and assigned profile membership. Profile 1, the "infrequent alcohol use, good health" profile, was set as our reference group, and analyses were conducted using the pooled imputed datasets. In the first model, we included all predictors that were significant in the prior analyses (social support for recovery, deviant peer affiliation during adolescence, sensation seeking, lifetime maximum MDD symptoms, lifetime maximum PTSD symptoms, sexual assaultive trauma) and all covariates (race, sex, and time since last AUD diagnosis [i.e., time between participants' last affected and first remitted assessments]) into a model to examine their association with assigned profile membership. Results are shown in Table 12, model 1. Individuals who affiliated with more deviate peers during adolescence were more likely to be in profile 2 than profile 1 (OR_{adj} = 1.11, 95% CI: [1.01, 1.22]). No other predictors were uniquely associated with assigned profile membership (all ps > .072). This model accounted for approximately 11.3% of the variance in assigned profile membership

(Nagelkerke's R^2 = .11). In the second model (see Table 12, model 2), we added the remaining predictors to the model. After adjusting for the remaining predictors, affiliation with deviant peers during adolescence was no longer significant (OR_{adj} = 1.10, 95% CI: [0.98, 1.23]). The second model accounted for 13.6% of the variance in assigned profile membership (Nagelkerke's R^2 = .14).

Table 11

Linear Regressions to Examine Joint Influence of Predictors on Profile Membership Probability

	Profile 1 Membership Probability		**Profile 2 Membership Probability**	
	b	**95% CI**	***b***	**95% CI**
Race	0.04	[-0.03, 0.12]	-0.04	[-0.12, 0.03]
Sex	0.10	[-0.01, 0.21]	-0.10	[-0.21, 0.01]
Time since last AUD diagnosis	**0.03**	**[0.00, 0.06]**	**-0.03**	**[-0.06, 0.00]**
Social support for recovery	0.10	[-0.01, 0.02]	-0.01	[-0.02, 0.01]
Peer deviance during adolescence	-0.02	[-0.04, 0.00]	0.02	[-0.00, 0.04]
Lifetime maximum depressive symptoms	**0.02**	**[0.00, 0.03]**	**-0.02**	**[-0.03, -0.00]**
Sensation seeking	-0.00	[-0.01, 0.01]	0.00	[-0.01, 0.01]
Lifetime sexual assault	0.06	[-0.08, 0.20]	-0.06	[-0.20, 0.08]
Lifetime maximum PTSD symptoms	0.00	[-0.01, 0.02]	-0.00	[-0.02, 0.01]
Age at first drink	0.01	[-0.01, 0.03]	-0.01	[-0.03, 0.01]
12-step affiliation	-0.26	[-0.57, 0.06]	0.26	[-0.06, 0.57]
History of professional treatment	0.29	[-0.02, 0.60]	-0.29	[-0.60, 0.02]
Lifetime physical assault	-0.01	[-0.13, 0.11]	0.01	[-0.11, 0.13]
Lifetime cumulative anxiety disorders	-0.03	[-0.13, 0.07]	0.03	[-0.07, 0.13]
Lifetime maximum AUD symptoms	-0.01	[-0.05, 0.02]	0.01	[-0.02, 0.05]
Lifetime maximum SUD symptoms	-0.01	[-0.03, 0.02]	0.01	[-0.02, 0.03]
Religious service attendance (log+1)	0.01	[-0.08, 0.09]	-0.01	[-0.09, 0.08]
Externalizing latent factor	0.05	[-0.03, 0.12]	-0.05	[-0.12, 0.03]
Adjusted R^2	0.07		0.07	

Note. Abbreviations: CI = Confidence interval; AUD = Alcohol use disorder; SUD = Substance use disorder. Models were conducted using pooled multiple imputations. The unstandardized regression weights are presented here because we used the pooled multiple imputations. The adjusted R^2 represents the average of the imputed parameters. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

Table 12

Logistic Regressions to Examine Joint Influence of Predictors Using Imputed Data

	Model 1		Model 2	
	OR	**95% CI**	***OR***	**95% CI**
Race	0.81	[0.57, 1.16]	0.83	[0.58, 1.20]
Sex	0.72	[0.43, 1.20]	0.63	[0.36, 1.12]
Time since last AUD diagnosis	0.89	[0.78, 1.02]	0.89	[0.77, 1.03]
Social support for recovery	0.95	[0.89, 1.02]	0.95	[0.89, 1.02]
Peer deviance during adolescence	**1.11**	**[1.01, 1.22]**	1.10	[0.98, 1.23]
Lifetime maximum depressive symptoms	0.94	[0.87, 1.01]	0.92	[0.87, 1.02]
Sensation seeking	1.02	[0.98, 1.06]	1.02	[0.97, 1.06]
Lifetime sexual assault	0.77	[0.39, 1.53]	0.80	[0.40, 1.61]
Lifetime maximum PTSD symptoms	0.98	[0.65, 11.51]	0.98	[0.92, 1.04]
Age at first drink			0.97	[0.87, 1.09]
12-step affiliation			4.73	[0.78, 28.57]
History of professional treatment			0.33	[0.06, 1.71]
Lifetime physical assault			1.90	[0.58, 2.06]
Lifetime cumulative anxiety disorders			0.97	[0.59, 1.60]
Lifetime maximum AUD symptoms			1.10	[0.93, 1.30]
Lifetime maximum SUD symptoms			1.02	[0.90, 1.16]
Religious service attendance (log+1)			0.97	[0.64, 1.47]
Externalizing latent factor			4.19	[0.16, 112.51]
Nagelkerke's R^2	0.113		0.136	

Note. Abbreviations: OR = Odds ratio; CI = Confidence interval; AUD = Alcohol use disorder; SUD = Substance use disorder. Model 1 included all covariates and only the predictors that emerged as significant in univariate *t*-tests and χ^2 tests. Model 2 included all covariates and all predictors, regardless of a significant association with profile membership in univariate *t*-tests and χ^2 tests. Models were conducted using pooled multiple imputations. Nagelkerke's R^2 represents the average of the imputed parameters. Profile 1 was set as the reference group. ORs less than 1 indicating a higher likelihood of being assigned to profile 1 and ORs greater than 1 indicating a higher likelihood of being assigned to profile 2. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

