Progression of Substance Use to Substance Use Disorder

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Matthew R. Lee, Yoanna E. McDowell, and Kenneth J. Sher

Introduction

Substance use disorders (SUDs) are among the most prevalent mental illnesses in the USA, with an estimated 21.5 million (8.1%) of Americans over age 12 warranting a past-year SUD diagnosis (Center for Behavioral Health Statistics and Quality [CBHSQ], 2015). This is of great public health concern, as problematic substance use costs the USA an estimated $700 billion per year (National Institute on Drug Abuse, 2015). Further, tobacco, alcohol, and illicit drugs represent the nation's first, third, and ninth leading causes of preventable mortality (respectively; Mokdad, Marks, Stroup, & Gerberding, 2004).

This chapter focuses on understanding SUD and other forms of problematic substance use (as opposed to substance use per se; see Defining Problematic Substance Use). Problematic substance use can occur with a variety of psychoactive substances including illegal substances, legal substances, and even pharmaceutical medications. This chapter is written primarily from a developmental perspective. Thus, when reviewing epidemiology, in addition to characterizing SUD prevalence rates and recent historic changes in these rates, we also emphasize the marked age-prevalence gradient in SUD rates that likely reflects changes in risk over the course of development. Age-prevalence gradients for various substances reveal a robust pattern of increasing SUD prevalence during adolescence and emerging adulthood, followed by reductions beginning in young adulthood and continuing throughout later developmental periods. This developmental pattern also informs our later reviews of research on SUD etiology and SUD desistance. In discussing etiology, we emphasize factors that can contribute to adolescent and emerging adult escalation of problematic substance use. In discussing desistance, we emphasize factors that contribute to age-related reductions in problematic substance use in young adulthood and later developmental periods. Indeed, both an understanding of how SUDs develop and an understanding of how natural desistance occurs can offer key insights toward informing prevention and treatment intervention efforts.

Writing of this chapter was supported by National Institute on Alcohol Abuse and Alcoholism grants K99-AA024236 to Matthew R. Lee and K05-AA017242 to Kenneth J. Sher.

M. R. Lee (✉)
Department of Applied Psychology, Graduate School of Applied and Professional Psychology, Rutgers University, New Brunswick, NJ, USA

Department of Psychological Sciences, University of Missouri, Columbia, MO, USA
e-mail: matthew.r.lee@rutgers.edu

Y. E. McDowell · K. J. Sher
Department of Psychological Sciences, University of Missouri, Columbia, MO, USA
e-mail: yem7c9@mail.missouri.edu; sherk@missouri.edu

Z. Sloboda et al. (eds.), *Prevention of Substance Use*, Advances in Prevention Science,
https://doi.org/10.1007/978-3-030-00627-3_5

Defining Problematic Substance Use

Clinical SUD Diagnosis

The current diagnostic system of the fifth edition of the American Psychiatric Association's (APA) *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*) operationalizes pathological substance use through a diagnosis of Substance Use Disorder (SUD; APA, 2013). The *DSM-5* defines SUD as "a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues using the substance despite significant substance-related problems" (APA, 2013, p. 483). An SUD diagnosis is made by assessing eleven criteria viewed as reflecting four different domains of symptomatology: (1) impaired control (e.g., unsuccessful efforts to control use), (2) social problems (e.g., failures in major obligations), (3) risky use (e.g., in hazardous situations), and (4) physiologic dependence (e.g., withdrawal). An SUD diagnosis is given if two or more of the eleven criteria are met, with severity specified as mild for 2–3 criteria, moderate for 4–5 criteria, and severe for 6 or more criteria.

The *DSM-5* diagnostic system differs substantially from those preceding it, as *DSM* editions dating back to the *DSM-III* (APA, 1980) distinguished between two disorders termed substance abuse and substance dependence. However, the same criteria were largely retained in the transition from *DSM-IV* to *DSM-5*, with the exception that the *DSM-5* dropped the *DSM-IV* "legal problems" criterion and added a "craving" criterion (for more *DSM* history, see Martin, Chung, & Langenbucher, 2016).

Although it is beyond this chapter's scope to further review the advances made in the *DSM-5* and remaining issues that have been raised (see Hasin, 2015; Martin et al., 2016; Wakefield, 2015), it is important to note that such issues are highly pertinent to etiologic and applied research aiming to inform or evaluate prevention and prevention intervention strategies. For instance, from a developmental standpoint, possible biases in some criteria that may inflate false-positive diagnoses at earlier ages should be understood as a possible source of age-related artifactual bias in research on SUD etiology, prevention, and treatment (Boness, Lane, & Sher, 2016).

Other Indices of Problematic Use

In addition to clinical diagnosis, pathological substance use can be indexed by a variety of other measures of substance-related problems and/or excessive consumption (see Del Boca, Darkes, & McRee, 2016). For assessing problematic/risky substance involvement, there exists a wide variety of surveys (e.g., the Rutgers Alcohol Problem Index; Neal, Corbin, & Fromme, 2006) and screening instruments (e.g., the Alcohol Use Disorders Identification Test; Allen, Litten, Fertig, & Babor, 1997), often assessing content that overlaps substantially with diagnostic criteria. Some such measures can be useful for prevention research purposes in providing relatively dimensional indices that capture variability at subdiagnostic levels of problematic use.

For assessing excessive consumption, the clearest definitions exist for alcohol. The National Institute on Alcohol Abuse and Alcoholism (NIAAA, 2004) defines binge drinking as reaching a blood alcohol concentration of 0.08% or above. This corresponds roughly to consuming five or more drinks in two hours for the average man and four or more drinks in two hours for the average women, so research often uses this definition to approximate binge drinking. For other substances, it is more difficult to quantify consumption, let alone establish definitions of excessive use. For nicotine, variations in smoking behavior lead to substantial variability in nicotine intake that is not captured by an assessment of cigarette use quantity (Hammond, Fong, Cummings, & Hyland, 2005). For illicit drugs, there is substantial variability in potency and purity (e.g., Parrott, 2004). Thus, assessments of illicit drug consumption often focus on frequency of use, for instance, with daily use reflecting a relatively severe pattern of consumption.

Epidemiology of SUDs

Prevalence Rates and Historic Trends

The US National Survey on Drug Use and Health (NSDUH) reports yearly SUD prevalence rates since 2002 (CBHSQ, 2015). A rougher picture over a longer historic period can be gleaned by contrasting data from the 1991 National Longitudinal Alcohol Epidemiologic Survey (NLAES) and the 2001 and 2012 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant, Peterson, Dawson, & Chou, 1994; Grant, Moore, Shepard, & Kaplan, 2003; Grant et al., 2014). Other national studies such as Monitoring the Future (MTF) provide rich data on substance *use* but not SUD (Miech, Johnston, O'Malley, Bachman, & Schulenberg, 2015).

Based on 2014 NSDUH data (see Fig. 5.1), among the 8.1% of the US population with some type of SUD, 67% had alcohol use disorder only, 21% had drug use disorder only, and 12% had both. However, NSDUH did not consider nicotine use disorder, which exceeds the prevalence of alcohol use disorder in the USA, according to NESARC data (e.g., 12.8% vs. 8.5% in 2001; Grant, Hasin, Chou, Stinson, & Dawson, 2004). Among SUDs with illicit substances, marijuana use disorder is by far most common. In 2014, US SUD prevalence rates were 1.6% for marijuana, 0.3% for cocaine, and 0.2% for heroin (see Fig. 5.1). A recent concern has been the abuse of pharmaceutical medications, with an SUD prevalence rate of 0.9% in 2014, thus surpassing SUD prevalence rates for both cocaine (0.3%) and heroin (0.2%).

While risky/problematic use is especially common for alcohol and nicotine, there have been relatively dramatic recent historic decreases associated with these substances, as described below.

Alcohol

As depicted in Fig. 5.1, NSDUH showed that alcohol use disorder prevalence rates dropped from 7.7% in 2002 to 6.4% in 2014. Further, more marked reductions over this period were shown for adolescents (ages 12–17; 5.9% to 2.7%) and young adults (ages 18–25; 17.7% to 12.3%). This is mirrored by MTF data showing reductions in heavy drinking over recent decades, but with far more pronounced reductions for adolescents than for college students or other young adults (Miech et al., 2015).

