Adolescent resilience to addiction: a social plasticity hypothesis

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Adolescent resilience to addiction: a social plasticity hypothesis

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The prevalence of substance use disorders is highest during adolescence; however, many adolescents experience a natural resolution of their substance use by early adulthood, without any formal intervention. Something appears to be unique and adaptive about the adolescent brain. In this Review, we examine the roles of the social environment and neurocognitive development in adolescents' natural resilience to substance use disorders. At present, little is known about the neurocognitive mechanisms that underlie this adaptive phenomenon, since neurodevelopmental studies have mainly focused on the risk side of the substance use equation: escalation of substance use. To provide a framework for future studies, we put forth a social plasticity model that includes developmentally limited enhanced social attunement (ie, the need to harmonise with the social environment), affective processing, and brain plasticity, which underlie adolescents' capacity to learn from and adapt to their constantly evolving social environments.

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Introduction

An increase in experimentation and risk taking is one of the primary features of adolescence. We define adolescence here as the developmental period between the onset of puberty and the assumption of adult roles and responsibilities, often discussed in the cognitive developmental literature as spanning from 12 years to 25 years of age.[1] Originally understood as a facet of the so-called storm and stress nature of adolescence, risky choices have been presumed to be driven by poor decision making, which some researchers believe demarcates adolescence.[2] However, this aspect of adolescence is increasingly seen as adaptive, with risk taking interpreted to be crucial to adolescents' successful maturation and movement towards independence, propelling them to go out, explore, and connect with peers, for exam ple.[1] Many neurodevelopmental models attribute this developmentally limited surge in adolescent risk taking to rapid changes in how the brain processes affective (eg, reward, arousal, and emotional) and social information, biasing adolescent decision making towards short-term rewarding outcomes.[1,3] Crucially, the opportunity of this developmental period is also its challenge; because of the nature of their developing brain, adolescents are less able to anticipate or perceive long-term negative outcomes, and therefore can make choices that have lasting negative health consequences, including those that stem from heavy substance use, such as injuries incurred during periods of intoxication.

Although the prevalence of heavy substance use and addiction (formally termed substance use disorder in the fifth edition of *Diagnostic and Statistical Manual of Mental Disorders*[4]) is highest during adolescence, most substance use during adolescence naturally resolves by early adulthood, without any treatment or formal intervention.[5] This finding is compelling, since there appears to be something particularly unique and adaptive about the adolescent brain that leads the majority of adolescents towards resilience against protracted substance use disorder trajectories. Unfortunately, little is known about the neurocognitive mechanisms underlying adolescent resilience to substance use disorders, since neurodevelopmental studies have mainly focused on the period of escalating substance use that culminates, almost exclusively, in substance use disorders.[6] However, this approach is remiss in considering the trajectory for the majority of youths, who do not in fact move into sustained substance use disorders.[7] We believe that an understanding of the nature of adolescents' resilience to substance use disorders could be very beneficial; for example, discovery of what drives adolescents' capacity for self-transformation from heavy use to minimal non-problem use can help researchers to understand the inherent capacity of the brain to recover. Furthermore, determination of the mechanisms that underlie this adaptive metamorphosis out of heavy substance use can guide prevention and intervention programming for this critical, and under-served, age group.

What makes the adolescent brain resilient to sustained heavy use and substance use disorders? The social environment plays a major part in guiding adolescent brain development towards adulthood. Adolescence marks a period in which the need to adapt to and harmonise with the social environment is high, a process that we have termed social attunement for the purpose of this Review. The unique capacity of adolescents to learn from and adapt to their constantly evolving social

Key messages

- Many adolescents experience a natural resolution of their substance use by early adulthood
- Little is known about the neurocognitive mechanisms underlying adolescent resilience to substance use disorders
- We propose a social plasticity hypothesis that captures the paradox of adolescent risk and resilience to substance use disorders
- Knowledge of the interplay between social context and brain plasticity could advance prevention and treatment of adolescent substance use disorders

environments is thought to be supported by enhanced socioaffective processing and brain plasticity.[1,8,9] Although these processes have been shown to be a liability, initially increasing the risk of substance use, we believe that this very same set of neurosocial factors might also insulate adolescents against a lifetime of protracted substance use disorder trajectories. In this Review, we examine the role of the social environment and neurocognitive development in adolescent resilience to substance use disorders. For this purpose, we first review typical trajectories of adolescent substance use and the role of the social environment, followed by an overview of adolescent neurocognitive development and implications for substance use disorder trajectories. Furthermore, we offer a new social plasticity hypothesis that captures adolescent resilience to substance use disorders, with a focus on the role of the social environment, social attunement, and brain plasticity. We conclude by detailing clinical implications and suggest avenues to propel growth in the understanding of substance use in this age group, as well as ways to help youths who are struggling to move towards positive change.

Adolescent substance use trajectories and the role of the social environment

In most high-income countries, adolescents are expected to separate and individuate, decreasing their time with parents and concomitantly increasing their time with peers.[10] Although parents' values remain important for many youths, peer input begins to take primacy.[11,12] Over the course of adolescence, youths begin to make decisions about whether and when to engage in substance use, situations that often arise in peer contexts. Despite the wide range of factors involved, the single best predictor in the decision to engage in substance use has been found to be the proportion of substance-using friends.[13] Furthermore, adolescents' perception of peer substance use, in the real world and on social media, has been directly related to both youths' current substance use and their substance use progressions,[14,15] robustly predicting use up to 7 years after initiation.[16]

Although the use of nicotine and alcohol is declining among adolescents, the use of other substances has been consistent over the past six decades in terms of rates and timing of use.[17–19] For most youths, initial experimentation is followed by a natural escalation in use that continues through the secondary school years into emerging adulthood.[17–19] This rise in use is likely to result from features in the adolescent environment that create opportunities for experimentation, including adolescents' exploration of new social groups, experimentation with new behaviours, and reductions in parental monitoring.[20–22] Although parents and caregivers worry about their adolescents' substance use, and particularly the risk of transitioning into addiction, even heavy-using adolescents do not see their substance use as problematic. Rather, most youths report strong positive experiences, especially in the social domain, as a result of their substance use (eg, increases in popularity and having more fun at parties).[23]

