Prevention of Male Life Course Persistent Antisocial Behavior

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Abstract

The prevention of mental, emotional, and behavioral problems in young people is a national mental health priority. Of all the various problems, this paper argues that the prevention of male life-course-persistent antisocial behavior (MLCPAB) should assume the highest priority. MLCPAB refers to the childhood onset of severe overt conduct problems such as physical aggression, opposition-defiance, and rule-breaking. These symptoms can emerge from early neurodevelopmental risk factors such as Attention Deficit Hyperactivity Disorder (ADHD) and family adversity risk factors. MLCPAB can greatly increase the patient's risk for delinquency, adult criminality, and a host of other problems. If prevention is to be effective, it will benefit by having three components: intensity, early beginning, and continuation over the long-term. This paper makes the case that ADHD is a major risk factor in the developmental sequence that leads to MLCPAB. Because there is extensive evidence that ADHD can be successfully treated, it follows that the early, intensive treatment of ADHD may be an effective means of preventing development of MLCPAB.

The Institute of Medicine, along with the National Research Council, has recently issued a call to make the prevention of mental, emotional, and behavior problems (MEB) in young people a high national priority (National Research Council and Institute of Medicine, 2009). MLCPAB refers to the childhood onset of severe overt conduct problems such as physical aggression, opposition-defiance, and rule-breaking that can emerge from early neurodevelopmental (e.g., Attention Deficit Hyperactivity Disorder). Family adversity risk factors greatly increase the risk for delinquency, adult criminality, and a host of other problems (Moffitt, 2003, 2006; Moffitt et al., 2008; Tremblay, 2010). For comprehensive discussions of the many factors related to the development of MLCPAB see Farrington (2009), Murray and Farrington, (2010), Moffitt (2003, 2006), and Tremblay (2010).

Of all the various MEB problems, a strong argument was made that the prevention of male life-course-persistent antisocial behavior (MLCPAB)-assume the highest priority (Eme, 2010). The focus on MLCPAB is significant as males are astonishingly 10 to 14 times more likely than females to develop life-course-persistent antisocial behavior (Moffitt, 2003, 2006). Indeed, of all the multiple bio-psycho-social risk factors for the development of severe conduct problems, "maleness" is by far the most robust predictor (Tremblay, 2010). Unfortunately, the prevention of MEB is not a national priority. As the National Research Council and Institute of Medicine (2009, p. xiii) observed, "Myopically, we devote minimal attention to preventing future disorders" and simply await their emergence and then attempt to treat them. For example, Richard Tremblay (2008, 2010), one of the foremost researchers on the developmental origins of

violent behavior, underscored the importance of distinguishing between normative physical aggression (which peaks between 2 and 4 years of age) and cases of abnormal, severe physical aggression, which can be identified in preschool. His research found that despite the fact that "most cases of chronic physical aggression were cases of chronic physical aggression during early childhood" (Tremblay, 2010, p.346), the majority of interventions are not initiated until years later during preadolescence and adolescence. Hence, it is not surprising that in the past half-century we have failed to reduce the rates of MLCPAB or adult criminal offending behavior (Dekovic et al., 2011; Frick & White, 2008).

If prevention of MLCPAB is ever to be effective, it will benefit by having three components: intensity, early beginning, and continuation over the long-term (Tremblay, 2010). This paper makes the case that ADHD is a major risk factor in the developmental sequence that leads to MLCPAB. Because there is extensive evidence that ADHD can be successfully treated, it follows that the early, intensive treatment of ADHD may be an effective means of preventing development of MLCPAB. A developmental perspective is examined as many disorders are risk factors for later disorders; therefore, treatment that prevents the development of another disorder can be considered to be a "prevention" (National Research Council and Institute of Medicine, 2009). This is the definition that will be adopted for the expository purposes of this paper.