Smoking and Nicotine Use

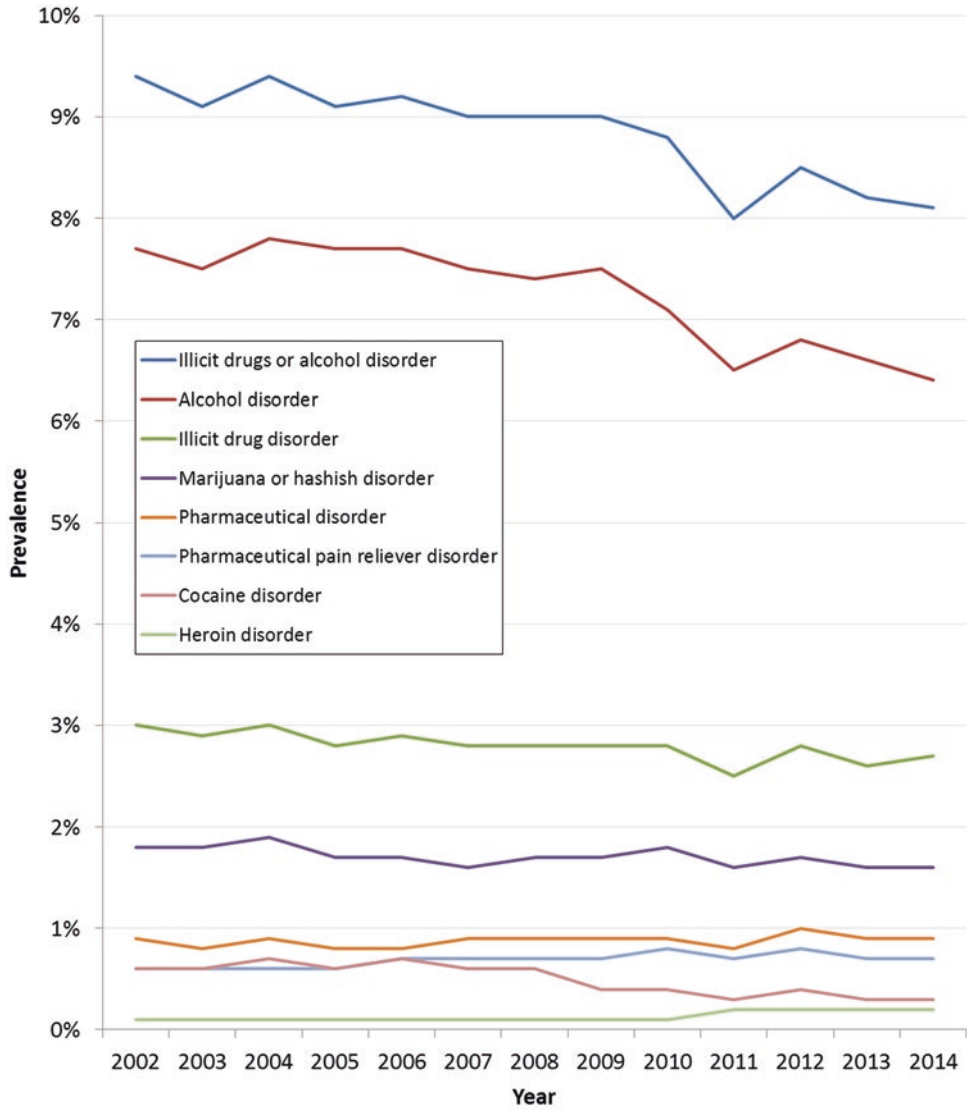
Both MTF and NSDUH show substantial smoking reductions over recent years. MTF data on high schoolers and young adults shows that, since a peak in daily smoking rates of around 20–25% in the late 1990s, rates dropped to a historic low of around 5–10% by 2015 (Miech et al., 2015). NSDUH data also shows that smoking reductions since 2002 were especially pronounced for adolescents and young adults compared to those over age 26 (CBHSQ, 2015). However, a recent concern is nicotine use via e-cigarettes and vaporizers, with MTF data showing that this has grown even more common than traditional smoking among high schoolers (Miech et al., 2015). This appears partially attributable to perceived risk, as less than 20% of 2015 high schoolers perceived great risk in regular vaporizer use, while over 40% perceived great risk in smoking 1–5 cigarettes per day (Miech et al., 2015).

Illicit Drugs

The MTF data tell an interesting story regarding historic trends in the use of marijuana and other illicit drugs among US high schoolers. Miech et al. (2015) describe a 1990s "relapse" characterized by spiking rates of illicit drug use. They argue that public policy reactions since then have succeeded in bringing these rates back down, with the exception that marijuana use has remained relatively elevated. This may reflect increased public permissiveness regarding marijuana, consistent with MTF data showing relatively low perceived harm of marijuana use (Miech et al., 2015).

Pharmaceuticals

Recent concerns about pharmaceutical medications are consistent with NSDUH data showing gradual increases in SUD prevalence rates for



Note. Prevalence rates are based on NSDUH data (CBHSQ, 2015) on rates of DSM-IV abuse or dependence (APA, 1994) among individuals aged 12 or older in the U.S.

Fig. 5.1 US Yearly Trends in Past-Year Substance Disorder Prevalence Rates for Different Specific Substances

these substances since 2002, especially for pharmaceutical pain relievers (see Fig. 5.1). MTF data also raise concerns about rising rates of abuse of Adderall and other pharmaceutical stimulants (Miech et al., 2015).

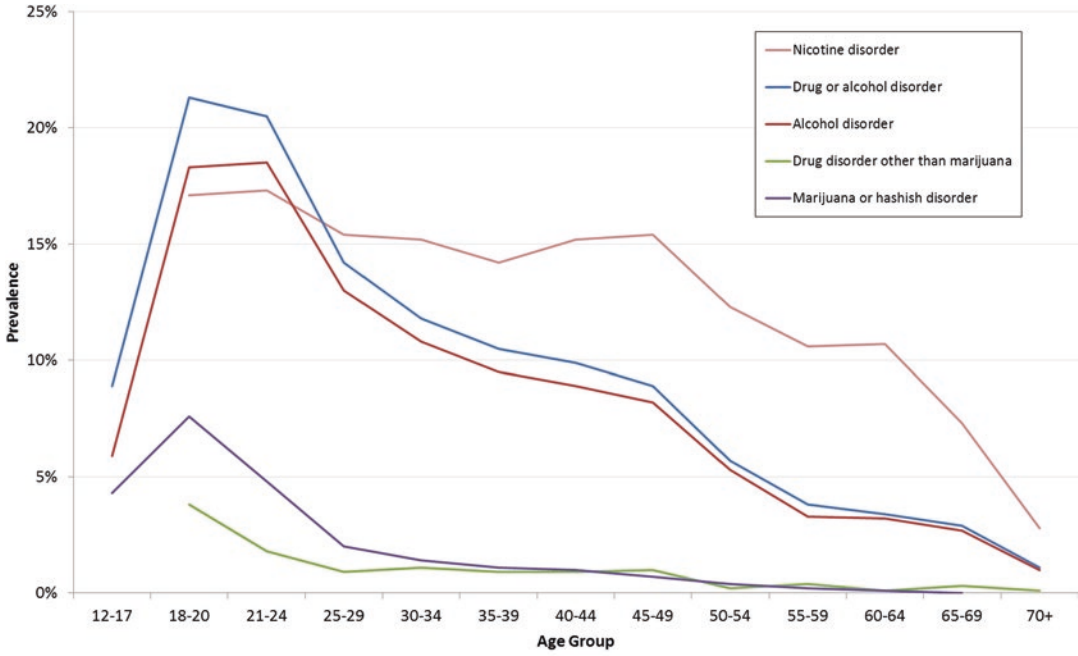
The Developmental Age Gradient of SUD Prevalence

Perhaps the most striking demographic feature of SUD is the age-prevalence gradient characterized

by increasing SUD rates during adolescence, peaks around ages 18–22, and reductions beginning in young adulthood and continuing throughout later developmental periods (see Jackson & Sartor, 2016). However, studies showing age differences in SUD rates for epidemiologic purposes tend to contrast relatively broad age groups, and a finer-grained depiction is informative from a developmental standpoint. Thus, as shown in Fig. 5.2, we conducted our own descriptive analyses of SUD prevalence rates as a function of age using NSDUH and NESARC data.