Heavy (binge) use is considered to place adolescents and young adults at the greatest risk of transition into substance use disorders and related problems. By the age of 22 years, 32% of emerging adults in the USA and Europe have engaged in binge drinking in the past month (defined as >5 drinks per drinking occasion), 23% in heavy cigarette or electronic cigarette use, and 5% in daily cannabis use.[17,18] Binge drinking is harmful because heavy users are likely to make poor choices. Unintentional substance use-related accidents and injuries affect a large proportion of adolescents and young adults,[24,25] and heavy users incur more substance use-related adverse outcomes, including physical fights, emergency department visits, arrests or detention, and legal consequences, as a result of their substance use.[26] Furthermore, heavy use often interferes with adolescents' life functioning, disrupting academic progress and growth, relationship development and success, and movement into gainful occupations.[27]

Paradoxically, in the 1960s Winick[28] reported a pattern wherein users in adolescence and emerging adulthood showed a natural decline in heavy use that coincided with their social community's movement into increasingly responsible roles, including serious employment, marriage, and childbearing. This natural desistance from substance use was coined "maturing out of addiction".[28]

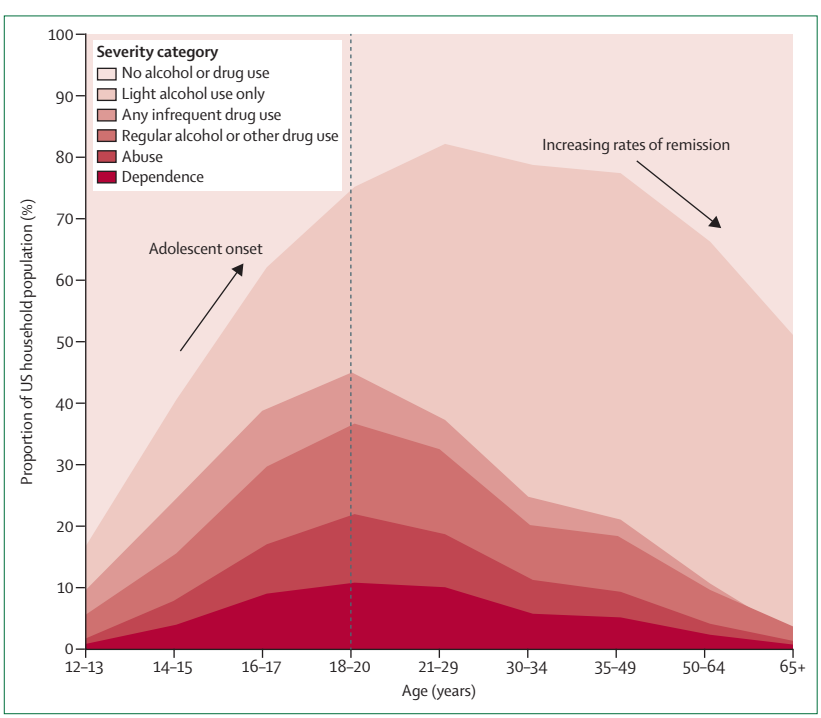

Figure 1: **Substance use and dependence across adolescence and adulthood**
Results from the latest available US household survey[29] in 2001 on drug abuse show that the prevalence of substance use, abuse, and dependence peak during adolescence and early adulthood. Reproduced from Dennis and Scott.[29]

Although the mechanisms of this apparent phenomenon are debated,[7] this pattern of natural reduction in substance use from adolescence to emerging adulthood has been consistently observed (figure 1), most often without users engaging in or receiving any form of treatment or intervention.

Adolescent neurocognitive development

Behavioural change

Cognitive development occurs rapidly during childhood. However, complex cognitive functions, including social cognition and (to some degree) cognitive control, develop throughout adolescence into adulthood. The onset of puberty occurs concurrently with surges in emotional arousal and reward sensitivity,[30] causing adolescents to often make choices that favour short-term outcomes.[31,32] Although short-term outcomes and affective context might initially drive decision making, particularly during early and middle adolescence (12–17 years of age), the capacity to effectively anticipate future events, and adaptively accommodate one's behaviours to protect oneself against potential negative consequences, emerges further in development, closer to the age of 25 years.[30]

In terms of social cognitive development, a wide range of processes undergo major changes during adolescence.[33] Physical and emotional separation from parents together with an increased sensitivity to, complexity of, and reliance on peer relationships are all signature features of adolescence. Unique to this developmental window is the surge in social attunement to peer behaviours; data support the idea that the mere presence of peers substantially shifts the nature of adolescent decision making in a way that has not been observed in children or adults.[34] The interplay between the changing social environment, heightened emotional arousal, and enhanced reward sensitivity means that risk taking and social interactions, which are inherently exciting, frightening, and fun for any age group, can feel much more so during adolescence. Moreover, parallel and steady improvements occur in introspection of one's own complex mental states, and the capacity to understand and care about the mental states of others (ie, mentalising).[35]

Structural and functional brain development

A central set of brain areas underlies changes in adolescent cognition (figure 2). Specifically, the executive network is the main substrate for cold executive control (ie, processing of events of low emotional salience) and includes frontoparietal brain areas, such as the posterior parietal cortex, dorsolateral prefrontal cortex, inferior frontal gyrus, and dorsal anterior cingulate cortex.[36] The salience network is the hub for emotion regulation, salience attribution, and integration of affective information (eg, reward, valence, and emotions) into decision making.[32,36] It includes the ventral anterior cingulate cortex, anterior insula, orbitofrontal cortex, and limbic

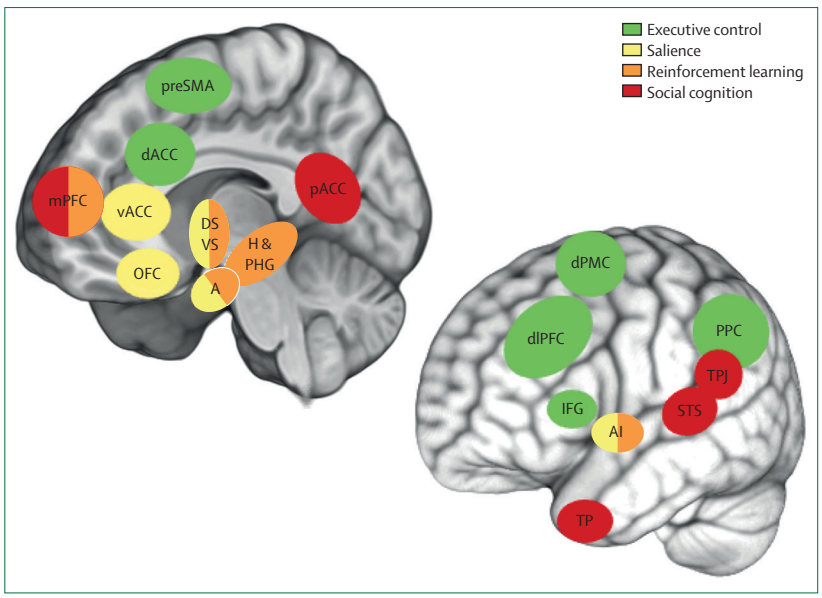

Figure 2: **Brain networks involved in adolescent risk and resilience to substance use and substance use disorders**