Sensitivity Analyses

As multiple imputation is based off of pooled parameter estimates from multiple copies of the dataset which are then combined to produce a final dataset with no missing data, we ran a set of sensitivity analyses to determine whether the data imputation process affected our pattern of results for Aim 2 (see Table 13). We largely observed the same pattern of results. Individuals who affiliated with more deviate peers during adolescence were more likely to be in profile 2 than profile 1 (OR_{adj} = 1.12, 95% CI: [1.01, 1.23]) even when accounting for social support for

recovery, sensation seeking, lifetime maximum MDD symptoms, lifetime maximum PTSD symptoms, sexual assaultive trauma, and all covariates (race, sex, and time since last AUD diagnosis). In contrast to our analyses using imputed data where this effect was attenuated when adjusted for all remaining predictors, affiliation with deviate peers during adolescence continued to exert a unique influence on assigned profile membership when using the non-imputed data (OR_{adj} = 1.14, 95% CI: [1.01, 1.28]). These minor differences in results between analyses using the imputed and non-imputed datasets are likely due to the fact that our imputed variables (sensation seeking and social support for recovery) either share underlying variance with deviant peer affiliation (sensation seeking) or negate the effects of deviant peer affiliation (social support for recovery). In light of these modest differences and the risk of a false positive (Type I error) using the non-imputed dataset, we opted to retain the original results presented above.

Table 13

Sensitivity Analyses Logistic Regressions to Examine Joint Influence of Predictors Using Non-Imputed Data

	Model 1		**Model 2**	
	OR	**95% CI**	***OR***	**95% CI**
Race	0.86	[0.59, 1.26]	0.83	[0.56, 1.24]
Sex	0.69	[0.40, 1.20]	0.58	[0.31, 1.07]
Time since last AUD diagnosis	0.86	[0.73, 1.00]	0.86	[0.73, 1.01]
Social support for recovery	0.95	[0.89, 1.02]	0.95	[0.89, 1.03]
Peer deviance during adolescence	**1.12**	**[1.01, 1.23]**	**1.14**	**[1.01, 1.28]**
Lifetime maximum depressive symptoms	0.96	[0.88, 1.03]	0.97	[0.89, 1.06]
Sensation seeking	1.03	[0.98, 1.08]	1.03	[0.98, 1.08]
Lifetime sexual assault	0.55	[0.27, 1.15]	0.57	[0.27, 1.22]
Lifetime maximum PTSD symptoms	0.99	[0.93, 1.06]	1.00	[0.93, 1.07]
Age at first drink			0.98	[0.87, 1.10]
Ever AA			2.93	[0.46, 18.72]
Ever professional treatment			0.39	[0.07, 2.17]
Cumulative lifetime physical assault			1.23	[0.62, 2.45]
Lifetime cumulative anxiety disorders			1.27	[0.69, 2.31]
Lifetime maximum AUD symptoms			1.12	[0.94, 1.34]

Lifetime maximum SUD symptoms		1.00	[0.87, 1.15]
Religious service attendance (log+1)		0.91	[0.58, 1.42]
Externalizing latent factor		0.72	[0.49, 1.06]
Nagelkerke's R^2	0.125	0.151	

Note. Abbreviations: OR = Odds ratio; CI = Confidence interval; AUD = Alcohol use disorder; SUD = Substance use disorder. Model 1 includes all covariates and only the predictors that emerged as significant in univariate *t*-tests and χ^2 tests. Model 2 includes all covariates and all predictors, regardless of significant association with profile membership in univariate *t*-tests and χ^2 tests. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$. $N = 280$.

Aim 3: Test Whether Genetic Liabilities Towards Alcohol Problems, Externalizing Behaviors, and Internalizing Behaviors are Associated with Profile Membership

Alcohol Problems

To examine genetic liability towards alcohol problems as a function of profile membership, we conducted linear regressions wherein we regressed probabilistic profile membership onto the alcohol problems PRS, all 10 ancestral PCs, sex, and time since last AUD diagnosis. We ran models separately for individuals of European ancestry and of African ancestry, which was determined based on ancestral PCs. Results from these models are shown in Table 14. Being female (β = 0.23, 95% CI: [0.12, 0.34]) and longer time since last AUD diagnosis (β = 0.08, 95% CI: [0.02, 0.14]) were associated with higher probability of being in profile 1 and associated with lower probability of being in profile 2 in the EA model. None of the PRS were significantly associated with probability of profile membership for either ancestral group.

We also conducted binary logistic regressions in which we regressed assigned profile membership onto the PRS and all covariates. We observed the same pattern of results as observed in models using probabilistic profile membership. Results from these models are shown in Table 15, with ORs less than 1 indicating a higher likelihood of being in profile 1 and ORs greater than 1 indicating a higher likelihood of being in profile 2. Sex (OR_{adj} = 0.35, 95% CI:

[0.18, 0.65]) and time since last AUD diagnosis (OR_{adj} = 0.69, 95% CI: [0.48, 0.95]) were significantly associated with assigned profile membership in the EA model, such that being female and having longer time since last AUD diagnosis, was associated with an increased likelihood of assignment to profile 1. The alcohol problems PRS did not significantly predict profile membership in either group (*p*s > .305).

Internalizing Characteristics

Next, to examine genetic liability towards internalizing characteristics as a function of profile membership, we conducted linear regressions wherein we regressed probabilistic profile membership onto the internalizing PRS, all 10 ancestral PCs, sex, and time since last AUD diagnosis. Again, we ran models separately for individuals of European ancestry and of African ancestry, which was determined based on ancestral PCs. Being female (β = 0.24, 95% CI: [0.12, 0.35]) and longer time since last AUD diagnosis (β = 0.08, 95% CI: [0.03, 0.14]) were associated with higher probability of being in profile 1 and associated with lower probability of being in profile 2 for the EA group (see Table 14). None of the PRS were significantly associated with probability of profile membership for either ancestral group.