While Fig. 5.2 generally illustrates that some form of age-prevalence gradient is observed across a variety of substances, it also suggests a unique developmental stability of nicotine use disorder relative to other SUDs. That is, while rates of other SUDs show rapid declines beginning around the 20s, rates of nicotine use disorder remain relatively elevated throughout the 20s, 30s, and 40s, with dramatic declines beginning only around the 50s. This is consistent with MTF data showing relative developmental stability in rates of daily smoking rates throughout the 20s–30s (Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2015). Also noteworthy in Fig. 5.2 are contrasts between illicit drugs and alcohol that are facilitated by our relatively fine-grained age grouping. SUDs rates for marijuana and other drugs show a relatively early downturn in the *early* 20s, whereas rates of alcohol use disorder begin to decline slightly later in the *late* 20s. This is consistent with MTF data showing that daily marijuana use declines rapidly throughout the 20s, whereas heavy drinking declines only gradually in the 20s and more rapidly from the late 20s to mid-30s (Johnston et al., 2015).

Of course, caution is warranted in interpreting cross-sectional age differences as reflecting patterns of developmental change. Indeed, the appearance of a developmental age gradient could be artifactually produced by factors such as differential mortality of those with SUDs and secular changes in prevalence rates. However, it



Note. Prevalence rates for ages 12 to 17 are based on U.S. 2002 NSDUH data (CBHSQ, 2015). Prevalence rates for ages 18 to 70+ are based on U.S. 2001-2002 NESARC data (Grant et al., 2014). Disorder rates reflect DSM-IV abuse or dependence except for nicotine disorder which reflects DSM-IV nicotine dependence (APA, 1994).

Fig. 5.2 The Age-Prevalence Gradient: US Past-Year Substance Disorder Rates Across Age Groups for Different Specific Substances

is unlikely that these other factors could plausibly explain the magnitude of age variability that is observed, given the somewhat limited extent of overall mortality and secular variation. Further, the age-prevalence gradient has also been observed in a number of longitudinal studies that can assess how prevalence rates change as a sample ages (e.g., Chen & Jacobson, 2012).

The robust evidence for an age-prevalence gradient motivates and informs the conceptualization of SUD from a developmental psychopathology standpoint (Chassin, Colder, Hussong, & Sher, 2016; Sher & Gotham, 1999). In particular, it motivates an emphasis on developmental factors that contribute to the escalation of problematic substance use leading up to the early 20s, as well as an emphasis on developmental factors that contribute to the later reductions beginning in young adulthood. These are the two primary topics covered throughout the remainder of this chapter.

Etiology of Problematic Substance Use

In this section, we discuss theoretical, etiologic pathways to SUD. Given earlier-discussed evidence for adolescence as the typical period of escalating substance problems, we largely frame this section as characterizing pathways of emerging risk during adolescence. In organizing the various factors believed to influence SUD development, we adopt a framework emphasizing three inter-related biopsychosocial risk pathways: (1) the "deviance proneness pathway," (2) the "stress and negative affect pathway," and (3) the "pharmacological effects pathway" (Sher & Gotham, 1999). Consistent with a developmental psychopathology perspective, these pathways incorporate genetic and early developmental risk factors. In fact, early risk effects on later substance problems are sometimes viewed as reflecting "heterotypic continuity" (Caspi & Roberts, 1999), with stable underlying risk merely manifesting differently in different developmental periods. The three etiologic pathways are not viewed as competing nor mutually exclusive. Rather, reflecting the principle of equifinality, different pathways may best explain SUD for different individuals, and many individuals may be influenced by multiple pathways. Indeed, our review below emphasizes findings suggesting ways that these pathways may be more interrelated than previously recognized. Our review also emphasizes potential prevention/intervention targets stemming from research on these pathways (for a more comprehensive review, see Chassin et al., 2016).

The Deviance Proneness Pathway

Deviance proneness models view problematic substance use as part of a broader "externalizing spectrum" that includes other problem behaviors (e.g., conduct disorder, antisociality). Developmentally speaking, these externalizing behaviors are viewed as generally originating from genetic risk and early impulsivity, in combination with contextual risk factors like poor parenting and deviant peer involvement (Gottfredson & Hirschi, 1990; Krueger, Markon, Patrick, & Iacono, 2005). Childhood impulsivity and deficient parenting are viewed as "setting the stage" for later school failure, affiliation with deviant peers, and a variety of deviant behaviors that include problematic substance use. As articulated in Jessor's problem behavior theory (Jessor & Jessor, 1977), personality, the environment, and behaviors are viewed as reciprocally influencing one another over time to either increase or decrease risk for future problem behaviors. Through this reciprocal interplay, the presence of one risk factor increases the likelihood that others will emerge, thus causing various problem behaviors to cluster together among at-risk individuals. Deviance proneness models place particular emphasis on heterotypic continuity, as links among problem behaviors across development (e.g., childhood conduct disorder and adolescent substance problems) may reflect stable deviance proneness risk (e.g., impulsivity) that manifests differently as development progresses (Schulenberg, Maggs, & O'Malley, 2003).

There is a great deal of empirical support for deviance proneness models, including prediction

of SUDs by a number of early childhood externalizing behaviors like aggression, defiance, achievement problems, and poor peer relations (King, Iacono, & McGue, 2004). Further, there is particularly marked comorbidity of SUDs with other externalizing disorders (e.g., conduct disorder, antisociality), along with factor analytic evidence that externalizing disorders can be viewed as facets of a broader externalizing spectrum of psychopathology (Cooper, Wood, Orcutt, & Albino, 2003). These factor analytic studies also confirm impulsivity as a key predictor of the externalizing spectrum, complementing other evidence for impulsivity as a predictor of SUDs, more specifically (Sher, Littlefield, & Lee, 2017).

Regarding contextual factors believed to influence deviance proneness, it is important to rule out the possibility that these are mere correlated contextual markers of more causal intraindividual risk processes. For instance, because parenting is genetically influenced, it is noteworthy that research has found poor parenting to predict adolescent substance problems even when controlling for genetic risk (e.g., Dick et al., 2007; Miles, Silberg, Pickens, & Eaves, 2005). Further, because parenting can be influenced by impulsivity of the child, it is noteworthy that research has shown bidirectionality of effects between parenting and impulsivity (Ge et al., 1996) and unique effects of both on later substance problems (Brody & Ge, 2001). Similar findings exist for contextual effects of deviant peer affiliation, with unique effects of peers on substance problems even when controlling for gene- and impulsivity-related peer selection (e.g., Burk, Van Der Vorst, Kerr, & Stattin, 2012; Chassin et al., 2012). Further highlighting the importance of contextual factors like parents and peers, there is evidence that positive contextual influences can buffer effects of intrapersonal risk factors, thereby reducing risk for substance problems among otherwise high-risk individuals (Dick et al., 2007; Miles et al., 2005).

Practical Implications

A key practical implication of research on the deviance proneness pathway is that contextual factors indeed appear capable of buffering risk for adolescent substance problems, as is also indicated by evidence that parenting changes can mediate intervention effects (e.g., Sandler, Schoenfelder, Wolchik, & MacKinnon, 2011). Thus, while deviance proneness models emphasize the developmental continuity of risk that can result from reciprocal effects between individual and context (as described above), it is critical to also note that exposures to positive contexts can create developmentally discontinuous turning points, diverting individuals off of a high-risk trajectory (Rutter, 1996; Schulenberg et al., 2003). Further, the concept of heterotypic continuity highlights that, even in early prevention among youth with no substance use experience, prevention of initiation among high-risk individuals can require disruption of an ongoing risk trajectory characterized not by substance involvement but by other earlier developmental manifestations of deviance proneness (e.g., externalizing behaviors, impulsivity).