ADHD and Conduct Disorder

ADHD, a disorder of self-control/self-regulation that results in extreme, age-inappropriate impairing symptoms of impulsivity, hyperactivity, and inattention (Barkley, 2006), is the most common neurodevelopmental disorder in juveniles (American Academy of Pediatrics, 2011). The prevalence estimate for juveniles in the United States aged 4-17, based upon parental report of ever having been diagnosed with ADHD, is 9.5% (13.2% male, 5.6% female) (Visser, Bitski, Danielson, & Perou, 2010). Furthermore, ADHD is highly comorbid with Conduct Disorder (CD). For example, in studies of clinic-based samples — which are the most relevant studies both because they represent more severe cases (Beauchaine, Hinshaw, & Pang, 2010; Frick & Moffitt, 2010; Gatzke-Koop et al., 2009; Klein et al., 1997; Lahey Loeber, Burke, & and Applegate, 2005; McMahon & Frick, 2007) and because the results of these studies generalize to the population of children whose parents seek help for treatment (Molina, 2011) — the vast majority of males with CD are comorbid for ADHD (Beauchaine, Hinshaw, & Pang, 2010; Frick & Moffitt, 2010).

Scientifically, the very high comorbidity between CD and ADHD does not necessarily mean that ADHD is a risk factor for the development of CD. However, there is a strong consensus among researchers' whose documentation states that there is substantial research that supports that the idea that ADHD is indeed a significant risk factor for the development of CD (Frick, 2012; Frick & Marsee, 2006; Loeber, Burke, & Pardini, 2009; Loeber & Burke, 2011; Waschbusch, 2002). This literature indentified three developmental pathways that lead to CD (Frick, 2012). A developmental pathway is the "orderly behavioral development between more than two problem behaviors with individuals differing in their propensity to progress along the successive problem behavior represented by the pathway during development" (Loeber & Burke, 2011, p. 34). Note that the conceptualization of a developmental pathway for CD is not deterministic, but refers to a propensity, a probability that CD will develop (Loeber & Burke,

2011). One developmental pathway that leads to CD begins with high levels of emotional and behavioral dysregulation that result in problems in the executive control of behavior (Frick, 2012). This is essentially a description of ADHD that predominantly presents with hyperactive-impulsive symptoms (Barkley, 2006, 2010; Willcutt et al., 2012). Note that this paper will adopt the proposed revision for the DSM-5 (American Psychiatric Association, 2012) to change subtypes to a specifier for how ADHD presents. In this model, ADHD behaviors emerge first, followed by Oppositional Defiant Disorder (ODD) behaviors reflecting a pattern of negativistic, defiant, disobedient, and hostile behavior towards authority figures. These behaviors are followed by more severe conduct behaviors. These CD behaviors reflecting a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal rules or norms are violated (Burke, Loeber, Lahey, & Rathouz, 2005; Connor, & Doerfler, 2008; Loeber, Burke, & Pardini, 2009; Waschbusch, 2002).

ODD as a Developmental Precursor to CD

The role of Oppositional Defiant Disorder (ODD) as a developmental precursor to CD has been well documented (Frick & Marsee, 2006; Lahey et al., 2005; Moffitt et al., 2008). Moreover, it is now understood that far from being simply 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 (Loeber et al., 2009; Loeber & Burke, 2011). Although the majority of children with ODD do not develop CD (Loeber, Burke, & Pardini, 2009), if childhood onset CD develops, it is almost always preceded developmentally by ODD (Burke et al., 2010). In addition, there is emerging evidence that there are subdimensions of ODD symptomatology that are not equally associated with the risk of developing CD (Pardini, Moffitt, & Frick, 2010; Rowe et al., 2010). The symptoms that indicate negative affect predict internalization of problems whereas oppositional symptoms such as often argues with adults, often actively defies or refuses to comply with adult's requests or rules, which indicate a "headstrong" dimension, tend to predict CD (Rowe et al., 2010; Stringaris & Goodman, 2009). The headstrong dimension of ODD has been found to be associated with ADHD (Stringaris & Goodman, 2009) and thus provides an apt segue to consider the role of ADHD in the development of ODD.

ADHD as a Developmental Precursor to ODD

ODD, which has a comorbidity rate of 52% with ADHD, is the most common comorbid condition of ADHD in juveniles (Willcutt et al., 2012). This comorbidity is best explained by the core impairments of behavioral and emotional impulsivity in ADHD (Barkley, 2006, 2010a, 2010b). Behavioral impulsivity is generally recognized as such an impairment in ADHD (Willcutt et al., 2012). In addition to behavioral impulsivity, it is now recognized that emotional impulsivity/dysregulation is also a core impairment (Barkley, 2010a, 2010b) These twin impairments commonly result in symptoms such as irritability, impatience, anger, low frustration threshold, and reactive aggression (Barkley, 2010a; Frick & Viding, 2009). This combination greatly increases the risk for coercive, oppositional interchanges (Barkley, 2006; Burns & Walsh, 2002; Lahey & Waldham, 2008; van Lier, van der Ende, Koot, & Verhulst, 2007). Indeed, it is estimated that the typical child with ADHD has an astonishing half-a-million of these negative interchanges each year (Pelham &Fabiano, 2008), thereby adding support to Barkley's (2010a)

assertion that having Attention-Deficit/ Hyperactivity Disorder-Combined Type ADHD-C virtually creates a borderline case of ODD in children.