The executive control network includes the dorsolateral prefrontal cortex (dlPFC), dorsal anterior cingulate cortex (dACC), posterior parietal cortex (PPC), dorsal premotor cortex (dPMC), pre-supplementary motor area (preSMA), and inferior frontal gyrus (IFG). The salience network includes the amygdala (A), ventral striatum (VS), dorsal striatum (DS), orbitofrontal cortex (OFC), ventral ACC (vACC), and anterior insula (AI). The reinforcement learning network includes the VS, DS, A, and AI, as well as the medial prefrontal cortex (mPFC), and hippocampus and parahippocampal gyrus (H & PHG). The social cognition network includes the mPFC, posterior ACC (pACC), temporoparietal junction (TPJ), superior temporal sulcus (STS), and temporal pole (TP).

areas such as the amygdala and striatum. In turn, social cognitive functioning (eg, cognitive processes involved in interaction with others)[35,37] is driven by a network comprising the medial prefrontal cortex and superior temporal brain areas (eg, the temporoparietal junction). Crucially, these executive and salience networks are also relevant in social cognitive processes that support real-time tracking and affective evaluation of social information.[35]

The behavioural changes observed during adolescence result from widespread changes in structure, function, and connectivity among these brain networks. Although the precise nature of adolescent brain development will continue to be elucidated through large-scale longitudinal studies (eg, IMAGEN[38] and the ABCD Study), trajectories of brain maturation, thus far, appear to have some degree of regional specificity. To that end, frontal brain regions involved in social cognition and control (figure 2) mature later than do regions of the frontolimbic salience network and more posterior temporal and parietal areas. Structural neuroimaging studies demonstrate region-specific decreases in grey matter volume from adolescence into adulthood.[39] Moreover, white matter myelination continues to increase, subsequently enhancing functional connectivity between distant brain regions, which drives faster, more efficient communication within and across brain networks into adulthood.[40]

Functional neuroimaging studies also support increased sensitivity of frontolimbic brain areas to social, emotional,

For more on **ABCD Study** see http://www.abcdstudy.org

and reward information in adolescents compared with children and adults.[35] Executive control functionality is highly sensitive to social and affective context during adolescence, which supports adaptive behavioural flexibility and social attunement to changing social environments.[1,35] Initially, this imbalance underlying adolescent neural sensitivity to peers might drive adolescent risky choices, including substance use.[35,41,42] For example, ventral striatum activation in adolescents, and not in adults, was greater when adolescents were observed by peers during a risk-taking task compared with when they were alone, and this increased activity also predicted higher risk taking.[34] Furthermore, real-life peer conflict has been associated with greater risk taking and stronger activation in the ventral striatum, especially in adolescents with low peer support.[43] However, the developmentally normative imbalance between social or emotional responsiveness and prefrontal cortex-mediated behavioural control and social cognition might also contribute to unique cognitive assets of adolescence, including cognitive flexibility, social awareness and adaptation, acquisition of new experiences, and an enhanced capacity to adaptively learn from these experiences and, in turn, modify behaviour.[1,20]

The role of brain plasticity

Brain plasticity refers to the dynamic biological capacity of the brain to change in response to the environment, supporting brain maturation as well as recovery after injury.[44,45] It involves the modulation of neuronal connections through complex interactions between genes and the environment. Learning, memory, and the underlying neural processes play an important part in this plasticity. An appreciation of how the environment, which is primarily social during the adolescent years, influences and shapes brain development is crucial to understand the effect of peers on adolescent risk behaviours, including substance use. Human research on adolescent brain plasticity is sparse.[44] However, work from the past year has shown that adolescence represents a crucial period of enhanced brain plasticity, specifically in areas associated with experience-dependent associative learning.[9,46] Brain plasticity is already high during childhood, marking key periods in sensory and language development. The onset of puberty is hypothesised to mark a shift in plasticity towards brain areas involved in learning and complex cognitive functions.[9]

Enhanced cortical plasticity during adolescence is supported by observations of extensive experience-dependent pruning and remodelling of cortical connections during this period.[47] Facilitated associative learning and learning from reinforcement (ie, associative learning from positive and negative behavioural outcomes) are associated with increased hippocampal plasticity during adolescence.[8,48,49] For example, studies in rodents show that the adolescent hippocampus produces thousands more neurons a day than does the adult hippocampus, with learning and environmental stimulation as major determinants for the survival of these neurons.[48,49]

Reinforcement learning and the underlying neural mechanisms have been compared in adolescents (13–17 years) versus adults (20–30 years), with a specific evaluation of the function of the hippocampus and striatum.[8] The hippocampus and striatum, together with the parahippocampal gyrus, medial prefrontal cortex, and components of the salience network (eg, the amygdala and anterior insula), are integral to associative learning and memory[50] (figure 2). During reinforcement learning, the extent to which the outcome deviates positively (positive reinforcement) or negatively (negative reinforcement) from what is expected (ie, the prediction error) is used to adapt existing action–outcome associations in an experience-dependent manner. Memory for positive events is better than that for negative events across age groups. However, positive memory bias is associated with improved learning, stronger prediction error-related activity (in the hippocampus), and enhanced functional connectivity between the hippocampus and striatum in adolescents, but not in adults.[8] These initial findings suggest the positive, adaptive nature of reward sensitivity and learning in adolescence, and the inherent capacity adolescents show for neural and behavioural resilience.