We also conducted binary logistic regressions in which we regressed assigned profile membership onto the PRS and all covariates. We observed the same pattern of results as observed in models using probabilistic profile membership. Results are shown in Table 15. Sex (OR_{adj} = 0.33, 95% CI: [0.17, 0.63]) and time since last AUD diagnosis (OR_{adj} = 0.68, 95% CI: [0.47, 0.94]) were significantly associated with assigned profile membership in the EA model. Being female and having longer time since last AUD diagnosis was associated with an increased

likelihood of assignment to profile 1. The internalizing PRS did not significantly predict profile membership in either group (*p*s > .182).

Externalizing Behaviors

Lastly, to examine genetic liability towards externalizing behaviors as a function of profile membership, we conducted linear regressions wherein we regressed probabilistic profile membership onto the externalizing PRS, all 10 ancestral PCs, sex, and time since last AUD diagnosis. The models were run separately for individuals of European ancestry and of African ancestry. Table 14 shows the results from these models. Among those in the EA group, being female (β = 0.23, 95% CI: [0.12, 0.35]) and longer time since last AUD diagnosis (β = 0.08, 95% CI: [0.02, 0.14]) were associated with higher probability of being in profile 1 and associated with lower probability of being in profile 2. None of the PRS were significantly associated with probability of profile membership for either ancestral group.

We also conducted binary logistic regressions in which we regressed assigned profile membership onto the PRS and all covariates. We observed the same pattern of results as observed in models using probabilistic profile membership. Results are shown in Table 15. Sex (OR_{adj} = 0.34, 95% CI: [0.18, 0.64]) and time since last AUD diagnosis (OR_{adj} = 0.69, 95% CI: [0.48, 0.96]) were significantly associated with assigned profile membership in the EA model, and time since last AUD diagnosis (OR_{adj} = 1.91, 95% CI: [1.06, 3.95]) was associated with assigned profile membership in the AA model. In the EA model, being female and having longer time since last AUD diagnosis was associated with an increased likelihood of assignment to profile 1. In the AA model, longer time since last AUD diagnosis was associated with an increased likelihood of assignment to profile 2. The externalizing PRS did not significantly predict assigned profile membership in either group (*p*s > .258).

Table 14

Linear Regressions to Examine PRS as Predictors of Profile Membership Probability

	European ancestry (*n* = 206)				**African ancestry (*n* = 74)**			
	Profile 1 Membership Probability		**Profile 2 Membership Probability**		**Profile 1 Membership Probability**		**Profile 2 Membership Probability**	
	β	**95% CI**	**β**	**95% CI**	**β**	**95% CI**	**β**	**95% CI**
Alcohol Problems PRS								
Sex (0 = Male)	***0.23***	***[0.12, 0.34]***	***-0.23***	***[-0.34, -0.12]***	0.10	[-0.14, 0.33]	-0.10	[-0.33, 0.14]
Time since last AUD diagnosis	***0.08***	***[0.02, 0.14]***	***-0.08***	***[-0.14, -0.02]***	-0.10	[-0.22, 0.03]	0.10	[-0.03, 0.22]
Alc-PRS	-0.02	-0.08, 0.03	0.02	-0.03, 0.08	-0.06	[-0.17, 0.06]	0.06	[-0.06, 0.17]
Adjusted R^2		0.167		0.167		0.179		0.179
Internalizing PRS								
Sex (0 = Male)	***0.24***	***[0.12, 0.35]***	***-0.24***	***[-0.35, -0.12]***	0.05	[-0.18, 0.28]	-0.05	[-0.28, 0.18]
Time since last AUD diagnosis	***0.08***	***[0.03, 0.14]***	***-0.08***	***[-0.14, -0.03]***	-0.09	[-0.22, 0.03]	0.09	[-0.03, 0.22]
Int-PRS	0.04	-0.01, 0.10	-0.04	-0.10, 0.01	0.06	[-0.06, 0.19]	-0.06	[-0.19, 0.06]
Adjusted R^2		0.174		0.174		0.180		0.180
Externalizing PRS								
Sex (0 = Male)	***0.23***	***[0.12, 0.35]***	***-0.23***	-0.35, -0.12	0.07	[-0.16, 0.30]	-0.07	[-0.30, 0.16]
Time since last AUD diagnosis	***0.08***	***[0.02, 0.14]***	***-0.08***	***[-0.14, -0.02]***	-0.11	[-0.23, 0.02]	0.11	[-0.02, 0.23]
Ext-PRS	0.03	-0.03, 0.09	-0.03	-0.09, 0.03	0.04	[-0.10, 0.18]	-0.04	[-0.18, 0.10]
Adjusted R^2		0.169		0.169		0.170		0.170

Note. Abbreviations: CI = Confidence interval; PRS = Polygenic risk score. The first 10 genetic principal components were included in the model to account for population stratification, but are not shown here. All variables were standardized. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

Table 15

Logistic Regressions Examining PRS as Predictors of Assigned Profile Membership

	European ancestry (*n* = 206)		**African ancestry (*n* = 74)**	
	OR	**95% CI**	***OR***	**95% CI**
Alcohol Problems PRS				
Sex (0 = Male)	***0.35***	***[0.18, 0.65]***	0.74	[0.23, 2.26]
Time since last AUD diagnosis	**0.69**	**[0.48, 0.95]**	1.81	[1.02, 3.65]
Alc-PRS	1.14	[0.84, 1.55]	1.34	[0.78, 2.42]
Nagelkerke's R^2	0.197		0.270	
Internalizing PRS				

Sex (0 = Male)	***0.33***	***[0.17, 0.63]***	0.96	[0.32, 2.86]
Time since last AUD diagnosis	**0.68**	**[0.47, 0.94]**	1.83	[1.01, 3.74]
Int-PRS	0.80	[0.57, 1.11]	0.77	[0.41, 1.40]
Nagelkerke's R^2	0.203		0.264	
Externalizing PRS				
Sex (0 = Male)	***0.34***	***[0.18, 0.64]***	0.85	[0.29, 2.49]
Time since last AUD diagnosis	**0.69**	**[0.48, 0.96]**	**1.91**	**[1.06, 3.95]**
Ext-PRS	0.82	[0.59, 1.15]	0.81	[0.42, 1.53]
Nagelkerke's R^2	0.200		0.260	

Note. Abbreviations: *OR* = Odds ratio; CI = Confidence interval; PRS = Polygenic risk score. Profile 1 was set as the reference group. ORs less than 1 indicating a higher likelihood of being assigned to profile 1 and ORs greater than 1 indicating a higher likelihood of being assigned to profile 2. The first 10 genetic principal components were included in the model to account for population stratification, but are not shown here. All variables were standardized. ***Bold italic*** type indicates $p < .01$. **Bold** type indicates $p < .05$.

