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Authors

Atherton, Olivia E
Lawson, Katherine M
Ferrer, Emilio
et al.

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The Role of Effortful Control in the Development of ADHD, ODD, and CD Symptoms

Olivia E. Atherton, **Katherine M. Lawson**, **Emilio Ferrer**, **Richard W. Robins**

University of California, Davis

Abstract

Many adolescents have difficulty regulating their impulses and become prone to externalizing problems (e.g., ADHD, ODD, and CD) and other adverse consequences. Using multi-method data from a longitudinal study of Mexican-origin youth (*N*=674), assessed annually from age 10 to 16, we examined the relations between effortful control and ADHD, ODD, and CD symptoms over time. Bivariate latent growth curve models showed negative correlations between the trajectories of effortful control and ADHD, ODD, and CD, indicating that steeper *decreases* in effortful control were related to steeper *increases* in ADHD, ODD, and CD symptoms. Using a novel statistical technique, the Factor of Curves Model (FOCUS), we found that ADHD, ODD, and CD share a common "externalizing" trajectory during adolescence. Although effortful control was strongly associated with this common trajectory, it had few unique associations with the individual disorder trajectories, above and beyond their shared trajectory. When we extended the FOCUS model to include the effortful control trajectory as an indicator, we found that ADHD and ODD had strong loadings, whereas effortful control and CD had comparatively weak loadings on the shared developmental trajectory. Follow-up analyses showed that a two-factor solution, with externalizing symptom trajectories on one factor and the effortful control facet trajectories on a separate factor, was a better fit to the data than a one-factor solution. Finally, parent ASPD symptoms were related to *increases* in CD, but had no significant influence on effortful control, ADHD, or ODD. We discuss the implications for personality and externalizing problem development.

Keywords

personality development; effortful control; ADHD; ODD; CD

Adolescence is a time of significant self-regulatory challenges, when youth are often enticed by new opportunities to engage in drug use, risky sexual behavior, delinquency, and other externalizing problems (Moffitt, 1993). With myriad avenues to engage in problem behaviors, adolescents' capacity to regulate their behavior is continually tested. Effortful control, the temperamental core of self-regulation, refers to the propensity to regulate one's impulses and behaviors, to motivate the self towards a goal when there are competing desires, and to focus and shift attention easily (Rothbart & Bates, 2006). Although some

Corresponding author: Olivia E. Atherton, Department of Psychology, University of California, Davis, One Shields Ave., Davis, CA 95616, oeatherton@ucdavis.edu.

youth are able to effectively regulate their behavior during adolescence, others fail to control their impulses and consistently succumb to temptation, often leading to dire consequences in school, peer, family, and neighborhood contexts. Thus, adolescence may be a crucial developmental period when individuals with poor effortful control are vulnerable to increased levels of externalizing problems.

The present study used data from a longitudinal study of Mexican-origin youth to examine how adolescents develop (or fail to develop) the ability to self-regulate via the associations between effortful control and three forms of externalizing problems: AttentionDeficit/ Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD) symptoms. Although ADHD was recently re-classified as a neurodevelopmental disorder in the DSM-5, it is well-established that ADHD is highly comorbid with other externalizing problems and should continue to be included in investigations of disruptive behavior symptoms (indicative of ODD and CD), in order to avoid obfuscating our understanding of externalizing problems in childhood and adolescence (Beauchaine & McNulty, 2013).

We addressed five fundamental questions in the present study: (1) What are the co-developmental associations between effortful control and ADHD, ODD, and CD symptoms from late childhood (age 10) through adolescence (age 16)? That is, do individuals who increase (or decrease) in effortful control during adolescence tend to increase (or decrease) in ADHD, ODD, and CD symptoms?; (2) Do ADHD, ODD, and CD symptoms share a common externalizing developmental trajectory over the course of adolescence?; (3) Does effortful control have unique associations with each individual trajectory (ADHD, ODD, and CD), or is the relation better explained by a common “externalizing” trajectory?; (4) Do effortful control and externalizing symptoms have a shared developmental trajectory?; and (5) Does parent ASPD symptoms serve as a shared antecedent for the development of effortful control and ADHD, ODD, and CD symptoms?

A Developmental Psychopathology Approach

A developmental psychopathology approach conceptualizes psychopathology as a divergence from normative development, and aims to understand how and why individuals diverge from normative development as a function of risk and protective factors ranging from biological processes to sociocultural factors (e.g., Cicchetti & Rogosch, 1996). Recently, Durbin and Hicks (2014) have advanced a new perspective that integrates developmental psychopathology with research and theory on personality development. From this perspective, psychopathology is conceptualized as a deviation from normative *personality* development, and large deviations from these normative personality trends may indicate periods of heightened risk for psychopathology. For example, if an adolescent experiences a particularly pronounced decrease in effortful control during adolescence, it may be much harder for him/her to recover or mature in preparation for the demands of adult life, leading to the emergence of psychiatric problems during young adulthood. In particular, this decrease in effortful control may put youth at risk for engaging in multiple forms of antisocial behavior because they are less capable of avoiding the developmental “snares” (e.g., school dropout) commonly encountered during adolescence (Moffitt, 1993). Moreover,

experiencing developmental snares could not only impede a person's advancement towards personality maturity, but also create lasting problems for the individual. Thus, by investigating how effortful control and multiple forms of externalizing problems co-develop, we can better understand the processes that underlie normative and non-normative self-regulation development and the cascading effects this has on behavioral problems. To understand what would be considered deviations from normative personality and psychopathology development, it is critical to characterize how effortful control and ADHD, ODD, and CD symptoms change, on average, over time.

Development of effortful control.

Previous research has shown that self-regulatory traits (e.g., effortful control, inhibitory control, conscientiousness) generally increase over the course of childhood, presumably due to improvements in cognitive, motor, and social functioning (Eisenberg, Duckworth, Spinrad & Valiente, 2012; King, Lengua, & Monahan, 2013; Kochanska, Murray, & Harlan, 2000; Li-Grining, 2007; Van den akker, Dekovic, Asscher, & Prinzie, 2014; Vazsonyi & Huang, 2010). However, it is not clear whether this pattern of increasing self-regulation continues across adolescence. From early to late adolescence, some researchers have found *increases* in various self-regulatory traits (Branje, van Lieshout, & Gerris, 2006; Donnellan, Conger, & Burzette, 2007; Roberts, Walton, Viechtbauer, 2006), whereas other researchers have found *no change* (Klimstra et al., 2009; Laceulle et al., 2012). Interestingly, a growing body of research suggests that some self-regulatory traits may even decrease, producing a temporary "self-regulatory dip" during adolescence (Borghuis et al., 2017; De Fruyt et al., 2006; Klimstra et al., 2009; Soto et al., 2011; Soto & Tackett, 2015; Van den akker, Dekovic, Asscher, & Prinzie, 2014), before these traits resume their well-established, mean-level *increases* across adulthood (Roberts et al., 2006). Given the mixed evidence regarding mean-level changes in self-regulation during adolescence, more research is needed to better understand the normative trajectory of self-regulatory traits during this critical developmental period.

Development of externalizing problems.