Regarding the central role of impulsivity in deviance proneness models, it is noteworthy that there has been increased recent attention to the idea of clinically targeted personality change (including impulsivity reduction; Magidson, Roberts, Collado-Rodriguez, & Lejuez, 2014). Further, it has been argued that universal prevention programs fostering early self-control could confer substantial benefits to most individuals and the population as a whole (Moffitt et al., 2011). Among clinical strategies for adolescent impulsivity reduction, family interventions should emphasize this as a goal of parenting skills training.

The Stress and Negative Affect Pathway

Stress and negative affect models have emphasized the role that substance use can play in alleviating negative emotions, with negative emotionality viewed as sometimes stemming from early stress and traumatic life events (Cappell & Herman, 1972; Greeley & Oei, 1999). However, while a role of affect in SUD etiology is suggested by comorbidity of affective and

substance problems, past research has often found weak or null effects of negative affect on substance problems, especially when tested prospectively or with key covariates (e.g., externalizing; Colder et al., 2013; Hussong, Ennett, Cox, & Haroon, 2017). Further, it has been noted that covariation between affective and substance problems could reflect affective consequences of substance use and the related role of affect in maintaining an existing substance problem (Sher & Grekin, 2007).

However, research has shown clearer effects of daily fluctuations in negative affect on daily fluctuations in problematic substance use. This research supports the notion that, on a day-to-day basis, at least some individuals use substances to cope with negative affect (Epstein et al., 2009; Hussong, Galloway, & Feagans, 2005; Hussong, Gould, & Hersh, 2008). Further, moderated effects show that those most prone to problematic substance use in response to negative affect are those high on impulsivity, externalizing behaviors, and coping-related drinking motives (Hussong et al., 2005; Hussong et al., 2008; Menary et al., 2015). Importantly, by incorporating impulsivity and externalizing behaviors, these findings represent a potential point of synthesis between the deviance proneness and negative affect pathways. This potential synthesis is also reflected in recent evidence for the important etiologic role of "negative urgency" (Settles et al., 2012), a facet of disinhibition characterized by *impulsivity* under conditions of *negative affect* (Cyders & Smith, 2008).

Regarding the stress/trauma component of negative affect models, there is consistent evidence, especially among females, that substance problem development is influenced by early stressful events (e.g., conflict/violence exposure, parental neglect/abuse; Kristman-Valente & Wells, 2013; Sartor et al., 2013; Young-Wolff, Kendler, Ericson, & Prescott, 2011). However, the prediction that this relationship is mediated by negative affect has not been supported. It is therefore noteworthy that early stress/trauma may also impede normal development of behavior and emotion regulation capabilities, and it may be through these mechanisms that early stress/trauma influences substance problem development (Andersen & Teicher, 2009). This represents another potential point of synthesis between the deviance proneness and negative affect pathways, suggesting that risk conferred by early trauma may be partially mediated by impulsivity, including impulsivity in response to negative affect (i.e., negative urgency). This is consistent with evidence that early stress effects on later substance problems are mediated by externalizing but not internalizing symptomatology (Haller & Chassin, 2013; King & Chassin, 2008). Reflecting these empirical advances, more recent articulations of stress and negative affect models have placed greater emphasis on etiologic risk from emotional and behavioral dysregulation, rather than from negative affect per se (e.g., Hussong, Jones, Stein, Baucom, & Boeding, 2011).

Practical Implications

A key practical implication of research on the stress/negative affect pathway stems from the robust evidence for contextual influences of early stress and trauma, which highlights the need for policy, prevention, and treatment intervention strategies to reduce childhood stress/trauma exposure. Further, the potential points of overlap between deviance proneness and stress/negative affect models highlight early stress/trauma exposure as an early risk factor that may have broader effects on a wider variety of later risk processes than has been previously recognized.

Regarding the apparent etiologic importance of negative urgency, in addition to evidence for its broad effects on various forms of psychopathology (Settles et al., 2012), our review highlights its potential role as a common mechanism that could help bridge deviance proneness and stress/negative affect models. Further, in line with our earlier discussion of the movement toward personality-targeting interventions, negative urgency may hold particular promise as a powerful mediator of change in such programs.

The Pharmacological Effects Pathway

Pharmacological effects models focus on individual differences in sensitivity to psychoactive substance effects, with individual differences in sensitivity believed to confer differential risk for SUD development (Newlin & Thomson, 1990; Schuckit, 1987; Wise & Bozarth, 1987). Interestingly, two competing theories make two different partially conflicting sets of predictions regarding how substance-effect sensitivity relates to etiologic risk. The low level of response (LLR) model suggests that high-risk individuals have an overall lower sensitivity to substance effects, with insensitivity viewed as conveying risk in part because greater quantities must be used to achieve desired effects (Schuckit, 1987). In contrast, the differentiator model suggests that the relationship between substance-effect sensitivity and risk varies across types of substance effects (Newlin & Thomson, 1990). While agreeing with the LLR model in predicting that high-risk individuals will be less sensitive to sedating or unpleasant effects, the differentiator model disagrees with the LLR model in predicting that high-risk individuals will be *more* sensitive to stimulating or rewarding effects (de Wit & Phillips, 2012; Quinn & Fromme, 2011). Despite a vast body of past research, inconsistencies between these models remain largely unresolved (de Wit & Phillips, 2012; Morean & Corbin, 2010; Quinn & Fromme, 2011).

However, it can be generally stated that there is evidence across various substances that substance-effect sensitivity relates to risk for future use and related problems. For alcohol, there is particularly clear evidence for risk associated with low sensitivity, especially for more sedating or unpleasant effects (de Wit & Phillips, 2012; Quinn & Fromme, 2011). Indeed, low response to alcohol is viewed as a key alcohol use disorder endophenotype for genetic research, with evidence that it is heritable, predicted by familial alcohol use disorder, and prospectively predictive of alcohol use disorder development (Ray, Mackillop, & Monti, 2010). However, an empirical challenge in human research has been disentangling inborn insensitivity (existing prior to substance initiation) from acquired tolerance to the substance, thus leaving questions regarding directionality of effects between insensitivity and substance problems. Nonetheless, animal research provides evidence for inborn insensitivity effects on substance problem development (de Wit & Phillips, 2012). Regarding nicotine and other drugs (e.g., marijuana, opiates, cocaine), research is generally sparser and extant findings are mixed. However, when effects are detected, they generally show risk associated with *higher* sensitivity to stimulating or rewarding effects and risk associated with *lower* sensitivity to sedating or unpleasant effects (de Wit & Phillips, 2012).

The alcohol literature provides prospective research characterizing a number of mechanisms that may mediate risk originating from substance insensitivity. Based on this research, such mechanisms may include (1) use of greater quantities of the substance to achieve desired effects, (2) selection of heavier substance-using peers, and (3) pro-substance changes in substance-related social norms, substance-effect expectancies, and motives for substance use (e.g., Schuckit et al., 2011; Schuckit, Smith, Trim, Tolentino, & Hall, 2010). The role of deviant peer group affiliation in these processes suggests a potential point of synthesis between deviance proneness and pharmacological effects models. Further, potential overlap between these two pathways is reflected by evidence for associations between impulsivity and substance-effect insensitivity (e.g., Kirkpatrick, Johanson, & de Wit, 2013; Scott & Corbin, 2014).

Arguably, these pathways can "set the stage" for escalation to more severe problematic substance use characterized by what is often termed "addiction." Although there is no precise agreed-upon definition of addiction, most models of addiction are based upon the notion that, with sufficient substance exposure, relatively durable changes in brain circuitry lead to compulsive patterns of use characterized by drug seeking even in the face of punishment. These changes are sometimes described as reflecting a shift from "liking" to "wanting" of a substance (e.g., as in incentive-sensitization theory; Robinson & Berridge, 2008), a shift from instrumental behavior to a

compulsive habit (e.g., Everitt & Robbins, 2005), or an "allostatic" shift in hedonic set-point (sparking a cycle of compensatory substance use and further deviations in the hedonic set-point; Volkow, Koob, & McLellan, 2016). Importantly, these changes suggest that early interventions that precede progression to addiction should perhaps be designed very differently than those targeting individuals exhibiting clear signs of addiction.