Finally, perhaps the most persuasive evidence that ADHD increases the risk for ODD and thereby increases the risk for CD, comes from the most recent findings of the Dunedin Multidisciplinary Health and Development study (Moffitt, et al., 2011) This longitudinal study that followed a complete birth cohort of 1,037 children from birth to age 32, found that self control assessed at age 3 predicted criminal offending at age 32. When the sample was segmented into the highest and lowest fifths on preschool self-control, the lowest fifth had much higher crime conviction rates as adults than the highest fifth: 43% vs. 13%. The instruments used to assess self-control were essentially measures of the core features of the behavioral and emotional impulsivity that characterize ADHD-C (behaviors of hyperactivity, impulsivity, inattention, lack of persistence, impulsive aggression, low frustration tolerance). The study found that preschool children with many symptoms of ADHD were at high risk for criminality compared to those without such symptoms. The study concluded that these findings provided impressive support of the need for preschool intervention programs that enhance self-control.

In conclusion, Sibley and colleagues (2011), in their discussion of the developmental progression of ADHD that leads to serious delinquency, articulated their consensus that "most agree with the hypothesis that this troubling path begins with impulsivity, ADHD, undercontrolled temperament, or some variant thereof" (p.22).

Treatment of School-Aged Children with ADHD

Stimulant medication is the first line of treatment for school-aged children (6-11) with ADHD (American Academy of Pediatrics, 2011; Pliszka, 2009). This approach is supported by hundreds of studies that have clearly documented the robust short-term efficacy (2-8 weeks) of stimulant treatment for core ADHD symptoms of inattention, impulsivity and hyperactivity for both sexes (American Academy of Pediatrics, 2011; Biederman & Spencer, 2008; Connor, 2006; Hinshaw, 2007; Pliszka, 2009). These studies have also found that the core symptoms of ADHD in children with comorbid ADHD and ODD/CD respond as well to stimulant treatment as do the symptoms in children with ADHD alone (Pliszka, 2009). The effect size of the response of stimulants relative to placebo is close to 1.0, thereby making stimulants among the most efficacious medications in all of health care, rivaling antibiotics in this regard (Pliszka, 2009).

Stimulants also have similarly robust effects on oppositional/defiant behaviors and overt aggression (Connor 2006; Connor, Glatt, Lopez, Jackson, &Melloni, 2002; Pliszka, 2009), especially when an optimal medication regimen is combined with behavior therapy (Blader, Pliszka, Jensen, Schooler, & Kafantaris, 2010). Perhaps the best indication of the efficacy of treating children comorbid for ADHD/ODD came from the Multisite Multimodal Treatment Study for ADHD (MTA: citation?). The MTA was the largest and most comprehensive study of both stimulant medication and behavior therapy of children with ADHD that has ever been conducted. It was a randomized clinical trial of four treatment strategies for 576 children aged 7-9 with ADHD: medication management, behavioral treatment, the combination of these two, and an active control condition based on usual treatment available in the community. It has generated vast literature since its publication in 1999, which is beyond the scope of this article to review.

The reader who is interested in this literature can consult comprehensive summaries in Molina et al. (2009) and Swanson et al. (2008a, 2008b). In the MTA study, 40% of the participants also had had ODD or CD (Smith, Barkley, Shapiro, 2006). Fourteen months of combined stimulant and behavior treatment resulted in a success rate of 68% (defined as functioning in the normal range), in contrast to a 25% success rate for the community control group (Swanson et al., 2001). Thus children with ADHD who are comorbid for ODD and CD can be very successfully treated with a combination of medication management and behavioral treatment for an extended period of time.