Externalizing problems are defined as maladaptive behaviors directed toward the external environment (American Psychological Association [APA], 2013). Externalizing problems typically emerge in childhood and peak in adolescence, with some youth desisting from externalizing behaviors following adolescence and others persisting throughout the life course (Moffitt, Caspi, Rutter, & Silva, 2001). The "triad" of youth externalizing problems encompasses symptomatology related to ADHD (i.e., inattention and hyperactive-impulsive behaviors), ODD (i.e., defying authority, easily losing one's temper, and acting vindictively towards others), and CD (i.e., severe externalizing problems such as theft, destruction of property, and physical assault; APA, 2013). Although there is some debate about the developmental trajectories of these three forms of externalizing symptoms, research has shown that ADHD symptoms generally decline from childhood through adolescence (Atherton, Ferrer, & Robins, in press; Pingault et al., 2015; Willoughby, 2003), ODD symptoms slightly increase from late childhood to mid-adolescence, before declining later in adolescence (Atherton, Ferrer, & Robins, in press; but see Tremblay et al., 2013; van Lier,

van der Ende, Koot, & Verhulst, 2007), and CD symptoms largely increase over the course of adolescence (Atherton, Ferrer, & Robins, in press; Moffitt, 2006).

Concurrent and Longitudinal Associations between Effortful Control and Externalizing Problems

A growing body of research has demonstrated robust, *concurrent* associations between low levels of effortful control and symptoms of ADHD (Beauchaine & McNulty, 2013; Foley, McClowry, & Castellanos, 2008; Martel & Nigg, 2006; Martel et al., 2014; Nigg et al., 2002), ODD (Martel, Gremillion, & Roberts, 2012) and CD (Eisenberg et al., 2009; Murray & Kochanska, 2002; Vazsonyi & Huang, 2010). These associations have been documented in early childhood (Dennis & Brotman, 2003; Lavigne et al., 2012; Lavigne et al., 2015; Martel, Roberts, & Gremillion, 2013; Martel, Gremillion, & Roberts, 2012), middle childhood (Martel & Nigg, 2006), and adolescence (Graziano et al., 2015; Samyn, Roeyers, Bijttebier, & Rosseel, 2015; Wolff et al., 2016). However, these concurrent associations tell us little about whether low effortful control and externalizing problems *co-develop* over time.

To answer questions about co-development, it is necessary to use prospective longitudinal studies. Prior longitudinal studies have found prospective associations between low effortful control and externalizing problems across early childhood (Choe, Olson, & Sameroff, 2013; Lavigne, Gouze, Hopkins, & Bryant, 2016; Olson, Choe, & Sameroff, 2016) and mid- to late-childhood (Eisenberg et al., 2004; Eisenberg et al., 2005; Lengua, 2006; Valiente et al., 2006; Zalewski et al., 2011). However, we know of only one longitudinal study conducted during adolescence, which found that lower levels of effortful control at age 11 were associated with higher levels of antisocial behavior at age 13 (Veenstra, Lindenberg, Verhulst, & Ormel, 2009).

Despite the dearth of research on effortful control and externalizing problems over time, there has been some research on the longitudinal associations between other self-regulatory traits (e.g., disinhibition and conscientiousness) and externalizing problems. A large body of research has documented positive concurrent correlations between disinihibition/low conscientiousness and externalizing problems among adolescents (Bogg & Finn, 2010; Castellanos-Ryan et al., 2016) and adults (Carlson, Pritchard, & Dominelli, 2013; Miller & Lynam, 2001; Ruiz, Pincus, & Schinka, 2008). Longitudinal work has found that disinhibition in early childhood and adolescence predicts externalizing problems in late adolescence and adulthood (Caspi, Moffitt, Newman, & Silva, 1996; Henry, Caspi, Moffitt, & Silva, 1996; Krueger, 1999). Additionally, changes in conscientiousness across adolescence predict later externalizing problems (van der Akker, Dekovic, & Prinzie, 2010). Last, research has also shown that meta-traits, like ‘stability’ (a composite of Conscientiousness, Agreeableness, and Neuroticism) negatively predict externalizing behavior among adolescent boys (DeYoung, Peterson, Seguin, & Tremblay, 2008). Together, this work suggests that multiple self-regulatory traits are associated with externalizing problems concurrently and longitudinally. However, to our knowledge, no work has examined how *changes* in self-regulatory traits are associated with *changes* in externalizing

problems across adolescence, leaving open many questions about the co-development of effortful control and ADHD, ODD, and CD symptoms across adolescence.

Theoretically, it is possible that low levels of effortful control predispose an individual to develop externalizing problems. Conversely, it is also possible that the experience of externalizing problems leads youth to develop poor effortful control. Thus, longitudinal relations between effortful control and externalizing problems may indicate a vicious cycle of adolescents experiencing increasingly more problem behaviors and worsening impulse control over time, leading their developmental trajectories to be correlated across individuals.

A Common Developmental Trajectory of Externalizing Problems and Associations with Effortful Control

An extensive body of research now indicates symptoms of psychopathology are best organized within a dimensional taxonomy, rather than by distinct diagnostic categories (Caspi et al., 2014; Kotov et al., 2017). Of particular relevance to the present study, previous work has shown that ADHD, ODD, and CD symptoms have few distinctive features that set them apart from one another, and should therefore be thought of as interrelated symptoms indicative of a broader ‘externalizing’ spectrum (Achenbach & Edelbrock, 1984; Beauchaine & McNulty, 2013; Tackett, 2010). This perspective has been challenged in two ways. First, some researchers have argued that ADHD is separable (both conceptually and empirically) from ODD and CD, which has led to its reclassification as a neurodevelopmental disorder, rather than an externalizing disorder, in the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM). Second, others have argued that the externalizing disorders are best understood as a developmental sequence, with youth progressing over time from ADHD to ODD, and then to CD (Beauchaine & McNulty, 2013). Moreover, recent research has shown that these disorders predict change over time in each other, implying that youth may be *accumulating*, or *co-developing*, multiple forms of externalizing problems simultaneously (Atherton, Ferrer, & Robins, in press). Thus, although concurrently it is clear that ADHD, ODD, and CD symptoms comprise a broader externalizing spectrum, very little longitudinal research has been done on the trajectories of these symptom clusters to determine whether ADHD, ODD, and CD are distinct developmental entities or three interrelated components of a common externalizing trajectory.

If ADHD, ODD, and CD are simply components of a broader externalizing construct, then there should be some underlying “externalizing core” that leads ADHD, ODD, and CD symptoms to develop through a shared developmental process. The present study provides the first test of how well the developmental trajectories of ADHD, ODD, and CD symptoms can be captured by a single, common trajectory, using a relatively novel statistical technique, the factor of curves (FOCUS) model (Isordia, Conger, Robins, & Ferrer, 2017). The FOCUS model is similar to a hierarchical factor model, but extended longitudinally, so that it models both a common developmental trajectory for all constructs, as well as separate construct-specific trajectories for each disorder. To better understand how and why effortful control and externalizing problems are related, it is also important to investigate whether effortful

control is associated with the specific manifestation of each disorder (ADHD vs. ODD vs. CD symptoms) versus a general "externalizing" factor that subsumes any and all unique associations with a particular disorder (Widiger & Smith, in press). Therefore, we extended the FOCUS model to investigate the degree to which effortful control is associated with this common externalizing trajectory, and whether it predicts the trajectory of each individual disorder after controlling for the common externalizing trajectory.

Effortful Control and Externalizing Problems May Be Part of the Same Latent Continuum

Thus far, we have discussed effortful control and ADHD, ODD, and CD symptoms as if they are distinct constructs. However, proponents of a hierarchical taxonomy of psychopathology often argue that traits like effortful control, conscientiousness, and disinhibition are lower-level indicators of the externalizing spectrum (Kotov et al., 2017; Krueger & Tackett, 2013; Widiger et al., 2018). In other words, effortful control and externalizing problems should be considered part of the same continuum (or spectrum), where effortful control measures capture the low range and externalizing measures capture the high range of the same latent continuum (De Bolle et al., 2012; Kotov et al., 2017). From this perspective, effortful control and externalizing problems are not distinct constructs.