Discussion

The goal of this study was to examine and predict patterns of functioning related to quality of life in a sample of individuals in AUD remission. Literatures related to recovery capital and epidemiological and genetic risk factors for substance use were integrated to inform three research questions: 1) Do individuals who remit from AUD exhibit differential profiles of functioning related to quality of life?; 2a) Are proximal sources of recovery capital (social support for recovery, extrinsic religiosity, pathway to recovery), and distal sources of negative recovery capital (deviant peer affiliation during adolescence, interpersonal trauma), associated with profile membership?; 2b) Are epidemiological risk factors (substance use history, externalizing behaviors, and internalizing symptoms) associated with profile membership?; and 3) Are genetic liabilities towards alcohol problems, externalizing behaviors, and internalizing behaviors (measured via genome-wide polygenic risk scores) associated with profile membership? We discuss our findings related to each research question in turn below and then zoom out for a global discussion of findings that highlights implications and contextualizes these

results. Lastly, we conclude by discussing the limitations of the present study and suggestions for future research.

Heterogeneous Patterns of Functioning Related to Quality of Life in AUD Remission

Our first hypothesis was somewhat supported. As hypothesized, we found evidence to suggest subgroups of individuals with heterogeneous patterns of functioning related to quality of life in our sample. Contrary to the hypothesized three-profile solution, we found that a two-profile solution best fit the data, with individuals in each profile demonstrating improvement across some domains of quality of life but not others. Approximately 40% of participants were assigned to the first "infrequent alcohol use, good health" profile, and 60% were assigned to the second "frequent alcohol use, good to very good health" profile. The two profiles were significantly different with respect to sex of participants, frequency of heavy episodic drinking, and self-reported physical health. Specifically, individuals in profile 1 were more likely to be female and reported less alcohol use and slightly worse (but still good) physical health than individuals in profile 2.

Our findings are consistent with and contribute to the growing body of evidence (e.g., Witkiewitz et al., 2019, 2020) suggesting that individuals demonstrate differential patterns of functioning when in AUD remission. Similar work by Witkiewitz and colleagues (2019, 2020) found evidence for four profiles characterized by infrequent non-heavy drinking/high functioning, frequent heavy drinking/low functioning, infrequent heavy drinking/low functioning, and occasional heavy drinking/high functioning. We note that we observed less heterogeneity in our sample relative to that in Witkiewitz's sample, but this may be due to several differences between the two samples. Specifically, our sample relative to Witkiewitz's was smaller (323 versus 806, respectively), younger (M_{age} = 24.7 versus 38.1), and had less

severe histories of alcohol use (measured via AUD symptoms on the SSAGA versus severity of dependence on the Alcohol Dependence Scale), had fewer individuals with a history of substance use treatment (4.33% versus 100%), and/or captured a different timeframe in participants' recovery journeys (average of 2.84 years in remission versus three years post-AUD treatment). Another important note is that we do not have information from Witkiewitz and colleagues about whether or not their sample had attempted and/or completed AUD treatment multiple times before participating in the parent study, whereas in the current study, we examined quality of life at individuals' first known remitted assessment. This may be influential, as psychosocial functioning tends to improve with increased time in self-reported recovery (Kelly et al., 2017). Nevertheless, findings from both studies support the conclusion that some individuals in remission may report high quality of life despite frequent heavy drinking.

In considering our profiles within the context of extant literature, individuals in profile 1 ("infrequent alcohol use, good health") may be those who are likely to consider themselves in recovery or have actively sought to resolve their AUD. Prior research suggests that individuals often exhibit high stress levels and low functioning during the early stages of self-reported recovery (Kelly et al., 2018), and psychosocial functioning and health tend to improve with time (Kelly & Hoeppner, 2015; Laudet et al., 2006; Laudet & White, 2008). The average time since last AUD diagnosis among those in profile 1 was approximately three years, which generally constitutes early remission (Kelly et al., 2018). Thus, we might expect that profile 1 individuals' psychosocial functioning in remission might improve over time.

In contrast, those in profile 2 ("frequent alcohol use, good to very good health"), may be individuals who matured out of AUD (Lee & Sher, 2018) and would be less likely to consider themselves in recovery. Contrary to evidence that continual engagement in heavy substance use

is associated with a variety of health risks (Kelly & Bergman, 2021), individuals in this profile reported better health than those in profile 1. However, given the young age of the sample (M_{age} = 24.72), this may reflect health conditions that have yet to be realized. They also reported fewer mental health challenges (recent depressive episode or recent anxiety-related disorder) despite having less time in recovery (approximately 2.75 years) than those in profile 1. In sum, this profile may constitute individuals who would not traditionally be categorized as "in recovery."

Within the context of the evolving definition of recovery to center the whole person, our findings underscore two important considerations. First, our findings underscore the need to consider individuals' self-reported recovery status and/or recovery identity (Dingle et al., 2015). When collapsing everyone who remits from AUD into one category, there is substantial heterogeneity in functioning. However, it would be worth considering whether the same types of heterogeneity in functioning are observed among a sample of individuals who have all adopted a recovery identity or identify as being a person in recovery. Moreover, collapsing everyone who remits from AUD into one remitted/recovered category may conflate recovery processes with maturing out. In doing so, this limits our understanding of recovery processes and mechanisms by which individuals achieve sustained remission and enhanced quality of life.