Treatment of Preschool-Aged Children with ADHD

Despite concerns expressed by some there is a strong consensus among multiple expert reviews that: a) preschool ADHD can be accurately diagnosed, b) patterns of impairments and the rates of comorbidity associated with the ADHD, especially severe ADHD, are similar if not worse than those seen in later childhood, and c) severe ADHD is a good predictor of ADHD later in life (American Academy of Pediatrics, 2011; Chacko Wakschlag, Hill, Danis, & and Espy, 2009; Dupaul & and Kern, 2011; Egger & and Angold, 2006; Keenan, et al., 2010; Sonuga-Barke &and Halperin, 2010; Sonuga-Barke, Koerting, Smith, McCann, & Thompson, 2011). Therefore treatment is warranted. A review of five studies of behavioral treatment of ADHD in preschool children (Murray, 2010) along with a more recent study (Webster-Stratton, Reid, & Beauchaine, 2011) have provided support for the short-term success of this type of intervention. If behavioral interventions do not provide significant improvement, then stimulant treatment should be considered (American Academy of Pediatrics, 2011). With regard to stimulant treatment, prior to the 2006 reporting of the largest and most sophisticated clinical trial in preschoolers to date, the Preschool ADHD Treatment Study (PATS) (Marsh, 2011), there were only 10 single-site, small randomized studies of stimulant treatment (methylphenidate) involving a total of 240 preschoolers with ADHD (Greenhill et al., 2006). The PATS, with 303 participants aged 3 to 5.5, had the largest sample ever of preschoolers with ADHD (Greenhill, Posner, Vaughan, &Kratochvil, 2008). In the crucial phase of the study, 165 preschool children with severe ADHD, 52% of whom were comorbid with ODD, were randomly assigned to a 5 week, doubleblinded crossover-titration trial with immediate release methylphenidate. The trial resulted in clinically meaningful improvement in ADHD and ODD symptoms for both sexes, though the effect sizes were smaller (0.4 to 0.8 compared to placebo) than those reported in the 14 month MTA study (0.5 to 1.3) (Greenhill et al., 2008). Although this may indicate that preschoolers benefit less from stimulant treatment than do their school age counterparts, there is an alternate plausible interpretation. The reduced efficacy may have been due to the greater severity of the ADHD symptoms in preschoolers than in the MTA study, and the reluctance to use higher levels of medication, which may have reduced optimal treatment (Greenhill, et al., 2008).

Lastly, the issue of possible "over-medication" of children with ADHD seems, in general, to be unwarranted. First, despite the fact that stimulant medication is the first line of treatment for juveniles with ADHD (American Academy of Pediatrics, 2011; Pliszka, 2009), in the most recent nationally representative sample of juveniles (4-17) currently diagnosed with ADHD, only 66.3% were taking medication (Visser et al., 2010). Second, the MTA study found that a substantial majority of children (274/406 or 62%) who were successfully treated with stimulants,

ceased taking the medication in adolescence (Molina et al., 2009). Thus the primary issue is under-rather than over-medication.

Conclusion

For the prevention of MEB to be a higher priority, a recommended component is the identification and treatment of preschool ADHD. This disorder clearly places a child at high risk for developing ODD, the gateway to disorder to what is arguably the most important of the MEB problems, MLCPAB. ADHD can be accurately diagnosed and effectively treated in preschool children. The major limitation to current treatment studies is that the extent to which clinical improvements of early, intensive treatment of ADHD can correct long-term negative developmental trajectories is currently unknown (Sonuga-Barke Halperin, 2010). Nevertheless, this does not gainsay the impressive evidence that such intervention should be an essential component of efforts to increase self-control in preschool children (Moffitt, Arseneault, Belsky, et al., 2011; Sonuga-Barke & Halperin, 2010). Screening for preschool ADHD should be implemented in primary medical care settings and should be a part of the mental health assessment mandated by Head Start (National Research Council and Institute of Medicine, 2009). Such screenings and subsequent interventions can be informed by the model Program for Early Detection and Intervention in ADHD (Sonuga-Barke et al., 2011), which was developed to identify and treat preschool children with ADHD who were most at risk for poor outcomes. This model is in the process of being implemented in Great Britain. If such programs prove successful in significantly reducing the long-term negative developmental trajectories of children with severe ADHD/ODD and thus MLCPAB, they would pay huge dividends by reducing a panoply of societal costs, saving taxpayers money, and promoting prosperity (Moffitt et al., 2011).

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