Empirical evidence for the spectrum hypothesis stems primarily from behavioral genetics and neurobiological data suggesting that temperament/personality traits and externalizing problems have shared genetic, psychophysiological, and neurobiological roots (Beauchaine & McNulty, 2013; Fox et al., 2005; Iacono, Malone, & McGue, 2003; Krueger et al., 2002; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Nelson & Trainor, 2007; Vrieze, Perlman, Krueger, & Iacono, 2012; Young et al., 2009). Additionally, researchers have used statistical methods, such as item-response theory (IRT) and factor analysis, to examine the spectrum idea in relation to measures of conscientiousness and psychopathy/Conduct Disorder, and found that these measures were essentially capturing the same latent construct (Walton et al., 2008). However, to date, most of the relevant research on the spectrum hypothesis has used concurrent data in adult samples, which provides little insight into whether self-regulatory traits and externalizing problems are distinct *developmental* constructs (Durbin & Hicks, 2014; Tackett, 2010).

To examine whether effortful control and externalizing symptoms are part of the same underlying longitudinal spectrum, we expanded our FOCUS model to include effortful control as an indicator, along with ADHD, ODD, and CD. This model allows us to evaluate the extent to which all four constructs follow a common developmental trajectory over the course of adolescence. We also conducted confirmatory factor analyses to test whether a one- or two-factor model of the longitudinal trajectories of effortful control and externalizing symptoms provides the best fit to the data, in order to examine a developmental variant of the spectrum hypothesis.

Finally, we examined parent Antisocial Personality Disorder (ASPD) symptoms as one possible etiological factor that might similarly influence the development of effortful control and ADHD, ODD, and CD symptoms. Virtually no prior longitudinal research has

systematically examined developmental precursors to effortful control and externalizing problems to determine whether these constructs have a shared etiology (Durbin & Hicks, 2014). If effortful control and externalizing symptom measures are assessing a similar latent dimension, then they should have similar etiological risk factors (because they are, more or less, the same construct). Parents' ASPD symptoms are a commonly studied etiological factor of offspring externalizing problems, in part because they serve as both a genetic and an environmental proxy of problem behaviors and signify problems with self-regulatory capacities more generally (Krueger et al., 2002; Odgers et al., 2007; Vaughn, Salas-Wright, DeLisi, & Qian, 2015). Conceptually, parent ASPD symptoms may play an important etiological role in the development of the child's effortful control and/or externalizing symptom through shared genetic influences, direct environmental effects (e.g., parent and child are offending together), and/or indirect environmental effects (e.g., parental modeling and socialization). Parents with a history of antisocial behavior often have children who exhibit more externalizing problems (Farrington et al., 2001; Frisell, Leichenstein, & Langstrom, 2011; Vaughn, Salas-Wright, DeLisi, & Qian, 2015), including increased risk for ADHD (Galera et al., 2011; Harvey, Breaux, & Lugo-Candelas, 2016; Tandon et al., 2011), ODD (Frick et al., 1992), and CD symptoms (Moffitt et al., 2008; Odgers et al., 2007). However, other studies suggest that parent antisocial behavior only increases risk for CD symptoms, not ADHD or ODD symptoms (Biederman et al., 1996; Faraone et al., 2000; Loeber et al., 2000; Petty et al., 2009). Although past research supports a general relation between parent antisocial behavior and child externalizing problems, we know of no work that has investigated the influence of parent ASPD symptoms on the *development* of the child's effortful control and externalizing problems over the course of adolescence.

If parent ASPD symptoms predict effortful control and externalizing symptom (ADHD, ODD, and CD) trajectories in the same way, then this would provide some initial (albeit weak) evidence that effortful control and externalizing symptoms are similar constructs. However, there are alternative explanations. For example, a mediation pathway between all constructs may exist, whereby parent ASPD symptoms influence the child's effortful control tendencies, which subsequently influences the child's engagement in externalizing problems.

The Present Study

In the present study, we used data from a large sample of Mexican-origin youth ($N = 674$) assessed biennially from age 10 to 16 to examine how effortful control and externalizing problems are related over time. Specifically, we addressed the following five questions. First, is effortful control longitudinally associated with ADHD, ODD, and CD symptoms? We will test this question by examining how effortful control co-develops with ADHD, ODD, and CD symptoms via bivariate latent growth curve models, which specify correlations between the levels of effortful control and externalizing problems (i.e., concurrent associations), the slopes (i.e., correlations between trajectories), and the level of effortful control and slope of externalizing symptoms and vice versa (i.e., prediction of each trajectory). Second, do ADHD, ODD, and CD symptoms contribute to a shared externalizing trajectory over the course of adolescence? We will test this question by conducting a factor of curves (FOCUS) model (Isordia et al., 2017), which allows us to examine whether ADHD, ODD, and CD

symptoms share a common "externalizing" trajectory. Third, is effortful control associated with the individual ADHD, ODD, and CD trajectories independent of their shared externalizing trajectory? We will extend the FOCUS model to evaluate whether effortful control has unique relations with the trajectory of each individual disorder, above and beyond its associations with the common externalizing trajectory. Fourth, do effortful control and externalizing symptoms have a shared developmental trajectory? We will test this question by: (a) examining the extent to which effortful control loads onto a common developmental trajectory in the FOCUS model, along with ADHD, ODD, and CD symptoms, and (b) conducting a confirmatory factor analysis of the slopes of effortful control and ADHD, ODD, and CD symptoms to see whether a one- or two-factor structure provides the best representation of the data. Last, we will examine whether parent ASPD serves as a risk factor for *both* effortful control *and* ADHD, ODD, and CD symptoms by entering parent ASPD symptoms as a predictor of the latent growth curve trajectories of effortful control, ADHD, ODD, and CD.

The current study extends previous research in several substantive and methodological ways. First, we used data from a large community sample rather than a clinical sample, which helps us to understand how individuals deviate from normative development (aligned with a developmental psychopathology approach) and to generalize our findings to the broader population. Second, we conducted multiple assessments of both effortful control and externalizing symptoms from late childhood (age 10) through adolescence (age 16), allowing us to draw inferences about co-developmental patterns during this period. Third, we utilized multi-method data by assessing effortful control via child self-reports and mother-reports of the child, in addition to assessing externalizing symptoms via a well-validated psychiatric interview. Fourth, we evaluated each of our research questions separately for the facets of effortful control (i.e., inhibitory control, activation control, and attention) and the facets of ADHD (i.e., inattention and hyperactivity) and ODD (i.e., defiance and emotion dysregulation). Fifth, we used a novel statistical technique to investigate whether ADHD, ODD, and CD symptoms share a common externalizing trajectory across the course of adolescence, as well as whether effortful control shares this common developmental trajectory. Sixth, we included an etiological factor (i.e., parent ASPD symptoms) that could plausibly influence the development of effortful control, ADHD, ODD, and CD symptoms. Last, we examined these research questions in an understudied ethnic minority group, Mexican-origin youth, which addresses recent calls to conduct research on a more diverse range of ethnic and racial groups.