Second, our findings warrant careful consideration of what it means to remit from mild AUD. Although most individuals in the analytic sample individuals (70.3%) experienced a two symptom or greater reduction between the assessment at which they experienced their lifetime maximum number of AUD symptoms and the assessment at which they first remitted, the average lifetime maximum number of AUD symptoms was 3.09 (*SD* = 1.61) for profile 1 and 3.28 (*SD* = 1.59) for profile 2. This means that individuals in both profiles met criteria for mild AUD at the time in their life when they were experiencing the most alcohol-related problems.

Moreover, the mean age at which individuals experienced the most alcohol-related problems was 21.93 (*SD* = 3.16). This is consistent with literature suggesting that problematic alcohol use tends to peak between the ages of 18 and 22 and decrease thereafter (Lee et al., 2018).

However, recent research suggests that the observed decrease in AUD symptoms may represent a "false-positive effect" in which young adults are likely to over-endorse particular criteria (Verges et al., 2012; Verges et al., 2021). According to Verges and colleagues (2021), the over-endorsement is thought to be related to differential interpretations of the criteria as a function of age (e.g., time spent criterion interpreted as time needed to obtain/access alcohol versus time spent drinking/recovering from drinking). Specifically, two criteria – persistent desire or unsuccessful efforts to cut down or control drinking, and drinking despite physical/psychological problems – are potentially problematic indicators of AUD among young adults, as these criteria were less predictive of AUD among young adults relative to older adults and were less likely to be consistently reported as lifetime symptoms of AUD among young adults (i.e., young adults would endorse these criteria as lifetime symptoms at one assessment but not at subsequent assessments). After examining endorsement rates for each AUD criterion across age, Verges and colleagues (2021) concluded that remission from AUD over time reflects "both (1) spurious desistance (particularly from milder AUDs) due to false-positive symptom endorsements and (2) valid desistance (particularly from severe AUDs) due to the substantial role/responsibility-related changes occurring in this period" (p. 13). In other words, it is highly likely that in samples of young adults, patterns of AUD remission reflect both individuals with false-positive AUD cases as well as individuals who mature out of problematic alcohol use.

Considering the research on false-positives in tandem with the pattern of results observed in the present study provides further support that we may have captured a mix of individuals in

our analytic sample, including those who remitted from AUD, those who matured out of AUD, and those who were false-positive cases of AUD to begin with. Among our sample, the symptoms most endorsed at the period in which they experienced the most alcohol-related problems (i.e., at their lifetime maximum AUD assessment) were tolerance (endorsed by 75% of the sample), drinking in larger amounts and spending more time (65%), drinking in dangerous situations (44%), unsuccessful attempts to cut down or cut back (41%), and experiencing related problems (37%). This pattern of endorsement was similar at individuals' first remitted assessment, although drastically reduced in magnitude. Thus, individuals in our sample endorsed a mix of criteria that may be over-endorsed among young adults (drinking despite problems, unsuccessful attempts to cut down/back) and criteria that do not show this age-related inflation. Future research with a larger sample size would be important to tease apart these subgroups of AUD cases and replicate the profile structure observed in the present study.

Recovery Capital and Epidemiological Risk Factors for Substance Use as Predictors of Heterogeneous Patterns of Functioning Related to Quality of Life in AUD Remission

With respect to our recovery capital predictors, we originally hypothesized that sources of positive recovery capital (social support, frequency of attendance at religious services, 12-step affiliation, and history of professional treatment) would be associated with a membership in profiles characterized by higher functioning related to quality of life, while distal sources of negative capital (deviant peer affiliation during adolescence and trauma exposure) would be associated with membership in profiles characterized by lower functioning related to quality of life. This hypothesis was somewhat supported. Among our recovery capital predictors, social support for recovery, exposure to sexual assaultive traumas, and affiliation with more deviant peers during adolescence differentiated the two profiles.

We hypothesized that epidemiological risk factors for substance use (substance use history, externalizing behaviors, and internalizing characteristics) would be associated with membership in profiles characterized by lower functioning related to quality of life. Again, we found partial support for this hypothesis. Among our epidemiological predictors, lifetime maximum depressive symptoms, lifetime maximum PTSD symptoms, and sensation seeking differentiated the two profiles.

Individuals in profile 1 ("infrequent alcohol use, good health") reported higher levels of social support for their recovery (measured via the social recovery capital-IPA, which assesses social network structure and social network abstinence behaviors), were more likely to have a history of sexual assault, and reported more lifetime depressive and PTSD symptoms relative to those in profile 2 ("frequent alcohol use, good to very good health"). Longer time since last AUD diagnosis, defined as the number of years between participants' last assessment at which they met criteria for an AUD diagnosis and their first remitted assessment, and higher lifetime depressive symptoms were associated with an increased probability of being in profile 1 and a decreased probability of being in profile 2. These differences between the profiles also could be related to the fact that profile 1 was composed of more females than profile 2, as females are more likely to report exposure to sexual traumas (Overstreet et al., 2017), be diagnosed with major depressive disorder (Kessler et al., 1993), and develop PTSD (Christiansen & Hansen, 2015) than males. They also reported less affiliation with deviant peers during adolescence and lower levels of sensation seeking than individuals in profile 2, which is consistent with literature suggesting that males exhibit more externalizing behaviors (Hicks et al., 2007). Importantly, the effect size for each of these differences was small.