Desistance from Problematic Substance Use

As described earlier, epidemiologic data show dramatic age-related reductions in problematic substance use beginning in young adulthood, thus motivating empirical efforts to understand SUD desistance from a developmental perspective. Knowledge of naturally occurring factors that drive desistance can offer unique insights into the nature of SUD and inform public health and clinical interventions (NIAAA, 2008). The following sections review evidence for different possible mechanisms of desistance, beginning with effects of young adult role transitions (e.g., marriage, parenthood) and personality maturation (e.g., decreased impulsivity and neuroticism). Further sections then discuss the need for more lifespan developmental research to explain the later substance-related reductions observed in developmental periods beyond young adulthood, noting some mechanisms that may be particularly relevant to desistance in these periods (e.g., problem recognition, substance-related health concerns).

A key point pertaining to all mechanisms reviewed here is that more research is needed on possible historic changes in how these mechanisms have operated. Preliminary descriptive evidence suggests historic differences across cohorts in the age-related trend of adolescent/emerging-adult escalation and subsequent young adult reduction of substance involvement (e.g., see Fig. 5-18d in Johnston et al., 2015). Key public policy insights could be gleaned from in-depth analyses of such cohort changes in age trends and how they may relate to cohort changes in desistance mechanisms (e.g., the prevalence, life course timing, and impact of adult role transitions). It is also noteworthy that evidence exists for gender, racial, and ethnic differences in both patterns and mechanisms of age-related drinking reductions (e.g., see Chassin et al., 2016). Although discussion of such differences is largely beyond the scope of the current chapter, this should be noted as another important topic in need of further exploration in future research.

Young Adult Maturing Out

Particular attention has been paid in past research to explaining the normative reductions in problematic substance use that occur in young adulthood (Winick, 1962). Speaking to the substantial nature of these reductions, in addition to the fact that declines are observed even in rates of syndromal SUDs (as opposed to less severe indices of problem use; see Fig. 5.2), there is even evidence that the majority of declines in this period occur among individuals with relatively severe pre-young adult patterns of problematic use (Jackson, Sher, Gotham, & Wood, 2001; Lee, Chassin, & Villalta, 2013). These findings indicate a clinical relevance of young adult maturing out, suggesting that efforts to understand this phenomenon could provide key insights guiding the design and improvement of prevention and treatment intervention efforts.

Effects of Young Adult Contextual Transitions

In explaining the reductions in problematic substance use that occur in young adulthood, much attention has been paid to the rapid contextual change that occurs in this developmental period. Of course, when considering possible contextual effects, it is important to bear in mind the distinction between socialization and selection effects (per role incompatibility theory; Yamaguchi & Kandel, 1985). That is, while a changing context *may* influence individuals' behaviors (i.e., socialization), apparent effects of context may instead reflect individuals' entry into contexts that are fit-

ting with their pre-existing individual characteristics (i.e., selection).

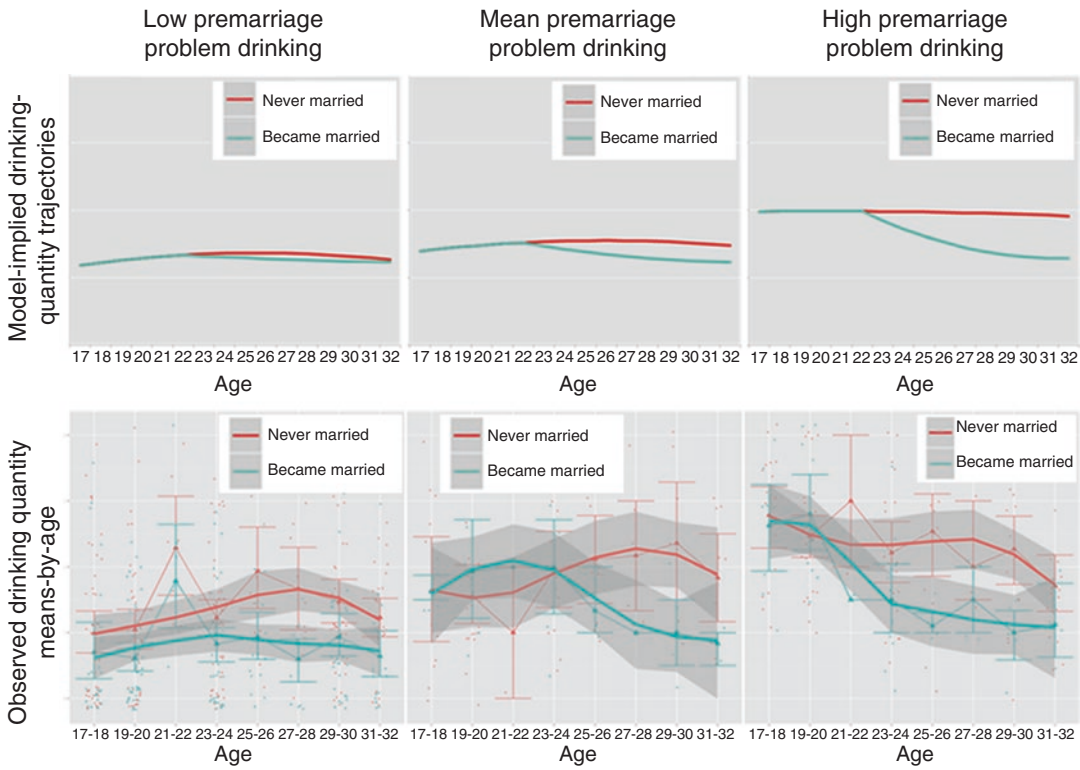
In conceptualizing how contextual change may influence young adult reductions in problematic substance use, it is relevant to consider not only transitions into low-risk environments (e.g., marriage, parenthood) but also contextual transitions out of high-risk environments (e.g., college graduation). For instance, prior to young adulthood, there is evidence for socialization effects of college attendance on increased substance involvement (Bachman, Wadsworth, O'Malley, & Johnston, 1997), as well as other socialization effects of more specific high-risk contexts within the college environment (e.g., fraternity/sorority affiliation; Park, Sher, & Krull, 2008). Thus, as may be expected, there is also evidence that transitions out of high-risk (e.g., college-related) environments may partially explain the subsequent reductions in problematic substance use observed to occur around young adulthood (Bartholow, Sher, & Krull, 2003; Sher, Bartholow, & Nanda, 2001). It is important to bear this in mind in addition to the more common explanation of young adult substance-related reductions as occurring due to normative transitions into lower-risk environments.

Indeed, most past research on young adult "maturing out" has focused on developmental transitions into relatively low-risk adult roles such as marriage, parenthood, and full-time employment. Young adulthood is marked by widespread adoption of such roles (Bachman et al., 1997), and well-established developmental theory views these transitions as key young adult developmental tasks (Erikson, 1968). In studies accounting for role selection as a potential alternative explanation, both young adult marriage and parenthood have generally been shown to convey role socialization effects on reduced substance use and related problems (e.g., Bachman et al., 1997; Curran, Muthen, & Harford, 1998; Flora & Chassin, 2005; Gotham, Sher, & Wood, 2003; Lee, Chassin, & MacKinnon, 2010; Warr, 1998). In contrast, previous research has often failed to show socialization effects of young adult employment on substance-related reductions (e.g., Bachman et al., 1997; Gotham et al., 2003; Warr, 1998), although with some evidence for certain specific occupational categories (e.g., "professional" employment; Staff et al., 2010).

Practical Implications of Effects of Young Adult Contextual Transitions

Supporting the practical (e.g., clinical) relevance of these young adult role effects, in addition to evidence that family roles can spur SUD desistance (e.g., Gotham et al., 2003), there is even evidence that family role effects may be strongest among those with relatively severe pre-role problematic substance use. As depicted in Fig. 5.3, Lee, Chassin, and MacKinnon (2015) found that young adult marriage spurred an especially large drinking trajectory downturn for those with particularly severe problem drinking symptomatology prior to marriage. It is also noteworthy that, beyond family role effects on substance-related maturing out, there is a growing consensus across diverse literatures that family roles (and marriage in particular) can convey various wide-ranging benefits, both catalyzing adaptation and mitigating psychopathology (Derrick & Leonard, 2016; Roberts, Wood, & Smith, 2005; Sampson, Laub, & Wimer, 2006; Walters, 2000).