Implications of adolescent brain development in the context of substance use

The escalating course of substance use towards substance use disorders is, at least in the adult literature, interpreted as a transition from goal-directed, voluntary use to choiceless, compulsive use, even in the face of negative consequences (eg, drinking heavily after work, even though it will cause family distress). From initial experimentation to heavy substance use, associative action–outcome learning exacerbates use. At the brain level, the salience network becomes increasingly attentive and responsive to substance use and cues that have previously been paired with it, resulting in the motivation to use, along with the automatic and often unconscious tendency to be drawn towards and respond to substance use-related cues (ie, attentional bias and approach bias). Importantly, substance use-related cues are highly personal and can be social (eg, friends and social places or events associated with use) as well as non-social (items associated with substance use) in nature. Poor behavioural control over these motivations, resulting from decreased functioning of the frontoparietal executive network, is believed to support further escalation of substance use into substance use disorders.[6,51]

These neural factors predispose adolescents to initial use and escalation into substance use disorders. First, developmental increases in the connectivity of the salience and reinforcement learning networks, and the

resulting heightened emotional arousal, reward sensitivity, and value of social information, might elevate the valence and rewarding effects of substance use and associated cues. Second, although behavioural control can already be relatively adult-like in cold situations, such control and functioning of the underlying executive network in the context of high affect, intense emotion, and highly salient rewards (including strongly valent social situations) are still developing. Third, the surge in the need to belong, attunement to overt and implicit peer feedback and behaviours, and development of the social cognition network might increase willingness to use substances in order to fit in with, be accepted by, or share a social experience with substance-using peers. Last, enhanced neural plasticity in the form of associative learning might further drive formation and strengthening of associations between substance use and the rewarding social outcomes, which might reciprocally reinforce and increase the likelihood of substance use in peer contexts. The role of reward sensitivity and prefrontal cortical control in adolescent experimentation with and escalation of substance use is well established and elaborately described elsewhere.[6] By contrast, little is known about the neural mechanisms underlying social attunement to peers and the relationship of these mechanisms with brain plasticity in the context of adolescent substance use.

Given that peer substance use is one of the major determinants of escalation of use and subsequent transition to long-term substance use disorders,[14,15] increased social neural attunement to peers during adolescence is highly likely to be a primary risk candidate.[35,52–54] A behavioural study[55] in this domain suggests a social contagiousness around substance use, whereby peer-to-peer use is transmitted through shared social norms (eg, "My friends are all using, so why shouldn't I?") and prototypes (eg, heavy drinkers being perceived as cool). This social contagiousness is further reflected by studies showing peer-to-peer imitation of drinking behaviour in semi-naturalistic bar laboratory settings[56,57] and on social media, in which celebration of pro-drinking behaviours by peers has been connected to shifts in young people's own perceptions of the risks or benefits of substance use.[58] Importantly, the social nature of adolescent substance use is highly multidimensional.[52] For some adolescents, social appraisal of substance use contributes to the development of new relationships and social status, reinforcing the value of substance use as positive and the rewarding aspects of use as social in nature. Some adolescents might use substances in peer settings to prevent negative peer feedback, anticipated peer rejection, or social anxiety, rendering a negative social reinforcement of substance use. Finally, particularly for those adolescents with pre-existing substance use vulnerabilities, intoxication itself is highly rewarding or could alleviate negative affect, which might be enhanced in, but not driven by, the social context.[52] Indeed, a long line of research indicates that youths who drink for enhancement (ie, to improve emotional state) and coping purposes (ie, to reduce negative affect) experience more alcohol-related problems than do those who drink for social motives.[59]

In turn, heightened adolescent brain plasticity, in the form of facilitated associative learning, also has important implications for the transition from experimentation to substance use disorders. Repeated substance use is likely to cause brain adaptations, leading to subsequent development of implicit cognitive biases (eg, attentional bias, automatic approach tendencies, and implicit positive memory association).[51] The formation of implicit cognitive biases towards substance use could be fast-tracked during adolescence. That is, the association between substance use and the social or physical rewarding outcomes might be more easily learned, and thus more easily result in adaptation of existing implicit behaviours in favour of substance use. For example, positive experiences of substance use with friends might automatically trigger motivations to use when the same group of friends is encountered. In this framework, neuroplasticity facilitates adolescent substance use and the transition into heavy substance use and substance use disorders.[60]

Adolescent resilience to addiction: a social plasticity hypothesis

Even in the case of substance use disorders, about half of adolescents who use naturally reduce their substance use by early adulthood[29] (figure 1). We hypothesise that the same neurosocial mechanisms that place adolescents at initial risk of introduction to and escalation of substance use underlie protective factors driving the natural desistance from substance use during emerging adulthood. According to contemporary models of adolescent substance use disorders,[6,51] normative improvement of top-down executive control over the salience network increases the capacity to resist substance use in tempting situations. As long-term goals become increasingly important, impulsive decision making decreases and emotional control improves. However, although progress in behavioural control is one part of the equation, it does not fully account for the process of maturing out. Rather, extending established substance use disorder models, we propose a social plasticity hypothesis of normative adolescent resilience to substance use disorders, underscoring social attunement and heightened brain plasticity as key protective factors in this equation (figure 3).

The notion of maturing out has been investigated extensively in population-based surveys.[61,62] Results from these studies show that role transitions (eg, marriage, employment, and starting a family) and changes in substance use motives are associated with reductions from heavy to moderate levels of, as well as desistance from, substance use. Moreover, the social context and a durable network of social relationships (eg, family, friends, mentors, and co-workers) also support natural