Social support for recovery, a potent form of social recovery capital, differed significantly between the profiles. Social support for recovery was higher among those in profile 1 than in profile 2, despite the fact that individuals in profile 1 reported higher lifetime maximum symptoms of MDD and PTSD and had higher (although not significantly different) endorsement rates of recent depressive episodes and recent anxiety-related disorder symptoms. Extant literature suggests that social support is an extremely important correlate of recovery, emerging as a commonly cited and critical resource in qualitative studies of individuals in recovery (Hennessy, 2017; Laudet et al., 2006) that can buffer against the pathogenic effects of stress (Cohen & Wills, 1985; Laudet et al., 2006; Smith et al., 2021). Moreover, there is research to suggest that the buffering effects of social support are stronger for females compared to males (Kendler et al., 2005), and females and males exhibit differential patterns of social support networks in recovery (Faleck, 2016). Thus, individuals in profile 1 may use their social support network to cope with stressors, drink less frequently, and avoid relapse. Indeed, results from quantitative studies indicate that greater recovery-oriented social support is associated with less substance use (e.g., Best et al., 2017; Laudet et al., 2006; McCutcheon et al., 2014, 2016; Moos & Moos, 2007), and this was consistent with individuals in profile 1 engaging in less substance use than individuals in profile 2. Moreover, individuals in profile 1 affiliated with less deviant peers during adolescence, and peer groups tend to remain relatively stable over time (Hawkins et al., 1992; Kendler et al., 2018). Therefore, it follows that these individuals had greater social support for their recovery relative to those in profile 2.

Prior research suggests that individuals often exhibit high stress levels and low functioning during the early stages of self-reported recovery (Kelly et al., 2018), and we found a similar pattern of findings among those in profile 1. Individuals in profile 1 were more likely

than those in profile 2 to report a history of sexual assaultive traumas and greater lifetime PTSD symptoms. Interpersonal trauma exposure is associated with negative sense of self (Kouvelis & Kangas, 2021), poor self-esteem, poor emotional regulation (Cicchetti & Toth, 2015; Toth & Cicchetti, 2013), and higher stress reactivity (McEwen, 2004), while PTSD is associated with less favorable responses to substance use treatment (Read et al., 2004), all of which impact one's psychosocial functioning in remission. These individuals also had higher levels of major depressive disorder symptoms than those in profile 2, and higher levels of major depressive disorder symptoms was associated with an increased probability of being in profile 1 and decreased probability of being in profile 2. Thus, individuals in profile 1 may be characterized as a more clinical/internalizing sample than profile 2.

In contrast, individuals in profile 2 affiliated with more deviant peers and reported less social support for their recovery than those in profile 1. They were also higher in sensation seeking and engaged in heavy episodic drinking more frequently than those in profile 1. Although not a statistically significant difference, individuals in profile 2 had more severe lifetime AUD (as defined by a higher maximum number of AUD symptoms) than those in profile 1 despite reporting fewer mental and physical health challenges. Thus, this profile may reflect a group of individuals who tend to be social drinkers who are influenced by deviant peers and a desire to seek novel experiences, rather than a desire to drink to cope with mental health challenges. They may also be less likely to consider their risky drinking as problematic. In conclusion, individuals in profile 2 may be characterized as a higher externalizing subsample. Moreover, profile 2 may reflect individuals who matured out of AUD but continue to occasionally engage in risky drinking rather than those who actively pursued remission.

Contrary to our hypotheses, neither affiliation with 12-step mutual help groups nor history of professional treatment was associated with profile membership. Previous research suggests that approximately half (53.9%) of individuals in self-reported recovery reported lifetime use of an assisted pathway, with mutual help groups and professional treatment being the most commonly endorsed assisted pathways (Kelly et al., 2017). The use of assisted pathways to recovery are important avenues through which individuals can cultivate a recovery identity, as well as accrue human, social, and community recovery capital (Moos, 2008; Rettie et al., 2021). However, there were low endorsement rates of the use of assisted pathways to recovery in our sample (e.g., 4.02% were ever affiliated with AA and 4.33% had a history of professional treatment), especially when considering all participants met lifetime criteria for AUD. This discrepancy may reflect that our sample was young (M_{age} = 24.72 at the time of their first remitted assessment) and had mild problem severity ($M_{\text{lifetime max AUD symptoms}}$ = 3.02), and the use of assisted pathways is higher among individuals in midlife and those with more severe SUD problems (Kelly et al., 2017). Our findings support this, as we found that individuals with severe AUD were more likely to have a history of treatment (12-step affiliation or professional AUD treatment) than those with mild AUD. [2]

Interestingly, although not statistically different, lifetime affiliation with 12-step groups was higher among individuals in profile 2 (5.15%) than those in profile 1 (2.33%). This is not surprising considering the fact that individuals in profile 2 reported higher average lifetime

[2] We conducted an analysis of variance (ANOVA) to test whether a history of any treatment (combined across 12-step affiliation and professional AUD treatment) significantly differed across AUD severity levels (as defined by lifetime maximum AUD symptoms). The model was significant ($F(2, 320) = 4.24$, $p = .015$), indicating a significant difference in treatment endorsement by severity. Post-hoc analyses suggested that individuals with severe AUD were significantly more likely to have a history of treatment than those with mild AUD ($M_{diff} = -0.13$, $p = .011$). There were no differences in treatment history between individuals with mild and moderate AUD or between individuals with moderate and severe AUD.

maximum number of AUD symptoms than those in profile 1, and individuals with more severe AUD are more likely to seek treatment than those with less severe AUD (Kelly et al., 2017). This may also reflect in part the fact that profile 2 was composed of more males and profile 1 was composed of more females, as extant literature suggests that males are more likely to seek treatment than females (Tucker et al., 2020). Lastly, this might reflect the fact that we only captured alcohol-related treatment in the present study. Individuals in profile 1 reported higher lifetime rates of major depressive and PTSD symptoms and higher rates of exposure to sexual assaultive traumas than those in profile 2; therefore, it is plausible that those in profile 1 sought other types of professional treatment.

