INTRODUCTION

Since developmental psychopathology has become the dominant paradigm in the study of the origins and maintenance of psychopathology [1], it is not surprising that it has made numerous major contributions to the reconceptualization of substance use disorders (SUDs) within a developmental framework [2]. One of the most important contributions is the identification of multiple pathways leading to the development of SUDs [2]. This short communication will present a brief explication of one such pathway model which, despite the fact that it is arguably one of the most prominent models for the development of externalizing behavior, has yet to be accorded sufficient recognition in the SUD literature. That model is the Ontogenic Process Pathway Model (OPPM) of externalizing psychopathology developed by Theodore Beauchaine and colleagues [3].

The Ontogenic Process Pathway Model of Externalizing Psychopathology

This model is rooted in the bedrock principle of developmental psychopathology that all psychopathologies are a developmental outcome of a complex longitudinal transaction between certain biological vulnerabilities and environmental risk factors in which there is a bidirectional effect [1]. Hence the designation ontogenic process. OPM is based on the finding from numerous studies that early-starting male antisocial behavior [1] that tends to be life course persistent begins with temperamental trait impulsivity (behavioral and emotional) as the core predisposing biological vulnerability [3]. This trait vulnerability is first manifested in preschool as a temperamental disposition to diminished effortful control with its most extreme expression being Attention-Deficit/Hyperactivity Disorder (ADHD). This vulnerability can progress into increasingly severe manifestations of antisocial behavior in the following rough temporal sequence depending upon transactions with various environmental risk factors such as coercive parenting, association with antisocial peers, neighborhood criminality.

The sequence of disorders following Attention-Deficit/Hyperactivity Disorder are Oppositional Defiant Disorder in elementary school, Conduct Disorder in middle school, and Substance Use Disorder in adolescence/early adulthood. In conclusion, the model presents a developmental pathway leading to substance abuse that is solidly anchored in developmental theory and robustly supported by empirical research.
or activate behavior when one does not want to do so [8]. In short, EC is the temperamental disposition to exercise self-regulation when doing so is difficult [9]. The extreme expression of low EC in preschool resulting in clinical deficits is ADHD. ADHD in this article will refer to the ADHD which primarily presents as hyperactive/impulsive as it is this presentation that indexes the behavioral facet of trait impulsivity in the OPPM model [1].

The emotional facet of trait impulsivity [1] can be defined as the tendency for "excessive and inappropriate expression of emotions and the rapid and poorly controlled shifts in emotion (lability)" [10]. It can be manifested clinically in symptoms such as irritability, temper outbursts, low frustration tolerance, and reactive aggression [10]. Recently, a strong case has been made for emotional impulsivity being a core symptom of ADHD on a par with behavioral impulsivity [11]. Thus those with ADHD will be as impulsive in their emotions as they are in their behavior.

In conclusion, behavioral and emotional trait impulsivity manifested in an extreme form in ADHD is the primordial biological vulnerability which can progress into more serious forms of antisocial behavior contingent upon transactions with precipitating environmental risk factors. As Molina and Pelham [12] concluded in their comprehensive review of ADHD and SUD, a consistent finding "replicated across many studies is the prediction of substance use from a constellation of temperament and personality traits that overlaps almost entirely with the defining symptoms of attention-deficit/hyperactivity disorder."

Oppositional Defiant Disorder

The progression from ADHD to ODD is best explained by the core ADHD symptoms of behavioral and emotional impulsivity which greatly increase the risk for coercive, oppositional interchanges [11,13]. Indeed, it is estimated that a typical child with ADHD has an astonishing half a million of these negative interchanges each year [14]. Hence it is not surprising that ODD, with a comorbid rate of 52% with ADHD, is the most common comorbid condition of ADHD in juveniles [15]. Comorbid ADHD/ODD in turn precipitates an additional adverse process which incrementally increases the risk for SUD — academic failure [12]. This is not surprising since children who by definition have trouble paying attention and have developed the disruptive behaviors of ODD are likely to have learning difficulties. Thus ADHD is commonly associated with clinically significant academic impairment as prevalence rates for learning and achievement problems range from 50 to 80% [16]. Furthermore academic underachievement in childhood and adolescence predicts later substance use even after controlling for delinquency and association with drug using peers [12].

Conduct Disorder

Although highly correlated, ODD and CD are different enough to warrant consideration as different dimensions of deviant behavior [17]. In this progression in the worsening antisocial pathway, ODD’s role as a developmental precursor to CD is well documented [18]. It is now understood that far from being a benign, milder form of CD, ODD plays a key role in the development of CD and is one of the strongest predictors of the onset of CD and of the course of CD symptoms over time [19]. In addition, although the majority of children with ODD do not go on to develop CD [19], if childhood-onset CD develops, it is almost always preceded developmentally by ODD [20]. This development from ODD to CD is most likely to occur when environmental risk factors such as maltreatment, neglect, hostile parenting, neighborhood violence and other forms of adversity increase rather than decrease the deviance proneness of ADHD/ODD [3,18,20]. Furthermore, CD along with the previously discussed academic failure precipitates what is one of the best predictors of SUD — association with deviant peers [21]. There is substantial evidence from numerous studies that antisocial youth selectively affiliate with deviant peers and this association in turn provides new opportunities to engage in antisocial behaviors such as the use and abuse of various substances [22].

Substance Use Disorder

The foregoing developmental cascade markedly increases the risk for SUDs as CD confers a six-fold increase in risk for developing an SUD [4]. The co-occurrence of heavier substance use and CD is a well-documented finding as is the emergence of conduct problems prior to substance use in children with ADHD [12, 22]. Indeed CD is the “best known correlate of ADHD that is omnipresent in discussions of SUD risk” [12]. For example, in a recent longitudinal study growth in ADHD symptoms over childhood was the strongest predictor of growth in CD symptoms, which in turn was the most consistent predictor of adolescent substance use [23].

CONCLUSION

In conclusion, it is “A sobering fact: ADHD leads to substance abuse [24].” Hence, it is not surprising that aggregate data from studies in the United States yield prevalence rates ranging from 38% to 50% of ADHD among adolescents in treatment for SUD [25]. The OPPM model presents a developmental pathway that is solidly anchored in developmental theory and robustly supported by empirical research. There are of course other pathways that lead to SUDs [2] and other biologically based vulnerabilities that increase risk for SUDs [4]. However this model articulates a more detailed, comprehensive developmental sequence that is more closely linked to research findings than other deviance proneness models (see reference 2 for a discussion of such models). It therefore merits serious consideration in the continuing study of the multiple pathways within a developmental framework that lead to SUDs.

REFERENCES