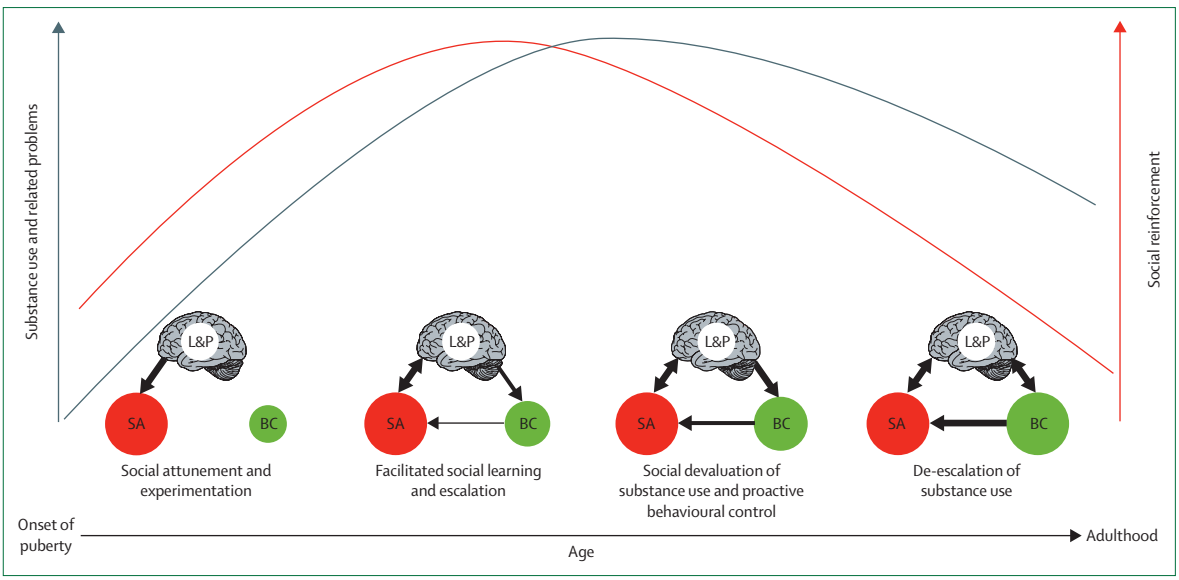

***Figure 3*: Social plasticity model of adolescent resilience to substance use and substance use disorders**
Age-limited increase and subsequent decrease in substance use and related problems are explained by the developmentally changing interactions between the social reinforcing value of substance use, social attunement (SA), learning and plasticity (L&P), and behavioural control (BC). Social attunement and experimentation: onset of puberty marks a shift in brain plasticity and surge in social attunement, and the need to belong and to attune to peer behaviours increases the social reinforcing value of substance use as adolescence progresses. Facilitated social learning and escalation: enhanced associative learning strengthens the association between substance use and the rewarding social outcomes, which can lead to escalation of substance use in social contexts. Social devaluation of substance use and proactive behavioural control: the transition to adulthood parallels social devaluation of substance use, increased importance of long-term goals, improved emotional control, and decreased impulsive decision making. De-escalation of substance use: high social attunement, brain plasticity, and behavioural control integratively allow youths to implicitly notice change in the social reinforcing value of substance use and successfully adapt their behaviour accordingly. The increasing thickness of the arrows refers to the hypothesised increasing strength of the interactions between social attunement, learning and plasticity, and behavioural control across development.

recovery, particularly in emerging adulthood.[63] These studies demonstrate the salient role of the social environment and individual attunement to it in resilience to long-term substance use disorders. Following adoption of serious adult roles and the accompanying change in social environment, the social benefits of substance use decrease (eg, binging and hangover are no longer cool), which devalues the social reinforcing effect of substance use over time. High social attunement allows youths to implicitly notice the change in their social environments around substance use and adapt their behaviour accordingly, often without explicitly being able to detail how and why. Thus, although the need to belong and to attune to peer behaviours initially pulls adolescents towards heavy substance use, it can also draw them away if their environment starts to support and appraise moderate levels of substance use (figure 3).

As with behavioural control and social context, social attunement alone is probably insufficient to drive successful behavioural change. In fact, the unique combination of increased social attunement and brain plasticity might synergistically prime the adolescent brain for this sensitive period of change. Enhanced functioning of associative learning mechanisms mediated by the hippocampus and striatum, paired with social attunement, might dynamically drive flexible and adaptive behavioural change in line with the changing reinforcing value of substance use in young adults. For example, such associative learning processes might amplify learning from situations in which abstaining from substance use is positively reinforced by the social environment; subsequently, uncoupling of social substance use-related cues with positive reinforcing effects, reduction in cognitive biases and motivation to use, and formation of alternative behavioural patterns and habits gradually draw adolescents away from heavy substance use. Although heightened brain plasticity during adolescence is supported by evidence from human[8] and animal[48,49] studies, to the best of our knowledge no studies have directly tested the link between brain plasticity and substance use trajectories. Notably, however, early pubertal onset and maturation might increase the risk of chronic substance use disorders.[64] Therefore, early reduction in brain plasticity, before substance use is socially devalued, could hinder de-escalation of substance use.

Our model implies that adolescent resilience to substance use disorders results from social devaluation of substance use due to high social attunement, integrated with the optimisation of behavioural control, during a period when brain plasticity is still high. Individuals might deviate from this trajectory in multiple ways, and individual differences in the developmental timeframe and strength of social attunement, behavioural control,

brain plasticity, and functionality of the underlying brain networks are likely to drive the specific nature and timing of maturing out. Moreover, the extent to which progress in behavioural control, social attunement, and heightened brain plasticity will result in maturing out in a certain individual is expected to depend on the interaction between the individual's established risk factors for substance use disorder, including sensation seeking, family history of substance use, and genetic vulnerability. We specifically propose that individuals whose substance use is driven by social reinforcement (eg, positive peer feedback and improved social status), as opposed to physical reinforcement (eg, intoxication effects), are more likely to de-escalate substance use during young adulthood. In theory, enhanced reinforcement learning facilitates uncoupling of substance use-related cues from both social and physical positive reinforcing effects if the reinforcing effects are no longer positive. However, the transition to adulthood parallels social, but not (or to a lesser extent) physical, devaluation of substance use. This social devaluation is also probably true for individuals who use to relieve social stress (eg, to cope with negative peer feedback and to avoid feelings of social anxiety) compared with individuals who use to cope with general non-social mental problems. From this perspective, maladaptive substance use that originates from either non-social positive or negative reinforcement is riskier than that resulting from social positive or negative reinforcement.

These hypotheses are in line with the literature on explicit drinking motives.[59,62] However, these motivational processes probably largely operate implicitly, without the youth's awareness. Empirically, these hypotheses are testable through comparison of behavioural and neural reactivity to social and non-social substance use-related cues and assessment of their predictive value for substance use trajectories. Heightened activity in the salience and reinforcement learning networks for social versus non-social substance use might specifically predict natural de-escalation of substance use over time. Moreover, functionality of the social cognition network during a social reinforcement learning task could provide insights into an individual's level of social attunement and capacity to adapt behaviour according to the social environment. Finally, it would be of value to investigate whether social versus non-social coping and enhancement motives (rather than coping and enhancement motives in general) differentially predict substance use disorder trajectories. Although research directly testing these hypotheses has yet to be done, a recent study[65] indeed showed an approach bias to social alcohol cues in heavy-drinking young adults. Taking into account the role of gender in future endeavours is also important in view of previous gender-specific findings when examining the effect of cognitive biases and drinking motives on alcohol intake in a social environment.[65,66]