Considering our findings holistically, when we examined the joint influence of all predictors and covariates on profile membership, the unique contributions of these predictors were attenuated. These findings may suggest that different mechanisms underlying AUD that can also influence functioning among individuals in remission. Notably, peer deviance was still associated with profile membership when considering the influence of social support for recovery, depressive and PTSD symptoms, sexual assault, and sensation seeking, but not when considering the joint influence of all predictors. This suggests that deviant peer affiliation may be more important than mental health backgrounds, but consistent with prior research, deviant peer affiliation may overlap with substance use histories and externalizing behaviors (Hawkins et al., 1992; Kendler et al., 2018). Indeed, affiliation with deviant peers, sensation seeking, substance use, and other externalizing behaviors (e.g., ADHD, ODD, CD, ASPD) are all related to a broader externalizing phenotype (Barr & Dick, 2019; Dick et al., 2010; Krueger et al., 2002).[3]

[3] Although affiliation with deviant peers, sensation seeking, substance use, and other externalizing behaviors are all related to a broader externalizing phenotype, for the purposes of this paper, we conceptualized affiliation with deviant peers as a form of negative recovery capital and substance use behaviors as distinct epidemiological risk

This makes sense because individuals who tend to affiliate with deviant peers during adolescence may be higher in externalizing behaviors and may embark on more severe substance use trajectories starting in adolescence. Moreover, this might suggest that deviant peer affiliation, especially among individuals with higher levels of externalizing behaviors, is a particularly relevant target for prevention. At the same time, individuals with mental health challenges also should be targeted for intervention programs, as previous research has found that the most common cause of relapse was related to negative physical, psychological, and social states (Marlatt, 1996). Thus, intervention programs that help individuals foster healthy coping strategies may reduce the likelihood that these individuals will drink to cope with stressors.

Genetic Risk as Predictors of Heterogeneous Patterns of Functioning Related to Quality of Life in AUD Remission

Lastly, we did not find support for our hypothesis that greater genetic risk for alcohol problems, externalizing behaviors, and internalizing symptoms would be associated with greater likelihood of membership in profiles characterized by lower functioning quality of life. This is unexpected considering a robust body of literature suggesting that SUDs share an underlying heritability towards externalizing behaviors (Barr & Dick, 2019; Dick et al., 2010; Krueger et al., 2002) and may substantially overlap with internalizing disorders, such as mood and anxiety disorders (Edwards et al., 2012; Hesselbrock & Hesselbrock, 2006; Kessler et al., 2003; Tully & Iacono, 2016). In our analytic sample, none of our indices of genetic liability were associated with profile membership.

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factors. We found that sensation seeking did not load onto our externalizing latent factor, so we included it as a separate manifest variable.

The null findings may be attributable to several things. First, as expected based on a priori power analyses, we were likely underpowered to detect an effect, particularly in the African ancestry sample. However, our findings were also null within the European ancestry group. This might be because of the phenotypes selected. These phenotypes (alcohol problems, internalizing symptoms, externalizing symptoms) are robust predictors of substance-related outcomes (Barr & Dick, 2019; Dick et al., 2010; Kranzler et al., 2019; Krueger et al., 2002; Edwards et al., 2012; Hesselbrock & Hesselbrock, 2006; Kessler et al., 2003; Tully & Iacono, 2016); however, they may not be as influential for patterns of functioning related to quality of life. Instead, polygenic scores for phenotypes related to social well-being may be important to consider. Moreover, the identified profiles were split in terms of infrequent alcohol use, good health versus frequent alcohol use, good to very good health. Given that heterogeneity, the PRS may not clearly predict profile membership. If we had profiles that were more clearly distinguishable as high functioning and low functioning, PRS may be better at predicting profile membership. Additional research is needed to more fully understand these null findings.

Global Discussion

Findings from the present study contribute to the growing body of literature regarding quality of life among individuals in remission and can inform our evolving understanding of recovery. First, our findings point to the need for recovery science to employ a multifaceted approach to measuring recovery-related constructs. Namely, recovery science researchers should measure remission from AUD and cessation of heavy drinking, which aligns with NIAAA's new definition of recovery (Hagman et al., 2022). They should also prioritize psychosocial wellbeing and improvements in functioning, a process recognized by numerous agencies and researchers (including NIAAA) as an integral component of the recovery journey. Lastly, researchers may

want to consider and measure individuals' self-reported recovery status (i.e., someone is in recovery if they say they are; Valentine, 2018) and/or their recovery identity, a component of recovery-related research that has been lagging behind the others and yet to be included in many formal definitions.

There has been growing recognition in the field of recovery science about the importance of recovery identities in the recovery journey (Best et al., 2015; Betty Ford Institute Consensus Panel, 2007; Dingle et al., 2015; Mawson et al., 2015). The Betty Ford Consensus Panel (2007) included citizenship, which refers to a sense of recovery identity and membership within a recovery community, in their definition of recovery. Informed by social identity theory (Best et al., 2015; Tajfel & Turner, 1979), forming a recovery identity involves adopting norms and values that align with those of recovery communities/organizations. Recovery identities afford individuals greater social recovery capital through providing a sense of belongingness and purpose (Best et al., 2015), which can help individuals overcome the effects of lower human recovery capital on sustained remission (Cloud & Granfield, 2008). Indeed, qualitative studies reveal a common theme among individuals in self-reported recovery: they discover their "real" selves during their recovery (Dingle et al., 2015). Moreover, individuals who strongly identity as in recovery and with non-using peers, relative to those without a strong recovery identity, report lower levels of substance use, higher levels of recovery capital, and better quality of life (Dingle et al., 2019; Mawson et al., 2015). In sum, this growing body of research underscores the importance of one's personal beliefs about themselves and own recovery journeys.

By concurrently measuring individuals' own beliefs about their recovery status, as well as their clinical remitted status and other psychosocial functioning measures, we can better triangulate who is in recovery (versus those who have matured out). Moreover, using a

multifaceted approach to measuring and defining recovery can allow researchers (and clinicians) to make use of both objective and subjective measures, including examining any discrepancies between the two types of measures. This also can contribute to a better understanding of the mechanisms that facilitate or impede "successful" recovery journeys, as well as the development of a recovery identity. That is, we can determine the extent to which one's agency, commitment, and beliefs about recovery influence their likelihood of sustained remission and enhanced psychosocial functioning. On the other hand, by examining the factors that prevent individuals from adopting or sharing their recovery identities with others, we can learn more about ways to reduce stigma and shame associated with recovery.