Method

Participants and Procedures

We used data from the California Families Project, a longitudinal study of Mexican-origin youth and their parents ($N = 674$).[1] Children were drawn at random from rosters of students from the Sacramento and Woodland, CA, school districts. The focal child had to be in the 5th

[1]Six papers from the California Families Project have examined effortful control (Atherton, Conger, Ferrer, & Robins, 2016; Atherton, Tackett, Ferrer, & Robins, 2017; Atherton, Zheng, Bleidorn, & Robins, in press; Clark, Donnellan, Conger, & Robins, 2015; Robins, Donnellan, Widaman, & Conger, 2010; Taylor, Widaman, & Robins, in press) and five papers have examined ADHD, ODD, and/or

grade, of Mexican origin, and living with his or her biological mother, in order to participate in the study. Approximately 72.6% of the eligible families agreed to participate in the study, which was granted approval by the University of California, Davis Institutional Review Board (Protocol # 217484–21). The children (50% female) were assessed annually from 5th (M_{age} at Wave 1=10.86, SD=0.51) through 11th grade (M_{age} at Wave 7=16.79, SD = 0.50). Data collection occurred from 2006 to 2013 for the waves in the present study. Of the original 674 families, 85%, 86%, 89%, 91%, 89%, and 90% were retained at Waves 2 through 7, respectively. To investigate the potential impact of attrition, we compared individuals who did and did not participate in the age 16 assessment on study variables assessed at age 10. No significant differences were found in gender, effortful control, or ADHD, ODD, and CD symptoms, all ps > .10.

Participants were interviewed in their homes in Spanish or English, depending on their preference. Interviewers were all bilingual and most were of Mexican heritage. Sixty-three percent of mothers and 65% of fathers had less than a high school education (median = 9th grade for both mothers and fathers); median total household income was between $30,000 and $35,000 at Wave 1 (overall range of income = < $5,000 to > $95,000). With regard to generational status, 83.6% of mothers and 89.4% of fathers were 1st generation, and 16.4% of mothers and 10.6% of fathers were either 2nd or 3rd generation. At Wave 1, 124 of the families were single-parent households (mothers only), and 549 of the families were two-parent households.

Measures

Effortful control.—Children and their mothers completed the Effortful Control scale from the short form of the *Early Adolescent Temperament Questionnaire—Revised* when the child was 10, 12, 14, and 16 years old (*EATQ-R*; Ellis & Rothbart, 2001). The EATQ-R Effortful Control scale assesses various aspects of self-control including the capacity to anticipate and suppress inappropriate responses; the capacity to focus attention and shift attention when desired; and the capacity to perform an action when there is a strong tendency to avoid it. This 16-item scale includes items such as, *"When someone tells [you/your child] to stop doing something, it is easy for [you/your child] to stop."* and *"[You/your child] pay close attention when someone tells [you/your child] how to do something."* Ratings were made on a 4-point scale ranging from 1 (*not at all true of you/your child*) to 4 (*very true of you/your child*). Child- and mother-reports of effortful control correlated between .40 and .45 across ages. Therefore, we computed a latent factor of 'effortful control' using four indicators, which were computed by creating parcels of randomly selected items and then averaging across child- and mother-reports of those items. The

CD symptoms (Aizpitarte, Atherton, & Robins, 2017; Atherton, Ferrer, & Robins, in press; Atherton, Schofield, Sitka, Conger, & Robins, 2016; Ferrer, Conger, & Robins, 2016; Schofield et al., 2012), but none have examined the concurrent or longitudinal associations between effortful control and ADHD, ODD, or CD symptoms. In Atherton, Zheng, Bleidorn, & Robins (in press), the trajectory of effortful control from age 10 to 16 was reported as a means for examining its co-development with school behavioral problems. In Atherton, Ferrer, & Robins (in press), the longitudinal trajectories of ADHD, ODD, and CD symptoms were reported as a substantive part of the paper. However, in the current paper, we focus on the co-development of effortful control and ADHD, ODD, and CD symptoms. We do not report the effortful control, ADHD, ODD, and CD trajectories as a substantive focus of the present paper, and we note in the Introduction and Discussion that these trajectories have been reported elsewhere. For a full list of California Families Project publications, see: https://osf.io/rn34p/.

loadings of the indicators ranged from .71 to .80 across waves. The omega reliabilities (ω) of the latent factors ranged from .75 to .87.

In addition to the broad 'effortful control' scale, we also computed three facet scales: 'activation control' (the capacity to perform an action when there is a strong tendency to avoid it), 'attention' (the capacity to focus attention as well as to shift attention when desired), and 'inhibitory control' (the capacity to plan and to suppress inappropriate responses). Each facet scale had four indicators, based on parcels of randomly selected items within rater and then averaging the same-item parcels across raters. The omega reliabilities (ω) of the latent factors ranged from .60 to .66 for activation control, .56 to .61 for attention, and .40 and .52 for inhibitory control across assessments.

Externalizing symptoms - ADHD, ODD and CD.—The NIMH Diagnostic Interview Schedule for Children-IV (DISC-IV) was administered to the child at every assessment from age 10 to 16 (i.e., seven waves of data). The DISC-IV is a comprehensive, psychiatric interview that assesses mental health problems for children and adolescents using DSM-IV and ICD-10 criteria; it is the most widely-used mental health interview that has been tested in both clinical and community populations and validated in both English and Spanish (Costello, Edelbrock, & Costello, 1985; Schwab-Stone et al., 1996; Shaffer et al., 2000; translated into Spanish by Bravo, Woodbury-Farina, Canino, & Rubio-Stipec, 1993). For the present study, we utilized the Attention-Deficit Hyperactivity Disorder (ADHD; 23 items), Oppositional Defiant Disorder (ODD; 12 items), and Conduct Disorder (CD; 23 items) modules of the NIMH DISC-IV. Responses were recorded dichotomously (0=no, 1=yes) as the symptom being present or not *in the past year*. The ADHD module included questions about attention-related behaviors such as, *"[Did you have] trouble keeping your mind on task for more than a short period of time?*" and hyperactivity problems such as *"[Did you] often climb on things/run around when you weren't supposed to?*" The ODD module included questions about irritability such as, "*[Did you] lose your temper?*" and defiant behaviors such as *"[Did you] do things on purpose that caretakers said not to do?*" The CD module included questions about more severe delinquent behavior such as "*[Did you] break/damage someone else's things on purpose?*" and "*[Did you] bully someone smaller who wouldn't fight back*?" For each externalizing disorder, we computed a symptom count variable by summing the responses at each wave to create composite scores of ADHD, ODD, and CD symptoms from age 10 to 16 (seven waves total). In addition, we computed symptom counts for the inattention (11 items) and hyperactivity (12 items) facets of ADHD, and for the emotion dysregulation (4 items) and defiant (8 items) facets of ODD.

Parent Antisocial Personality Disorder (ASPD) symptoms.—At Wave 1, mothers (*N*=637) and fathers (*N*=402) completed the 47-item Antisocial Personality Disorder (ASPD) module of the CIDI (Kessler & Ustun, 2004). These items asked about their past (since age 15) and current antisocial behavioral problems, and included questions such as, "*Have you sometimes made money illegally, perhaps by selling things you knew were stolen, selling drugs, prostitution, providing false IDs, or any other way?*", "*Have you sometimes used a stick, knife, gun, bottle, or bat to hurt someone?*", and "*Have you often driven when you were high or drowsy on alcohol or drugs?*" Responses were recorded

dichotomously (0 = no, 1 = yes) as the symptom being present or not. We summed the number of endorsed symptoms separately for: (1) mother ASPD symptoms (M=1.36, SD=2.65, range=0–18), and (2) father ASPD symptoms (M=2.91, SD=3.96, range=0–22). Mother and father ASPD symptoms were significantly and positively correlated (r = .44, p < .001).