Clinical implications

Our social plasticity account of adolescent resilience to addiction supports the importance of including the social context when developing substance use prevention and intervention programmes for this age group. Crucially, new interventions are increasingly moving in this direction. For example, Kelly and colleagues[67] have developed an integrated 12-step facilitation treatment involving non-substance-using social groups for teenagers, following the model of Alcoholics Anonymous (AA). Initial findings suggest potential iatrogenic outcomes stemming from group-based interventions focused on shifting peer norms and changing peer context.[68] Furthermore, adolescents can be particularly difficult to engage in group interventions, probably due to the importance of peer influence and increases in reward-seeking behaviour during this time.[69] Some teams have been able to mitigate these negative outcomes through careful and attentive monitoring by skilled clinicians to prevent the occurrence of negative cross-talk (eg, when youths try to outperform other group members in risk behaviour).[70] Moreover, other approaches that clinicians can use include keeping groups small and homogeneous in terms of age, gender, and risk level,[70] and inclusion of positive prosocial adolescents in group treatment contexts.[71,72]

In addition to the promise of prosocial community and group-based interventions, the emerging field of e-health has started to incorporate social media and smartphone apps. E-health interventions include content from and communication via these technologies to engage youths and mobilise their social communities towards change. Although these approaches appear to have high acceptability and feasibility in adolescents, their treatment effects are still inconclusive, and more clinical data are required to determine whether these methods translate to behavioural change in relevant risk contexts.[73] One potentially relevant complicating factor that needs to be considered and mitigated in friend-based interventions in this age group is the highly intense but dynamic nature of friendships, wherein even deep profound friendships might not always endure the test of time.[74]

In addition to directly shifting the social sphere, one potential approach could be to focus on enhancing learning and plasticity in the adolescent or emerging adult themselves. Although neuromodulation methodologies (eg, transcranial direct current stimulation and transcranial magnetic stimulation) have been primarily applied to the dorsolateral prefrontal cortex to boost cognitive control, examination of other neural loci that might be relevant to substance use behaviours has been recommended.[75] To this end, we believe that one previously unexamined and highly salient missing link is the role of enhanced learning via enriched encoding of salient reward–hippocampal communication.[76] With youths who are currently heavy drinking or have alcohol-related problems, one potential avenue might be to use innovative

Search strategy and selection criteria

References for this Review were identified through searches in PubMed with the following search terms: "adolescence" and ("resilience" or "recovery" or "maturing out" or "brain development" or "brain plasticity" or "social development") and/or ("addiction" or "substance use disorder" or "alcohol" or "cannabis" or "smoking"). Original research articles and reviews were considered if they were published before July 15, 2017, written in English, and focused on the development of adolescents and young adults (aged 12–28 years) regarding heavy and problem alcohol, cigarette, and substance use (including addiction), social neurocognitive development, and brain plasticity. Articles were also identified through cross-referencing and searches of the authors' own files. The final reference list was generated on the basis of recency and relevance to the scope of this Review.

low-risk routes of neurostimulation, such as transcranial magnetic stimulation or transcranial direct current stimulation, to stimulate brain areas involved in learning.[77] Brain areas that we believe might be most relevant to adolescent social context and substance use are regions integral to communication between social cognition and reinforcement learning (eg, the medial prefrontal cortex, hippocampus, and parahippocampal gyrus; figure 2). Neuromodulation might thereby offer an integrative method to incorporate knowledge about the developing brain into the applied treatment context in a manner that is highly relevant to adolescent addiction treatment providers.[78]

Conclusions

Most studies investigating adolescent substance use and substance use disorders focus on escalation of substance use and related problems. The paradox of adolescent risk and resilience to substance use disorders underscores the need for a refined model of adolescent substance use and substance use disorders, and more studies that aim to understand adolescent resilience in this equation. In this Review, we propose a social plasticity hypothesis. The model we put forward is simplified and needs rigorous testing and validation, but it provides a framework for future studies to start to resolve the paradox of both adolescent risk and resilience to heavy substance use and substance use disorders. We recommend future longitudinal investigations to address the study of chronicity versus natural recovery in adolescence. These studies should take into account the role of gender, individual differences, and existing risk factors for substance use disorders. Operationalisation of social attunement and brain plasticity will be challenging; however, we believe that knowledge of the interplay between these processes has great potential to improve our understanding of adolescent substance use disorders and identify targets for prevention and treatment.

Contributors

All authors contributed intellectually to the development of the proposed model and to writing of the manuscript. JC created the figures.

Declaration of interests

We declare no competing interests.

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References

1 Crone EA, Dahl RE. Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nat Rev Neurosci* 2012; **13:** 636–50.

2 Hall GS. Adolescence: its psychology and its relations to physiology, anthropology, sociology, sex, crime, religion and education. New York: D Appleton and Company, 1904.

3 Casey BJ, Getz S, Galvan A. The adolescent brain. *Dev Rev* 2008; **28:** 62–77.

4 American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 5th edn. Arlington: American Psychiatric Publishing, 2013.

5 Chassin L, Fora DB, King KM. Trajectories of alcohol and drug use and dependence from adolescence to adulthood: the effects of familial alcoholism and personality. *J Abnorm Psychol* 2004; **113:** 483–98.

6 Conrod P, Nikolaou K. Annual research review: on the developmental neuropsychology of substance use disorders. *J Child Psychol Psychiatry* 2016; **57:** 371–94.

7 Verges A, Haeny AM, Jackson KM, et al. Refining the notion of maturing out: results from the national epidemiologic survey on alcohol and related condition. *Am J Public Health* 2013; **103:** e67–73.

8 Davidow JY, Foerde K, Galván A, Shohamy D. An upside to reward sensitivity: the hippocampus supports enhanced reinforcement learning in adolescence. *Neuron* 2016; **92:** 93–99.

9 Piekarski DJ, Johnson CM, Boivin JR, et al. Does puberty mark a transition in sensitive periods for plasticity in the associative neocortex? *Brain Res* 2017; **1654:** 123–44.

10 Windle M, Spear LP, Fuligni AJ, et al. Transitions into underage and problem drinking: developmental process and mechanisms between 10 and 15 years of age. *Pediatrics* 2008; **121** (suppl 4): S273–89.

11 Ernst M, Pine DS, Hardin M. Triadic model of the neurobiology of motivated behavior in adolescence. *Psychol Med* 2005; **35:** 1–14.

12 Sebastian C, Burnett S, Blakemore S-J. Development of the self-concept during adolescence. *Trends Cogn Sci* 2008; **12:** 441–46.