However, despite the potential importance of family roles from a public health standpoint, surprisingly little is currently known about processes explaining their effects on substance-related maturing out. Existing mediation findings show the most robust support for mediation of family role effects via decreased socializing with peers, with additional mixed evidence for mediation via changes in drinking-related attitudes and increased religiosity (Bachman et al. 2002; Lee et al., 2010; Staff et al., 2010; Warr, 1998). Mediation via reduced socializing with peers is particularly consistent with a role incompatibility explanation, which emphasizes how demands of new family roles can restrict opportunities for substance involvement. However, as articulated in Platt's (1964) commentary on ways to achieve "strong inference," future studies should conduct "riskier" tests of the role incompatibility explanation. This means testing hypotheses that could



Note. These plots contrast the predicted trajectory if marriage never occurred versus if first marriage occurred at age 23, although age 23 is arbitrary, as the model estimates a uniform marriage effect across ages. Plots of observed means-by-age show triangles for means (with connecting lines), color-coded dots for individual data points, bars two standard deviations from means, ans smoothes loess lines with shaded 95% confidence regions.

Fig. 5.3 Problem drinking severity moderates marriage effects on drinking trajectories: Marriage effects on drinking quantity trajectories at three different levels of premarriage problem drinking

provide discriminating support for this over other plausible explanations, and testing hypotheses that could disconfirm this in favor of other plausible explanations. For instance, an explicit assessment of conflict between drinking and family role demands could provide discriminating support for the role incompatibility explanation (Lee, Chassin, & MacKinnon, 2015). Further, this should be tested against other plausible explanations including those emphasizing possible role-driven personality maturation (Lee, Ellingson, & Sher, 2015) and the relational bonds that family roles can forge (e.g., Roberts & Chapman, 2000; Sampson et al., 2006).

Effects of Young Adult Personality Development

Despite a vast, longstanding literature linking personality to substance use and related pathology (Sher et al., 2017), research has only recently considered how personality may relate to maturing out of problematic substance use. This may be due to the traditional view of personality emphasizing stability of personality traits, with research only recently attending to the ways that personality traits change across the lifespan. For instance, Fig. 5.4 depicts meta-analytic evidence for lifespan increases in conscientiousness and emotional stability (akin to lack of neuroticism) (Roberts, Walton, & Viechtbauer, 2006). Perhaps motivated by this work on personality maturation, a subsequent series of studies showed that problem drinking reductions from age 18 to 35 were correlated with decreasing impulsivity, increasing conscientiousness, and decreasing neuroticism across the same age span (Littlefield, Sher, & Wood, 2009, 2010a). A follow-up study using the same data (Littlefield, Sher, & Wood,

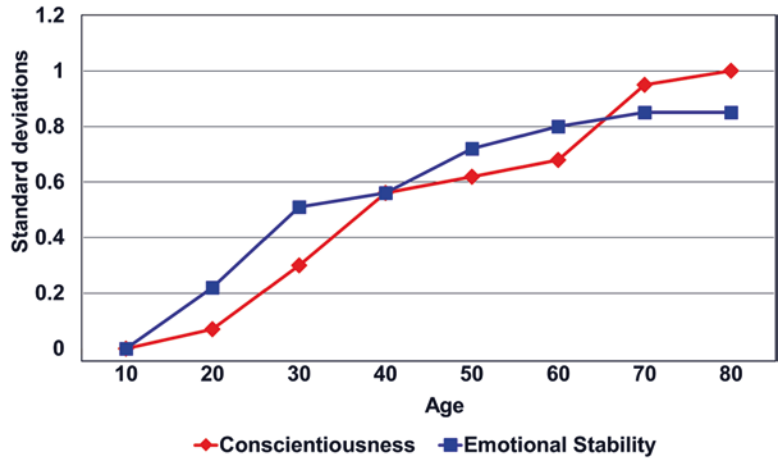


Fig. 5.4 Developmental personality maturation across the lifespan

Note. This figure was adapted from Roberts et al. (2006). It depicts results from their meta-analysis characterizing developmental changes in personality across the lifespan.

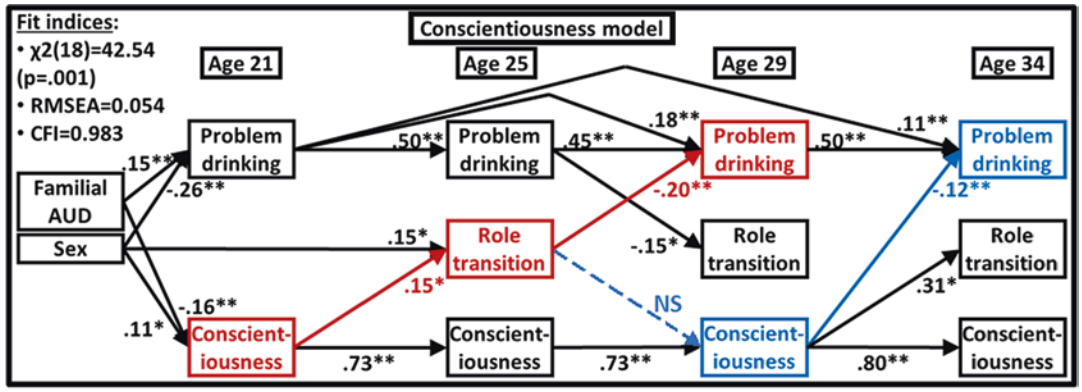
2010b) also showed that the correlated change between personality maturation and problem drinking reductions was mediated by reductions in coping-related drinking motives. Although most research on this topic has focused on problem drinking, similar evidence for correlated change has also been found linking developmental impulsivity reductions with reductions in marijuana and cigarette use (Quinn, & Harden, 2013; Littlefield & Sher, 2012).

The above studies of correlated change between personality and substance problems have forged an entirely new avenue for maturing out research, with an important next step being the investigation of different possible directions of effects. Toward this objective, Lee, Ellingson, and Sher (2015) estimated cross-lag models testing bidirectional effects between personality and problem drinking across four waves spanning ages 21–35. As depicted in Fig. 5.5, results showed prospective effects where both lower impulsivity and higher conscientiousness predicted lower subsequent problem drinking. This evidence for prospective effects complements earlier evidence for correlated change, thereby bolstering confidence in effects of impulsivity and conscientiousness maturation on substance-related maturing out. In contrast, results did not show prospective effects between neuroticism and problem drinking in either direction.

Past studies of correlated change between personality and problem drinking controlled for effects of family roles (Littlefield et al., 2009, 2010a), but beyond this, little else has been done to establish an integrated model of adult role and personality effects on maturing out. Toward this objective, Lee, Ellingson, and Sher's (2015) cross-lag models (described above) included family role transitions (marriage *or* parenthood) at each wave to test mediation between roles and personality in predicting problem drinking. As shown in Fig. 5.5, personality effects were mediated by family role transitions. Specifically, higher conscientiousness and lower impulsivity at age 21 predicted transitions to a family role by age 25, which in turn predicted lower problem drinking at age 29. In contrast, role effects were not mediated by personality, as prospective role effects on personality were not found at any age (see Fig. 5.5).