Statistical Analyses

All analyses were conducted using M*plus* Version 7 (Muthén & Muthén, 1998–2011). We used a robust maximum likelihood estimator (MLR) to account for non-normal distributions of observed variables and full information maximum likelihood procedure (FIML) to account for missing data (Allison, 2003; Schafer & Graham, 2002). For effortful control, we used item parcels as indicators because they produce more reliable latent variables than individual items (Little, Cunningham, Shahar & Widaman, 2002). To compute more accurate fit indices for model comparisons with large samples, we assessed adequate model fit by changes in chi-square and degrees of freedom, comparative fit index (ΔCFI) less than or equal to .01, and McDonald's non-centrality index (ΔNCI) less than or equal to .02 (Cheung & Rensvold, 2002; Meade, Johnson, & Braddy, 2008). We also note values of the root-mean-square error of approximation (RMSEA), for which adequate fit is indicated by values less than or equal to .06 (Hu & Bentler, 1998; Hu & Bentler, 1999).

To evaluate measurement invariance over time for effortful control, we compared three measurement models: (1) freely estimating the factor loadings for the latent factors at each age of assessment (i.e., configural invariance); (2) constraining the respective factor loadings to be equal at each age of assessment (i.e., weak invariance); and (3) constraining the factor loadings and intercepts to be equal at each age of assessment (i.e., strong invariance). If the more constrained models do not fit worse than the lesser constrained models, then we can conclude that the structure of the latent constructs is the same over time. Effortful control (and the activation control and inhibitory control facets) were partially strong invariant over time; the attention control facet was strong invariant (see Table S1 in the Supplemental Material for model comparisons).

We used univariate latent growth curve (LGC) models to examine the growth of effortful control, ADHD, ODD, and CD (and related facets) over time. LGC models describe the average initial level (intercept) and growth over time (slope) of a construct, as well as how much variability there is in the intercept and slope. To find the best-fitting growth trajectory for each disorder, we conducted a series of model comparisons and evaluated changes in chi-square, degrees of freedom, root mean-square-error of approximation (RMSEA), Comparative Fit Index (CFI), and the Tucker-Lewis Index (TLI). Specifically, we compared three models: (1) *no growth model*, where the slope is fixed to be zero over time; (2) *linear growth model*, where the slope linearly increases by one unit over time, with the first time point centered at '0', the second time point fixed at '1' (or '2' for the biennial assessments of effortful control),…, and the last time point is fixed at '6'; and (3) *a latent basis model*, where the first and last time points of the slope are fixed (at '0' and '6', respectively) and the middle time points are freely estimated to the data. In all models, path coefficients from the intercept to the repeated assessments are fixed to 1, and the intercept and slope are allowed

to covary. Tables S2–S4 show the univariate LGC model comparisons for effortful control (and facets) and ADHD, ODD, and CD symptoms (and facets). To examine the co-development of effortful control and externalizing symptoms, we conducted several *bivariate* LGC models and specified correlations among the levels and slopes of effortful control and each form of externalizing symptoms (i.e., ADHD, ODD, and CD).

To examine whether ADHD, ODD, and CD symptoms share a common externalizing trajectory, we used the FOCUS model (Isordia, Conger, Robins, & Ferrer, 2017; McArdle, 1988), which evaluates the extent to which a higher-order common factor accounts for the interrelations among lower-order developmental processes. The first level of a FOCUS model consists of individual univariate LGCs that characterize the independent developmental trajectory of each construct. At the second level of a FOCUS model, second-order common intercept and slope factors are specified to assess whether a higher-order factor structure drives the relation among the developmental trajectories of the lower-order constructs. The second-order intercept represents the shared lower-order functions at the initial time point, and the second-order *slope* represents the shared growth trajectory of the constructs over time (i.e., they are changing over time in a similar way) (see Figure 1 for a visual depiction of the FOCUS model). Therefore, the FOCUS model captures what is shared among different lower-order developmental processes over time. To run this model, we fixed the covariances among the latent intercepts and slopes to 0, and specified these associations as part of two higher-order common factors: the common externalizing problem intercept (f0) and the common externalizing problem slope (fs). To scale the common externalizing problem factors, we specified ADHD symptoms to serve as the reference variable (Bollen & Curran, 2006). That is, we fixed the loadings connecting the latent intercept and slope factors of ADHD symptoms to the common externalizing problem factors to 1 and estimated the remaining factor loadings for ODD and CD symptoms (Duncan, Duncan, & Stryker, 2006). To identify the model, we fixed the common externalizing intercept and slope factors to zero and estimated the lower-order means of the intercepts and slopes. Thus, the resulting parameter estimates in this model include a common externalizing intercept variance ($\sigma f0^2$) and common externalizing slope variance (σfs^2); the covariance between the common externalizing intercept and slope factors ($\sigma f0,fs$); and the loadings (λ) of the ADHD, ODD, and CD intercepts on the common externalizing intercept factor and the loadings (λ) of the ADHD, ODD and CD slopes on the common externalizing slope factor.

To understand whether effortful control is related to the common externalizing trajectory (above and beyond the individual disorders themselves), we extended the FOCUS model by including correlations with the univariate LGC trajectory of effortful control (see Figure 1). Specifically, we conducted a series of analyses where we specified correlations between effortful control (intercept, slope) and the higher-order externalizing intercept and slope factors, as well as correlations between effortful control (intercept, slope) and one of the disorders lower-order intercept and slope, simultaneously. These analyses help us to disentangle whether effortful control co-develops with the higher-order externalizing intercept and slope factors (shared by the three disorders), and/or if effortful control has unique associations with each individual disorder, above and beyond the common externalizing trajectory.

To investigate whether effortful control shares a common developmental trajectory with ADHD, ODD, and CD symptoms, we conducted two sets of analyses. First, we re-ran the FOCUS model with the effortful control trajectory as an indicator, along with the trajectories of ADHD, ODD, and CD symptoms. Second, we saved the factor slopes (from age 10 to 16) from the univariate latent growth curve models for the effortful control facets (inhibitory control, activation control, attention control), ADHD facets (inattention, hyperactivity), ODD facets (emotion dysregulation, defiance), and total CD symptoms. Then, we conducted confirmatory factor analyses, and model comparisons using chi-square difference tests, of the factor slopes to evaluate whether a one-factor structure fit significantly worse than a two-factor structure.

Last, we examined whether mother/father ASPD symptoms influenced the initial level and growth of effortful control and ADHD, ODD and CD symptoms by entering each as a time-invariant covariate into the univariate LGC models; specifically, the level and slope of each trajectory is regressed on mother (or father) ASPD symptoms.

Results

Table 1 shows descriptive statistics for effortful control and ADHD, ODD, and CD symptoms. In terms of the concurrent correlations, effortful control was significantly negatively associated with ADHD (range = −.23 to −.29 across ages 10, 12, 14, and 16), ODD (range = −.20 to −.24), and CD (range = −.13 to −.22) symptoms, all *p*s < .05.[2]

Does Effortful Control Co-Develop with ADHD, ODD, and CD Symptoms?

Table 2 shows results from the bivariate LGC models. The initial *levels* of effortful control and ADHD, ODD, and CD symptoms were all significantly negatively correlated (see Figure 2). That is, youth who had poorer effortful control at age 10 tended to exhibit more ADHD, ODD, and CD symptoms at age 10. These associations held for all three facets of effortful control (i.e., activation control, inhibitory control, attention control) as well as both facets of ADHD (i.e., inattention, hyperactivity) and both facets of ODD (i.e., emotion dysregulation, defiance).

We also found significant correlations among the *slopes* (see Figure 3). Specifically, greater decreases in effortful control were related to greater increases in ADHD, ODD, and CD symptoms over time. These associations held for all of the facets of effortful control with ADHD, ODD, and CD symptoms (except for attention control with ODD symptoms), and for the facets of ADHD and ODD symptoms with effortful control (except for effortful control with hyperactivity). Thus, individuals who increased (decreased) in effortful control (and its facets) from age 10 to 16 tended to increase (decrease) in externalizing problems (and its facets) across the same period.