13 Chassin L, Hussong A, Barrera M, Molina B, Trim R, Ritter J. Adolescent substance use. In: Lerner R, Steinberg L, eds. Handbook of adolescent psychology, 2nd edn. New York: Wiley, 2004.

14 D'Amico EJ, McCarthy DM. Escalation and initiation of younger adolescents' substance use: the impact of perceived peer use. *J Adolesc Health* 2006; **39:** 481–87.

15 Nesi J, Rothenberg WA, Hussong AM, Jackson KM. Friends' alcohol-related social networking site activity predicts escalations in adolescent drinking: mediation by peer norms. *J Adolesc Health* 2017; **60:** 641–47.

16 Feldstein Ewing SW, Filbey FM, Loughran TA, Chassin L, Piquero AR. Which matters most? Demographic, neuropsychological, personality, and situational factors in long-term marijuana and alcohol trajectories for justice-involved male youth. *Psychol Addict Behav* 2015; **29:** 603–12.

17 Johnson LD, O'Malley PM, Bachman JG, Schulenberg JE, Miech RA. Monitoring the future national survey results on drug use, 1975–2014: volume 2, college students and adults ages 19–55. Ann Arbor: University of Michigan, 2016.

18 ESPAD Group. ESPAD Report 2015: results from the European school survey project on alcohol and other drugs. Lisbon: European Monitoring Centre for Drugs and Drug Addiction (EMCDDA), 2016.

19 WHO. Psychoactive substance use among adolescents. Geneva: World Health Organization, 2017.
20 Giedd J. The amazing teen brain. *Sci Am* 2015; **312:** 32–37.
21 Azofeifa A, Mattson ME, Schauer G, McAfee T, Grant A, Lyerla R. National estimates of marijuana use and related indicators—national survey on drug use and health, United States, 2002–2014. *MMWR Surveill Summ* 2016; **65:** 1–28.
22 Simons-Morton B, Haynie D, Liu D, Chaurasia A, Li K, Hingson RW. The effect of residence, school status, work status, and social influence on the prevalence of alcohol use among emerging adults. *J Stud Alcohol Drugs* 2016; **77:** 121–32.
23 Feldstein Ewing SW, Apodaca TR, Gaume J. Ambivalence: prerequisite for success in motivational interviewing with adolescents? *Addiction* 2016; **111:** 1900–07.
24 Scott-Parker B, Oviedo-Trespalacios O. Young driver risky behaviour and predictors of crash risk in Australia, New Zealand and Colombia: same but different? *Accid Anal Prev* 2017; **99:** 30–38.
25 Heron M. Deaths: leading causes for 2010. *Natl Vital Stat Rep* 2013; **62:** 1–96.
26 Hingson RW, Zha W, White AM. Drinking beyond the binge threshold: predictors, consequences, and changes in the US. *Am J Prev Med* 2017; **52:** 717–27.
27 Merrill JE, Carey KB. Drinking over the lifespan: focus on college ages. *Alcohol Res* 2016; **38:** 103–14.
28 Winick C. Maturing out of narcotic addiction. *Bull Narc* 1962; **14:** 1–7.
29 Dennis M, Scott CK. Managing addiction as a chronic condition. *Addict Sci Clin Pract* 2007; **4:** 45–55.
30 Steinberg L. Cognitive and affective development in adolescence. *Trends Cogn Sci* 2005; **9:** 69–74.
31 Todd RM, Cunningham WA, Anderson AK, Thompson E. Affect-biased attention as emotion regulation. *Trends Cogn Sci* 2012; **16:** 365–72.
32 Ochsner KN, Gross JJ. The cognitive control of emotion. *Trends Cogn Sci* 2005; **9:** 242–49.
33 Burnett S, Sebastian C, Cohen Kadosh K, Blakemore S-J. The social brain in adolescence: evidence from functional magnetic resonance imaging and behavioural studies. *Neurosci Biobehav Rev* 2011; **35:** 1654–64.
34 Chein J, Albert D, O'Brien L, Uckert K, Steinberg L. Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Dev Sci* 2011; **14:** F1–10.
35 Kilford EJ, Garrett E, Blakemore S-J. The development of social cognition in adolescence: an integrated perspective. *Neurosci Biobehav Rev* 2016; **70:** 106–20.
36 Seeley WW, Menon V, Schatzberg AF, et al. Dissociable intrinsic connectivity networks for salience processing and executive control. *J Neurosci* 2007; **27:** 2349–56.
37 Murray RJ, Debbane M, Fox PT, Bzdok D, Eickhoff SB. Functional connectivity mapping of regions associated with self- and other-processing. *Hum Brain Mapp* 2015; **36:** 1304–24.
38 Schumann G, Loth E, Banaschewski T, et al. The IMAGEN study: reinforcement-related behaviour in normal brain function and psychopathology. *Mol Psychiatry* 2010; **15:** 1128–39.
39 Mills KL, Goddings AL, Herting MM, et al. Structural brain development between childhood and adulthood: convergence across four longitudinal samples. *Neuroimage* 2016; **141:** 273–81.
40 Uhlhaas PJ, Singer W. The development of neural synchrony and large-scale cortical networks during adolescence: relevance for the pathophysiology of schizophrenia and neurodevelopmental hypothesis. *Schizophr Bull* 2011; **37:** 514–23.
41 Casey BJ, Jones RM. Neurobiology of the adolescent brain and behavior: implications for substance use disorders. *J Am Acad Child Adolesc Psychiatry* 2010; **49:** 1189–201.
42 Gálvan A, Hare T, Voss H, Glover G, Casey BJ. Risk-taking and the adolescent brain: who is at risk? *Dev Sci* 2007; **10:** F8–14.
43 Telzer EH, Fuligni AJ, Lieberman MD, Miernicki ME, Galván A. The quality of adolescents' peer relationships modulates neural sensitivity to risk taking. *Soc Cogn Affect Neurosci* 2015; **10:** 389–98.
44 Fuhrmann D, Knoll LJ, Blakemore S-J. Adolescence as a sensitive period of brain development. *Trends Cogn Sci* 2015; **19:** 558–66.
45 Pascual-Leone A, Amedi A, Fregni F, Merabet LB. The plastic human brain cortex. *Annu Rev Neurosci* 2005; **28:** 377–401.
46 Juraska JM, Willing J. Pubertal onset as a critical transition for neural development and cognition. *Brain Res* 2017; **1654:** 87–94.
47 Huttenlocher PR, Dabholkar AS. Regional differences in synaptogenesis in human cerebral cortex. *J Comp Neurol* 1997; **387:** 167–78.
48 Aoki C, Chowdhury TG, Wable GS, Chen Y-W. Synaptic changes in the hippocampus of adolescent female rodents associated with resilience to anxiety and suppression of food restriction-evoked hyperactivity in an animal model for anorexia nervosa. *Brain Res* 2017; **1654:** 102–15.
49 DiFeo G, Shors TJ. Mental and physical skill training increases neurogenesis via cell survival in the adolescent hippocampus. *Brain Res* 2017; **1654:** 95–101.
50 Garrison J, Erdeniz B, Done J. Prediction error in reinforcement learning: a meta-analysis of neuroimaging studies. *Neurosci Biobehav Rev* 2013; **37:** 1297–310.
51 Wiers RW, Bartholow BD, van den Wildenberg E, et al. Automatic and controlled processes and the development of addictive behaviors in adolescents: a review and a model. *Pharmacol Biochem Behav* 2007; **86:** 263–83.
52 Caouette J, Feldstein Ewing SW. Four mechanistic models of peer influence on adolescent cannabis use. *Curr Addict Rep* 2017; **4:** 90–99.
53 Steinberg L. A social neuroscience perspective on adolescent risk-taking. *Dev Rev* 2008; **28:** 78–106.
54 Somerville LH, Jones RM, Casey BJ. A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain Cogn* 2010; **72:** 124–33.
55 Teunissen HA, Kuntsche E, Scholte RH, Spijkerman R, Prinstein MJ, Engels RC. Friends' drinking norms and male adolescents' alcohol consumption: the moderating role of performance-based peer influence susceptibility. *J Adolesc* 2016; **53:** 45–54.
56 Dallas R, Field M, Jones A, Christiansen P, Rose A, Robinson E. Influenced but unaware: social influence on alcohol drinking among social acquaintances. *Alcohol Clin Exp Res* 2014; **38:** 1448–53.
57 Larsen H, Overbeek G, Granic I, Engels RC. The strong effect of other people's drinking: two experimental observational studies in a real bar. *Am J Addict* 2012; **21:** 168–75.
58 Steers M-LN, Moreno MA, Neighbors C. The influence of social media on addictive behaviors in college students. *Curr Addict Rep* 2016; **3:** 343–48.
59 Kuntsche E, Knibbe R, Gmel G, Engels R. Why do young people drink? A review of drinking motives. *Clin Psychol Rev* 2005; **25:** 841–61.
60 Lewis M. Addiction and the brain: development, not disease. *Neuroethics* 2017; **10:** 7–18.
61 Lee MR, Ellingson JM, Sher KJ. Integrating social-contextual and intrapersonal mechanisms of "maturing out": joint influences of familial-role transitions and personality maturation on problem-drinking reductions. *Alcohol Clin Exp Res* 2015; **39:** 1775–87.
62 Littlefield AK, Sher KJ, Wood PK. Do changes in drinking motives mediate the relation between personality change and "maturing out" of problem drinking? *J Abnorm Psychol* 2010; **119:** 93–105.
63 Granfield R, Cloud W. Social context and "natural recovery": the role of social capital in the resolution of drug-associated problems. *Subst Use Misuse* 2001; **36:** 1543–70.
64 Mendle J, Turkheimer E, Emery RE. Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Dev Rev* 2007; **27:** 151–71.
65 Groefsema M, Engels R, Kuntsche E, Smit K, Luijten M. Cognitive biases for social alcohol-related pictures and alcohol use in specific social settings: an event-level study. *Alcohol Clin Exp Res* 2016; **40:** 2001–10.
66 Smit K, Groefsema M, Luijten M, Engels R, Kuntsche E. Drinking motives moderate the effect of the social environment on alcohol use: an event-level study among young adults. *J Stud Alcohol Drugs* 2015; **76:** 971–80.
67 Kelly JF, Yeterian JD, Cristello JV, Kaminer Y, Kahler CW, Timko C. Developing and testing twelve-step facilitation for adolescents with substance use disorder: manual development and preliminary outcomes. *Subst Abuse* 2016; **10:** 55–64.