Practical Implications of Effects of Young Adult Personality Development

In line with our earlier discussion of the movement toward personality-targeting interventions, the above research on personality and maturing out further highlights the likely utility of intervention programs aimed at reducing impulsivity and increasing conscientiousness. Littlefield et al. (2009) speculated that such programs could



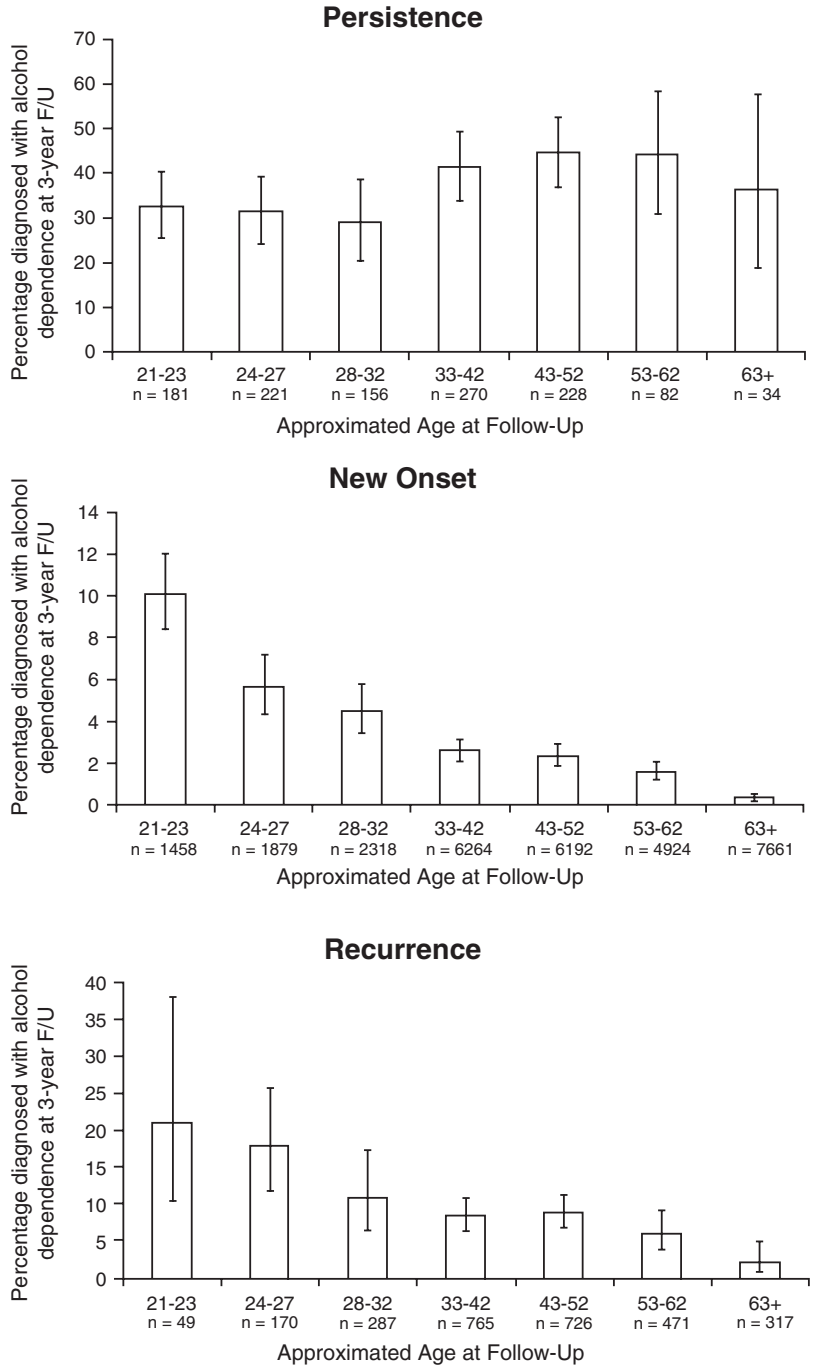
Note. Colors indicate hypothesized mediation paths influencing problem drinking, with results confirming hypothesized mediation of personality effects via role transitions (red) but not confirming hypothesized mediation of role transition effects via personality (blue). For role transition: 0= remained unmarried and a non-parent; 1=became married or a parent. For familial AUD: 0=family-history negative; 1=family-history positive. For sex: 0=male; 1=female. *p < .05. **p < .01.

Fig. 5.5 An integrative model of role and personality effects on maturing out of problem drinking: Results of a cross-lagged panel model of problem drinking, familial role transitions (marriage or parenthood), and conscientiousness across four longitudinal time points

perhaps cause relatively durable changes in drinking behavior by addressing a relatively "deep" underlying component of susceptibility. Consistent with this notion, a recent review concluded that brief cognitive-behavioral treatments for substance use often have enduring effects on personality (Roberts et al., 2017). Further, Lee, Ellingson, and Sher (2015) noted based on their mediational findings that early impulsivity- and conscientiousness-targeting programs could convey protective effects in part by aiding successful subsequent transitions into young adult family roles.

Maturing Out of Substance Problems beyond Young Adulthood

As discussed earlier, epidemiologic data shows that age-related reductions in problematic substance use are not confined to young adulthood, but rather begin in young adulthood and continue throughout the adult lifespan. Beyond this epidemiologic evidence, some additional research exists offering a more precise account of changes in problematic substance use across the adult lifespan. Vergés et al. (2012, 2013) assessed changes across the lifespan in rates of SUD persistence, onset, and recurrence to understand their unique contributions to overall age-related reductions in SUD rates. As depicted in Fig. 5.6, results showed especially marked age reductions in new onsets (Fig. 5.6, middle panel). Thus, although the term "maturing out" may be taken to imply age increases in *desistance*, the continual declines in SUD rates observed throughout the lifespan instead appear largely attributable to age reductions in *new onsets*. In contrast, although not emphasized by Vergés et al., rates of desistance appeared to peak in young adulthood. For instance, based on their alcohol dependence persistence rates (Fig. 5.6, upper panel), it can be inferred that the rate of desistance peaked at 72% by ages 28–32, then declined to a low of 55% by ages 43–52, and then remained somewhat low thereafter. Thus, an interesting possibility is that risk for SUD onset may continually decline throughout the lifespan, whereas potential for desistance from an existing SUD may peak in young adulthood. Perhaps confirming and extending the latter notion, a recent study by this chapter's authors (Lee et al., 2018) investigated desistance across the lifespan while differentiating mild, moderate, and severe alcohol use disorder (per *DSM-5*; APA, 2013). Results showed that, for those with a *severe* alcohol use disorder, desistance rates were substantially higher in young adulthood than in later developmental



Note. This figure, taken from Vergés et al., (2012), contrasts alcohol dependence persistence (i.e., non-desistance; upper panel), new onset (middle panel), and recurrence (lower panel) as three distinct contributors to age differences in overall alcohol dependence prevalence rates. Rates of persistence, new onset, and recurrence over a three-year period are depicted within different age groups of the NESARC sample (Grant et al., 2014). Persistence rate was defined as the percentage of participants with a past-year alcohol dependence diagnosis at the baseline wave who also had a past-year alcohol dependence diagnosis at the three-year follow-up wave. New onset rate was defined as the percentage of participants with no lifetime history of alcohol dependence at baseline who had a diagnosis of past-year alcohol dependence at the three-year follow-up wave. Recurrence rate was defined as the percentage of participants with lifetime but not past-year alcohol dependence at the baseline wave who had a diagnosis of past-year alcohol dependence by the three-year follow-up wave. Brackets show the 95% confidence intervals around means,

Fig. 5.6 Deconstructing the age-prevalence gradient: Rates of longitudinal alcohol dependence persistence, onset, and recurrence within different age groups

periods (e.g., 46–49% at ages 25–34 vs. 25–29% at ages 35–55).

The above evidence for differences across the lifespan in patterns of desistance suggests there may also be important differences across the lifespan in mechanisms of desistance. Assessing this possibility should be a key goal of future research, as key insights have clearly been gleaned by attending to developmental differences in etiologic processes across earlier developmental periods (i.e., across childhood and adolescence; Chassin, Sher, Hussong, & Curran, 2013). Below we consider some specific ways that the mechanisms influencing desistance may vary across periods of the adult lifespan.

Maturing Out vs. Natural Recovery Models

Predictions regarding developmental differences in desistance mechanisms can perhaps be made based on Watson and Sher's (1998) review highlighting dramatic differences in how desistance is viewed between the "maturing out" and "natural recovery" literatures. As discussed earlier, the maturing out literature focuses on young adulthood and has largely viewed desistance as stemming from maturational contextual changes in this developmental period (e.g., marriage; Bachman et al., 1997) and accompanying role demands that conflict with substance involvement (Yamaguchi & Kandel, 1985). Importantly, these processes are rarely conceptualized as involving acknowledgement or concern regarding one's substance use (Jackson & Sartor, 2016; Watson & Sher, 1998). A starkly different view of desistance comes from the "natural recovery" literature, which has investigated precursors of desistance, mostly in midlife samples (e.g., mean age = 41 [SD = 9.1] in a review by Sobell, Ellingstad, & Sobell, 2000). Informed in part by stage models of behavior change, this literature often views desistance as stemming from an accumulation of consequences that can prompt (1) deliberate reappraisals of one's substance use, followed by (2) self-recognition of a substance problem, and then (3) targeted efforts to change substance use behaviors (Klingemann & Sobell, 2007).