[2]Note that none of the Effortful Control items overlap with any of the ODD and CD symptoms, or the ADHD-hyperactivity symptoms. However, three items on the attention control subscale were similar to ADHD-inattention symptoms. Despite the three overlapping items, the magnitude of the concurrent correlations between attention control and ADHD-inattention (rs = −.35 to −.42 across waves), was not that much higher than the correlation of ADHD-inattention with activation control (*r*s = .25 to .33) or inhibitory control (*r*s = .18 to .31). Therefore, it seems unlikely that the minimal content overlap is inflating the observed associations between effortful control and ADHD, especially given that ADHD-inattention is also associated with the two facets of effortful control that do not have overlapping content.

Finally, we examined the correlation between initial levels of effortful control at age 10 and change over time in ADHD, ODD, and CD symptoms (and vice versa). Lower levels of effortful control at age 10 were related to greater *decreases* in ADHD and ODD symptoms (but unrelated to change in CD symptoms) from age 10 to 16. As Figure 4 shows, these seemingly counterintuitive associations reflect the fact that youth who are higher on effortful control at age 10 typically have low and stable trajectories of ADHD and ODD, whereas youth who are lower on effortful control at age 10 show greater decreases in ADHD and ODD over time, but their predicted trajectories are still higher overall. Additionally, it is possible that youth who have lower effortful control at age 10 simply have more "room to mature" when compared to their peers who have higher effortful control at age 10, and consequently show greater decreases in ADHD and ODD symptoms over time.

Conversely, when we examined whether initial levels of externalizing symptoms predict the slope of effortful control, we found that youth who are higher on CD symptoms at age 10 typically have low and stable trajectories of effortful control, whereas youth who are lower on CD symptoms at age 10 typically show greater decreases in effortful control, even though their effortful control trajectory is still much higher overall (see Figure 5). ADHD and ODD symptoms at age 10 were not significantly associated with the slope of effortful control.[3]

Do ADHD, ODD, and CD Symptoms Share a Common Externalizing Trajectory?

The factor of curves (FOCUS) model for ADHD, ODD, and CD trajectories had adequate fit (CFI = .87, TLI = .86, RMSEA = .07), indicating that the specification of a higher-order developmental factor structure (i.e., "externalizing symptoms") was acceptable for representing common growth in the three constructs. There was significant variance in the externalizing symptoms intercept ($\sigma f0^2 = 1.71$, $p < .001$) and slope ($\sigma fs^2 = .04$, $p < .001$), which indicates that there were individual differences in externalizing symptoms at the initial level and in change over time. The standardized factor loadings of the externalizing symptom intercept factor were moderate-to-high and significant for ADHD (λ=.86), ODD (λ=.92), and CD symptoms (λ=.69), all *p*s < .001. These results indicate that all three lower-order intercepts contributed significantly to a common externalizing intercept factor. The standardized factor loadings of the externalizing symptom slope factor were .99 for ADHD, .80 for ODD, and .25 for CD symptoms, all *p*s < .001. These findings indicate that while ADHD, ODD, and CD symptoms all contributed significantly to a common source of change over time in externalizing problems, there were substantial differences in the magnitude of their contribution. ADHD and ODD showed very high loadings onto the common externalizing symptoms slope, whereas CD symptoms contributed much less to this common source of externalizing change.

We conducted follow-up analyses using the facets of ADHD (inattention, hyperactivity) and ODD (emotion dysregulation, defiance) in combination with CD symptoms in the FOCUS model, to get a better idea of which lower-level aspects are the driving forces behind the

[3]We also conducted follow-up analyses using parent ASPD symptoms as control variable in the bivariate latent growth curve models. Table S5 in the supplemental material shows the results. Overall, many of the findings remain the same in terms of magnitude and statistical significance when controlling for parent ASPD symptoms (with a few exceptions with father ASPD symptoms), suggesting that that the co-developmental associations between effortful control and externalizing problems are not entirely accounted for by this confounding variable.

common externalizing trajectory. The model had adequate fit (CFI = .90, TLI = .89, RMSEA = .05). The standardized factor loadings of the externalizing symptom intercept factor were high and significant for ADHD-inattention (λ=.90), ADHD-hyperactivity (λ=.83), ODD-emotion dysregulation (λ=.83), ODD-defiance (λ=.90), and CD symptoms (λ=.69), all *p*s < .001. The standardized factor loadings of the externalizing symptom slope factor were also high and significant for inattention (λ=.70), hyperactivity (λ=.79), emotion dysregulation (λ=.84), defiance (λ=.93), and CD symptoms (λ=.61), all *p*s < .001. Thus, it seems as though the defiance component of ODD accounts for the largest portion of the shared externalizing trajectory.

Are the Associations between Effortful Control and ADHD, ODD, and CD Trajectories Accounted for by a Common Externalizing Trajectory?

We conducted a series of analyses examining the strength of the associations between the effortful control trajectory and the higher-order externalizing trajectory, and between the effortful control trajectory and each of the individual disorder trajectories. Table 3 shows the results of these extended FOCUS model analyses.[4] In general, the associations between effortful control and ADHD, ODD, and CD symptoms seem to be subsumed by a general externalizing factor common to the three disorders, as evidenced by the strong and significant correlations between the effortful control level/slope and the higher-order externalizing symptom level/slope. Above and beyond this common externalizing factor, there were two significant correlations between effortful control and the lower-order disorders. After accounting for the associations with the externalizing symptoms common intercept factors, effortful control at age 10 was significantly positively related to ODD symptoms at age 10 and significantly negatively related to CD symptoms at age 10. Comparatively, although both statistically significant, the correlation between effortful control and CD symptoms was much smaller in magnitude than the correlation between effortful control and ODD symptoms at age 10. Taken together, although there are unique *concurrent* associations between effortful control and the individual disorders (ADHD, ODD, and CD), there are few unique *longitudinal* associations between effortful control and the individual disorders, above and beyond their shared externalizing developmental trajectory. Thus, the shared externalizing trajectory may better capture the core of externalizing problems and how youth develop during adolescence, especially given that ADHD, ODD, and CD symptoms rarely occur in isolation from one another.

Do Effortful Control and Externalizing Symptoms Have a Shared Developmental Trajectory?

We re-ran the FOCUS model and included the effortful control trajectory as an indicator of the general developmental trajectory, along with the ADHD, ODD, and CD symptom trajectories. The FOCUS model had adequate fit (CFI = .88, TLI = .88, RMSEA = .06), indicating that the specification of a higher-order developmental factor structure was reasonable for representing change in the four constructs. The standardized loadings on the general factor level (in descending order) were .90 for ADHD symptoms, .87 for ODD

[4]We also conducted the same extended FOCUS analyses, but with the facets of ADHD/ODD and CD. See Table S6 (supplemental material) for the results.

symptoms, .72 for CD symptoms, and .54 for effortful control (all *p*s < .001). The standardized loadings on the general factor slope (in descending order) were .99 for ADHD symptoms, .80 for ODD symptoms, .35 for effortful control, and .26 for CD symptoms (all *p*s < .001). Although the uniformly positive loadings might suggest the presence of a general factor (albeit a weak one given how small the effortful control and CD loadings are), it is not uncommon to find such a pattern even when the underlying structure is two dimensional.