Importantly, any consideration of recovery identities should be examined within an intersectional framework (Crenshaw, 1989). Having a substance use and/or recovery identity can be associated with shame and stigma (Romo & Obiol, 2021), which can jeopardize one's quality of life and sustained remission (Livingston et al., 2012), and this can be further exacerbated among individuals with multiple marginalized identities (e.g., Black, Indigenous, people of color [BIPOC], women, LGBTQIA+). For example, it is well-established that BIPOC individuals are more likely to face systemic racism and structural barriers related to healthcare (Cook et al., 2019). As expected, research indicates that racism negatively impacts recovery capital and quality of life among those who resolved an alcohol or drug problem (Vilsaint et al., 2020). Similarly, research suggests that women's and men's experiences in recovery may be qualitatively different (Andersson et al., 2021). Namely, women may face more barriers to treatment (Tweed et al., 2022), have greater unresolved mental health challenges (Andersson et al., 2021), and accumulate more negative recovery capital (e.g., trauma exposure) than men (Neale et al., 2014). Women may also face more stigma related to substance use and recovery

than men (Lloyd, 2010). Generally, these findings aligned with the results from our study. Many of the differences related to psychological challenges (e.g., sexual assaultive trauma exposure, PTSD, depressive symptoms) that were observed between the profiles may be explained by the fact that profile 1 was composed of more females than profile 2. In conclusion, it is imperative that recovery processes are understood not only in the context of individuals' recovery identity, but also their gender identity, racial/ethnic identity, as well as any other marginalized identities that may intersect with and influence their recovery journey.

Limitations and Future Research Directions

The results of the present study should be considered in the context of its limitations. First, this study represents a secondary data analysis of the COGA dataset, which was not originally designed to measure recovery-related constructs (e.g., recovery capital) or quality of life. Because of this limitation, our indicators of quality of life among individuals in remission may not represent ideal measures for our selected constructs. Similarly, our measure of time since last AUD diagnosis was imperfect. We operationalized time since last AUD diagnosis as the number of years between each participant's last assessment at which they met criteria for an AUD diagnosis and the first assessment at which they remitted. However, participants were invited to be re-assessed every two years (on average) and AUD symptoms measured at each assessment used a past 12-month timeframe. Thus, it is possible that our time since last AUD diagnosis variable could be inflated, particularly for participants who missed a re-assessment period before later participating again. Additionally, the measures focused on well-being and current functioning had relatively high levels of missingness (e.g., the Daily Hassles and Uplifts scale; ~37% missing). Although preliminary analyses suggested that these data were missing completely at random and FIML was used to address missingness in our indicators, this may

have influenced our pattern of results. Future work is needed to replicate and expand upon our profile structure using a dataset with more relevant and complete measures, particularly those related to recovery capital, well-being, and positive psychology.

Next, our analytic sample was relatively small and represents individuals who are young, who are early in their AUD remission, and who may not consider themselves in recovery and/or have adopted a recovery identity, all of which limits the generalizability of our findings. We used indicators of quality of life from participants' first assessment at which they remitted from AUD. However, a robust body of literature suggests that many individuals may need multiple attempts to sustain their remission (Kelly et al., 2019), so patterns of functioning related to quality of life may look different at subsequent periods of remission. Namely, at subsequent periods of remission, individuals may have accrued more sources of recovery support on which they can rely and to which they can return. Moreover, research suggests that quality of life and recovery capital tends to increase with length of time in remission (Kelly & Hoeppner, 2015), so profile structures may look different among individuals in long-term recovery. Lastly, a burgeoning body of literature underscores the importance of adopting a recovery identity as a way to increase one's recovery capital, sustain their remission, and improve their quality of life (Dingle et al., 2019; Mawson et al., 2015). Patterns of quality of life among those who adopt a recovery identity may look qualitatively different than those who remit from AUD but do not consider themselves as in recovery. Additional research, including longitudinal studies, is needed to investigate this. Growth mixture models and latent class trajectory analyses may allow researchers to examine changes in these profiles over time. Importantly, all of these processes and phenomena may vary as a function of one's gender identity (Andersson et al., 2021) or

primary substance of choice (Christie et al., 2021), so research that examines the moderating effects of gender and substance of choice is needed.

Finally, the racial/ethnic makeup of the current analytic sample also represents a limitation of the present study. Our analytic sample was predominantly White (69.66%), followed by a smaller proportion of Black/African American (20.12%) and participants of any other racial/ethnic background (10.22%). Because of small sample sizes, participants who identified as any other race/ethnicity or as Asian were collapsed into one group. First, this may limit our ability to generalize our results to individuals from other racial/ethnic groups. BIPOC individuals are more likely to face systemic racism and structural barriers related to healthcare (Cook et al., 2019), which negatively impacts their recovery capital and quality of life (Vilsaint et al., 2020). Second, the racial/ethnic makeup of our sample has implications for our genetic analyses. For genetic analyses, we used genetic principal components to derive each participant's ancestral group. Participants who were of European or African descent, regardless of self-reported race/ethnicity, were included in the genetic analyses, and those from any other ancestral group were excluded from genetic analyses. Despite these being the two largest ancestral groups in our analytic sample, power analyses indicated that we were underpowered to detect an effect in the African ancestry subsample. Together, the exclusion of other ancestral groups and the low statistical power in the African ancestral group limits the generalizability of our results. Although future research is needed to expand this work into more genetically diverse populations, this work represents one small step forward with respect to the inclusion of Black/African American participants in genetic analyses.

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Vita

Rebecca Lynne Smith was born on May 20, 1991, in Toledo, Ohio. She graduated from Woodbridge Senior High School in Woodbridge, Virginia in 2009. She received her Bachelor of Science in Psychology from the University of Mary Washington in 2013 and her Master of Social Work with a concentration in Clinical Social Work from Virginia Commonwealth University in 2015. After graduate school, she worked as a clinical social worker at Tucker Psychiatric Pavilion and as a research analyst at the Virginia Department of Juvenile Justice. In 2017, Rebecca returned to Virginia Commonwealth University to pursue her doctoral degree in Developmental Psychology. She earned her Master of Science in Developmental Psychology in 2019. Rebecca received a Ruth L. Kirschstein National Research Service Award (NRSA) Individual Predoctoral Fellowship (F31) from the National Institute on Alcohol Abuse and Alcoholism in April 2021.