Predictions can perhaps stem from an overarching premise that the maturing out and natural recovery literatures may both offer valid conceptualizations of desistance, but with maturing out models applying predominantly to young adulthood and natural recovery models applying predominantly to later developmental periods. That is, young adult desistance may more often stem from the rapid contextual changes occurring in this period, while desistance in later periods may more often stem from more deliberate processes of problem recognition and effortful change. These predictions are consistent with the general notion that contextual effects on behavioral outcomes may decrease with age as individuals increasingly exert control over their environments (Kendler et al., 2007; Scarr & McCartney, 1983). Although quite speculative, these predictions illustrate the potential for lifespan desistance research to reconcile ostensibly discrepant conceptual models, thereby advance the field toward a more unified understanding of desistance and guiding developmentally informed programs.

Older Adult Health and Desistance

Older adulthood brings various health-related physical and cognitive challenges that may increase in importance as possible desistance mechanisms in this late developmental period (White, 2006). For instance, there is evidence that over 50% of US seniors drink alcohol at levels deemed risky in the context of co-occurring medical conditions (Moore et al., 2006). Further, along with these health issues comes increased use of medications that could interact harmfully with alcohol or other substances, with a striking 76% of US seniors using multiple prescription medications (Gu, Dillon, & Burt, 2010). Of the small extant literature on older adult substance use, health issues are among the most commonly reported reasons for desistance (e.g., Schutte, Moos, & Brennan, 2006). However, studies of prospective effects of health problems on substance-related reductions are more equivocal (e.g., Moos, Brennan, Schutte, & Moos, 2010;

Schutte et al., 2006), perhaps owing to the complex relevance of affect- and coping-related issues to older adult substance use (Schulte & Hser, 2014). For instance, there is evidence that health problems can spur substance use reductions, but can also have the opposite effect for those who use substances to cope (Moos et al., 2010).

An important objective should be to expand upon existing research in this area. This should include further study of how affect- and coping-related factors may impede adaptive responding to substance-related health issues, as well as how these processes are influenced by aging-related substance-effect sensitivity (Heuberger, 2009) and changing social support systems (White, 2006). This is particularly important given the increases in older adult substance problems that are projected to coincide with the aging of the "baby boomer" generation (Han, Gfroerer, Colliver, & Penne, 2009), thus suggesting a great future need for empirically informed substance use interventions for older adults.

Concluding Comments

Substance use and SUDs are among the most common risky behaviors and mental health issues in the developed world, creating considerable burden to society and suffering of individuals and their loved ones. Studying the course of substance use and SUDs in the general population reveals a marked age gradient characterized by escalation during adolescence and peaks around ages 18–22. This developmental escalation of risk, and individual differences in this escalation, can be understood to occur through multiple etiologic risk pathways. Broadly speaking, key pathways to SUD include (1) a "deviance proneness pathway" involving an impulsivity-based general tendency toward risky/deviant behaviors; (2) a "stress/negative affect pathway" involving early stress/trauma exposure, negative emotionality, and emotional/behavioral dysregulation; and (3) a "pharmacological effects pathway" involving individual differences in sensitivity to substance effects. Each risk pathway is distally influenced by genetic and early developmental risk factors, and also mediated and moderated by contextual influences. Our review highlights research indicating points of potential overlap among these three pathways that should be further investigated toward advancing a more unified understanding of SUD etiology.

Following the peak developmental period of SUD risk around ages 18–22, the modal course beginning in young adulthood is characterized by desistance and reduced risk for onset or relapse. The shift toward "maturing out" in young adulthood has long been recognized as owing to developmental transitions into adult roles (e.g., marriage, parenthood), although closer examination is needed to better understand the mechanisms of these role effects. Recent research shows that psychosocial maturation is another key contributor to young adult substance-related maturing out, with particularly strong evidence for effects of age-related decreases in impulsivity and increases in conscientiousness. More research is needed to establish an integrated model of adult role and personality effects on young adult maturing out.

Recent findings also highlight that developmental reductions in substance-related risk continue throughout the adult lifespan. Future research should investigate the possibility that certain desistance mechanisms may operate predominantly in young adulthood (e.g., family role effects), while others may become more important in later developmental periods (e.g., "problem recognition" and effortful change, substance-related health concerns). Such work may help reconcile diverse conceptual models of desistance and thereby advance the field toward a more unified understanding of how desistance occurs.

Practical Implications

As discussed throughout this chapter, key insights guiding prevention and treatment intervention efforts can be gleaned from research on problematic substance use epidemiology, etiologic risk pathways, and desistance mechanisms.

Epidemiologic data on age differences identifies periods of normative escalation and normative peaks in risk, thereby guiding decisions about optimal developmental timing for implementing different levels of prevention and treatment intervention programs. An understanding of the etiologic pathways through which problematic substance use develops can assist intervention efforts by (1) informing strategies for early identification of at-risk individuals (e.g., for selective prevention), (2) indicating modifiable risk factors that should be targeted for clinical change, and (3) suggesting ways that other (e.g., non-modifiable) risk factors may moderate program effects. An understanding of naturally occurring processes of desistance from problematic substance use can inform interventions aimed at goading similar changes.

A key conclusion that should be drawn from our review is the substantial impact that contextual factors can have on substance problem trajectories. For instance, this is reflected in the potential for positive parenting and peer influences to buffer risk for substance problem development in deviance proneness models, as well as the potential for adult role transitions to spur maturing out of problematic substance use. This evidence that positive contextual influences can create turning points that disrupt established high-risk trajectories should motivate continual efforts to improve public policy and clinical programs aimed at early intervention with high-risk individuals. In addition, the influence of context highlights the importance of programs preventing exposure to high-risk environments, as is illustrated by the evidence for various mechanisms of risk that can stem from early stress and trauma.

Our review also highlights that certain dispositional characteristics (e.g., impulsivity) track the modal rise and fall of substance-related risk across the entire lifespan. This holds broad practical relevance, as there are likely various applications for interventions targeting such dispositional characteristics, ranging from early prevention to adult SUD intervention. This is particularly noteworthy in light of recent attention to impulsivity reduction as a target for programs ranging from childhood universal prevention to adult clinical treatment.

Regarding mechanisms of desistance, a richer understanding of the specific processes through which normative young adult role transitions (e.g., marriage, parenthood) spur maturing out may reveal ways that these naturally occurring processes can be leveraged in a clinical setting. For young adult problematic substance users, an efficient clinical strategy may be to emphasize anticipated or ongoing adult role transitions in order to initiate or amplify potentially ongoing normative processes of adult role preparation and adaptation. Further, it may be possible for prevention programs to spur earlier initiation of these maturational processes and thereby prevent onset or escalation of substance problems during the critical risk period around ages 18–22. Regarding desistance in later developmental periods, greater empirical attention to possible developmental differences in mechanisms of desistance could help guide lifespan developmental tailoring of prevention and treatment intervention programs.

Limitations

This review is restricted in that the developmental course described here, although characterizing modal trends in the USA and other developed countries, might not be universal. Caution is therefore warranted in generalizing to other cultures (Jackson & Sartor, 2016). Even within the USA, there is evidence for differences among non-Hispanic Caucasians, Hispanics, and African-Americans in age gradients of problematic substance use (see Chassin et al., 2016), perhaps reflecting differences in the timing and nature of adult role transitions and employment opportunities. Also, most empirical knowledge on this subject is based on alcohol research. Although the age-prevalence curve for most other drug use disorders appears largely similar to that of alcohol use disorder (Vergés et al. 2012; Vergés et al. 2013), we noted earlier that this is not true for smoking and tobacco use disorder. This may be one relatively clear example of a broader

issue: that developmental patterns can vary across drugs as a function of factors such as intrinsic addiction potential and/or social acceptability. Thus, greater attention to commonalities and differences across cultures, ethnicities, and substance types is needed to establish an accurate developmental account of problematic substance use.

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