To further evaluate the structure of the effortful control and externalizing trajectories, we conducted confirmatory factor analyses of the saved trajectories (i.e., slopes) estimated from the univariate growth curve models. Specifically, we compared the fit of a one-factor model, in which the trajectories of the effortful control facets (inhibitory control, activation control, attention control), ADHD facets (hyperactivity, inattention), ODD facets (emotion dysregulation, defiance), and total CD symptoms all loaded on the same factor, to the fit of two-factor model, in which the effortful control facets loaded onto one factor and the externalizing measures loaded onto another. A chi-square difference test showed that the one-factor solution fit the data significantly worse than the two-factor solution ($\Delta\chi^2$ = 174.14, $\Delta df = 1$, $p < .001$). As shown in Table 4, the two-factor solution provided a clean structure, with relatively strong loadings on the primary factor and uniformly small cross-loadings. Thus, these analyses do not support the idea that effortful control and externalizing problems necessarily have a shared developmental trajectory.[5]

Do Parent ASPD Symptoms Serve as a Shared Risk Factor for Effortful Control and ADHD, ODD, and CD Symptoms?

Table 5 shows results from models that include mother and father ASPD symptoms as predictors of effortful control, ADHD, ODD, and CD trajectories. Mother ASPD symptoms were significantly associated with initial levels of effortful control, ADHD, ODD, and CD symptoms. Specifically, mothers who had more ASPD symptoms tended to have children who were lower in effortful control and higher in ADHD, ODD, and CD symptoms at age 10. These concurrent associations all replicated with fathers' ASPD symptoms, except the association with child's CD symptoms at age 10 was not significant. Both mother and father ASPD symptoms predicted change over time in their child's CD symptoms, in that parents who had more ASPD symptoms tended to have children who showed greater *increases* in CD symptoms from age 10 to 16. In addition, father ASPD symptoms at age 10 predicted change over time in effortful control from age 10 to 16; however, this effect did not replicate for mother ASPD symptoms.

Discussion

Increasing attention has been given to research that lies at the intersection of personality and psychopathology development, in part because researchers have found that personality and psychopathology are less distinct than was traditionally believed (Durbin & Hicks, 2014).

[5]We also conducted model comparisons of a one- and two-factor structure for the saved *levels* (estimated at age 10 from univariate growth curve models). A chi-square difference test indicated that the one-factor solution fit the data significantly worse than the two-factor solution for the levels ($\Delta\chi 2 = 694.64$, $\Delta df = 1$, $p < .001$), with the effortful control levels loading onto one factor and the externalizing levels loading onto the other. The pattern of factor loadings was similar to the pattern shown in Table 4 for the slopes.

There has been a strong push for researchers to examine the associations between cognitive control systems (like effortful control) and various externalizing pathology (ADHD, ODD, and CD) across development (NIMH, 2018). The present study aimed to fill this gap by examining the co-development of effortful control and ADHD, ODD, and CD symptoms using multi-method data from a longitudinal study of 674 Mexican-origin youth assessed annually from age 10 to 16. In general, results showed that effortful control and ADHD, ODD, and CD symptoms co-develop from late childhood through adolescence. Specifically, youth who experienced greater *decreases* in effortful control were more likely to have greater *increases* in ADHD, ODD, and CD symptoms over time. We also found that ADHD, ODD, and CD symptoms shared a common externalizing trajectory, with ADHD and ODD contributing more to this trajectory than CD. Further, the co-developmental effects between effortful control and externalizing problems are better accounted for by the common externalizing trajectory, with few unique associations with each individual disorder. Relatedly, when we extended the FOCUS model to include the effortful control trajectory as an indicator, we found that ADHD and ODD symptoms contributed more to a shared developmental trajectory than did effortful control and CD symptoms. Follow-up analyses at the facet level provided support for a two-factor solution, with the effortful control facet trajectories loading on one factor and the externalizing facet trajectories loading on another factor. Last, parents with more ASPD symptoms had children with lower effortful control and higher ADHD, ODD, and CD symptoms at age 10. Mother and father ASPD symptoms also predicted greater *increases* in CD symptoms from age 10 to 16. Below we discuss the implications of our findings for research on adolescent personality and psychopathology development in more detail.

Effortful Control and ADHD, ODD, and CD Symptoms Co-Develop in Adolescence

Aligned with previous research that suggests there are decreases in self-regulatory traits during adolescence (Borghuis et al., 2017; De Fruyt et al., 2006; Klimstra et al., 2009; Soto et al., 2011; Soto & Tackett, 2015; Van den akker, Dekovic, Asscher, & Prinzie, 2014) and as shown in previous work with the *California Families Project* data (Atherton, Zheng, Bleidorn, & Robins, in press), on average, effortful control declines from age 10 to 16. However, there was significant individual variability in the developmental trajectories of effortful control, with some youth decreasing rapidly and others actually showing increases across the study period. Nonetheless, on average, it seems as though adolescence may be a period of disruption, where youth experience slight declines in their regulatory capacities. Moreover, as already shown in previous work with this sample (Atherton, Ferrer, & Robins, in press), ADHD symptoms decrease over the course of adolescence; ODD symptoms slightly increase before declining in late adolescence; and, CD symptoms increase from late childhood through adolescence. These distinct developmental trajectories of externalizing symptomatology suggest that not all forms of problem behaviors may change in the same way during adolescence.

Previous longitudinal research conducted in childhood has found prospective associations between effortful control and externalizing problems (Choe, Olson, & Sameroff, 2013; Eisenberg et al., 2004; Eisenberg et al., 2005; Lavigne, Gouze, Hopkins, & Bryant, 2016; Lengua, 2006; Olson, Choe, & Sameroff, 2016; Valiente et al., 2006; Zalewski et al., 2011).

Moreover, other research has demonstrated a longitudinal prediction from disinhibition/conscientiousness to later externalizing problems in adolescence and adulthood (Caspi, Moffitt, Newman, & Silva, 1996; Henry, Caspi, Moffitt, & Silva, 1996; Krueger, 1999; van der Akker, Dekovic, & Prinzie, 2010). However, little-to-no research has investigated *correlated change* in adolescence, specifically. We found that youth who experienced greater *decreases* in effortful control were more likely to have greater *increases* in ADHD, ODD, and CD symptoms from age 10 to 16. Aligned with a developmental psychopathology perspective (Cicchetti & Rogosch, 1999; Durbin & Hicks, 2014), these findings provide some initial evidence that deviations from normative developmental trajectories may create a vicious cycle between effortful control and externalizing problems. Furthermore, these deviations from normative development may serve to reinforce personality and psychopathological tendencies even further, thus leading youth to be at higher risk for experiencing the long-term consequences associated with poor self-control and externalizing problems. The longitudinal associations between effortful control and externalizing symptoms provide one avenue for how individuals may be propelled into trajectories of poor self-regulation, more generally.

Further, we examined the robustness of these co-developmental effects by including mother/father ASPD symptoms as controls in the bivariate latent growth curve models. In general, almost all of the findings remain the same in terms of magnitude and statistical significance when controlling for parent ASPD symptoms, which suggests that the co-developmental associations between effortful control and externalizing problems are not entirely accounted for by this plausible third variable that could give rise to the development of both. There were several results that became marginally- or non-significant when controlling for father ASPD symptoms; however, it is worth noting that we had a smaller subsample of participants with father data (N=402–476), which reduces the statistical power we had to detect effects with these complex longitudinal models.