68 Dishion TJ, McCord J, Poulin F. When interventions harm. Peer groups and problem behavior. *Am Psychol* 1999; **54:** 755–64.

69 Onrust SA, Otten R, Lammers J, Smit F. School-based programmes to reduce and prevent substance use in different age groups: what works for whom? Systematic review and meta-regression analysis. *Clin Psychol Rev* 2016; **44:** 45–59.

70 Feldstein Ewing SW, Walters S, Baer JS. Approaching group MI with adolescents and young adults: strengthening the developmental fit. In: Wagner CC, Ingersol KS, eds. Motivational interviewing in groups. New York: Guilford Press; 2013: 387–406.

71 Mason M, Light J, Campbell L, et al. Peer network counseling with urban adolescents: a randomized controlled trial with moderate substance users. *J Subst Abuse Treat* 2015; **58:** 16–24.

72 Nash A, Collier C. The alternative peer group: a developmentally appropriate recovery support model for adolescents. *J Addict Nurs* 2016; **27:** 109–19.

73 Grist R, Porter J, Stallard P. Mental health mobile apps for preadolescents and adolescents: a systematic review. *J Med Internet Res* 2017; **19:** e176.

74 Feldstein Ewing SW, Bryan AD. A question of love and trust? The role of relationship factors in adolescent sexual decision making. *J Dev Behav Pediatr* 2015; **36:** 628–34.

75 Spagnolo PA, Goldman D. Neuromodulation interventions for addictive disorders: challenges, promise, and roadmap for future research. *Brain* 2017; **140:** 1183–203.

76 Murty VP, Adcock RA. Enriched encoding: reward motivation organizes cortical networks for hippocampal detection of unexpected events. *Cereb Cortex* 2014; **24:** 2160–68.

77 Demeter E, Mirdamadi JL, Meehan SK, Taylor SF. Short theta burst stimulation to left frontal cortex prior to encoding enhances subsequent recognition memory. *Cogn Affect Behav Neurosci* 2016; **16:** 724–35.

78 Feldstein Ewing SW, Tapert SF, Molina BS. Uniting adolescent neuroimaging and treatment research: recommendations in pursuit of improved integration. *Neurosci Biobehav Rev* 2016; **62:** 109–14.