Taken together, our findings show that *greater decreases* in broad temperament dimensions like effortful control are related to *greater increases* in externalizing symptoms. Knowing that deviations from normative effortful control development are related to deviations in ADHD, ODD, and CD symptom development is only the beginning of our understanding of the dimensionality of basic systems and psychopathology. However, now that we have some evidence to suggest that broad temperament dimensions like effortful control are longitudinally associated with externalizing symptoms, researchers can begin to look to smaller units of analysis (like genes and physiology) to better understand which “control systems” are disrupted under the skin. Although at the broad trait level we see that effortful control co-develops with all forms of externalizing symptomatology (ADHD, ODD, CD), investigating different units of analysis may shed light on the nuances to different forms of externalizing pathology. For example, ADHD was recently re-classified as a neurodevelopmental disorder, instead of a disruptive behavior disorder (Beauchaine & McNulty, 2013). Despite this, findings from the FOCUS model in the present study suggest that ADHD follows a common *externalizing* trajectory, along with ODD and CD symptoms. Thus, it may be critical to understand whether there are distinct abnormalities in neural cognitive control systems that set apart the development of ADHD symptoms from ODD and CD symptoms. Digging into smaller units of analysis may shed some light on whether

cognitive control systems (related to effortful control) have distinct associations with different forms of externalizing problems or not.

Relatedly, it may also be critical to consider abnormalities in multiple systems in conjunction with one another, especially with respect to externalizing development in adolescence. For example, recent findings suggest that effortful control (and the engagement in externalizing behavior, more broadly) may be best defined as a dual-systems model, where the strength of the impulse (reward-seeking) can be separated from the ability to control the impulse and avoid exhibiting the relevant behavior (regulation). Although there may be some adaptive consequences of increases in reward sensitivity in the brain during adolescence (Telzer, 2016), adolescents may be at a relatively high risk for engaging in problem behaviors because the brain regions associated with reward-seeking develop earlier than brain regions associated with impulse control (Casey & Caudle, 2013; Shulman, Harden, Chein, & Steinberg, 2014), which creates an imbalance between the strength of the impulse and the capacity to control it. These intriguing findings also suggest that brain development may be related to concomitant changes in temperament/personality development, given that personality trajectories tend to map onto similar imbalances (e.g., decreases in effortful control/conscientiousness traits and increases in sensation seeking traits during adolescence; Shulman, Harden, Chein, & Steinberg, 2014). Thus, understanding how other traits, like sensation seeking, interact with effortful control to produce developmental changes in externalizing problems would be a critical next step. Further, these interactions between temperamental traits could be extended to the smaller units of analysis, in that, it is possible that youth who develop externalizing pathology may have abnormalities (or an imbalance) in both cognitive control and positive valence systems.

A Shared “Externalizing” Trajectory in Adolescence and Relations with Effortful Control

Given the high comorbidity among ADHD, ODD, and CD symptoms, previous research has contested the utility of examining externalizing disorders, like ADHD, ODD, and CD, separately from one another (Beauchaine & McNulty, 2013; Widiger & Smith, in press). Instead, researchers have suggested that problem behaviors are better represented by an overall externalizing factor that subsumes ADHD, ODD, and CD symptoms (Achenbach & Edelbrock, 1984; Beauchaine & McNulty, 2013; Caspi et al., 2014; Kotov et al., 2017; Tackett, 2010). Prior work has often used statistical models such as bivariate and multivariate comorbidity models to show the superiority of using a general externalizing factor (Krueger & Markon, 2006). However, examining the factor structure of externalizing symptoms is a different question than the extent to which externalizing symptoms have a shared developmental trajectory. Given that ADHD, ODD, and CD are components of a broader externalizing construct and often predict change in each other (Atherton, Ferrer, & Robins, in press; Krueger & Markon, 2006), it is possible that there is an underlying “externalizing core” that leads ADHD, ODD, and CD symptoms to develop through a shared developmental process. The present study is the first to examine whether ADHD, ODD, and CD symptoms have a shared externalizing trajectory by utilizing a relatively novel statistical technique, the FOCUS model (Isordia, Conger, Robins, & Ferrer, 2017).

Replicating prior work, we found that the *levels* (estimated at age 10) of externalizing symptoms form a common externalizing factor, with moderate-to-high loadings for ADHD, ODD, and CD symptoms. As an extension of previous work, we also found that ADHD, ODD, and CD symptoms all contributed significantly to a common source of change over time in "externalizing" problems, but there were differences in the magnitude of their contribution. ADHD and ODD showed very high loadings onto the common externalizing symptoms slope, whereas CD symptoms contributed much less to this common externalizing symptoms slope. Although this may seem surprising given that ODD and CD symptoms tend to be more highly comorbid than ADHD and ODD symptoms (Beauchaine & McNulty, 2013; but see Harvey, Breaux, & Lugo-Candelas, 2016), it is less surprising when examined through a developmental lens. Specifically, ADHD and ODD both typically have their onset in childhood and both tend to decline across adolescence, whereas CD symptoms (e.g., stealing, destroying public property, and getting into fights) emerge later in adolescence. Consequently, during the developmental period examined, ADHD and ODD symptoms were more commonly experienced than CD symptoms, which may also have contributed to the lower factor loading for CD. If we were to look later in development, beyond age 16, CD symptoms might contribute more to a shared externalizing trajectory by virtue of becoming more prevalent.

Follow-up analyses indicated that defiance against authority figures (e.g., parents, teachers) was the primary driving force behind this shared externalizing trajectory. Given the nature of ADHD, ODD, and CD symptoms, it is possible that defiance is such a strong indicator of this shared developmental process because acting out against the external world necessarily leads individuals to defy social norms and standards. For example, the hyperactive adolescent who cannot sit still in the classroom (indicative of ADHD) is often defying the rules and norms of how students should act in school when the teacher is trying to lecture. Similarly, youth who often argue with parents and/or teachers are typically defying the authority and rules put forth by these figures (indicative of ODD); and, adolescents who vandalize, steal, and set fires (indicative of CD) are often defying societal norms and rules for what is considered acceptable behavior. Thus, these findings suggest that ADHD and ODD symptoms (and CD to a lesser extent) share an underlying externalizing trajectory across the course of adolescence, and that defiance may be a core feature of this shared developmental process. Identifying the presence and features of a shared externalizing trajectory can aid in better understanding why some youth are set on trajectories of engaging in more problem behaviors, whereas others desist. Adhering to societal and social norms, rules, and standards may be one particularly important component that can lead youth to either thrive or falter in this domain.

As another extension of previous work, we examined whether the co-development of effortful control with each of the individual ADHD, ODD, and CD trajectories was largely accounted for by associations between effortful control and a common externalizing trajectory. In general, we found strong associations between effortful control and the shared externalizing trajectory, such that greater *decreases* in effortful control were related to greater *increases* in externalizing problems over time. Above and beyond this common externalizing factor, there were only two significant correlations between effortful control and the individual disorders and both were concurrent associations. Therefore, these results

suggest that effortful control has few unique *longitudinal* associations with the individual disorders (ADHD, ODD, CD), above and beyond their shared developmental trajectory. These findings provide some evidence about the extent to which temperamental traits, like effortful control, impact the specific presentation or manifestation of externalizing problems (Widiger & Smith, in press). Given the lack of unique, longitudinal associations between effortful control and the individual disorders, our understanding of externalizing pathology development may be more efficiently pursued by examining the causes and consequences of a shared externalizing trajectory, rather than investigating each of the disorders separately over time. However, further research is needed to understand whether other potential influences (besides effortful control) have unique associations with the individual externalizing trajectories, above and beyond their shared externalizing trajectory, to determine whether the present findings generalize to other etiological factors.

The Extent to Which Effortful Control and ADHD, ODD, and CD Symptoms Have a Shared Developmental Trajectory

To fully understand the relations between effortful control and externalizing symptoms, it is critical to know whether they should be treated as distinct constructs, or part of the same underlying dimension (Kotov et al., 2017; Krueger & Tackett, 2013; Widiger et al., 2018). In the present study, we aimed to replicate and extend findings in this area by evaluating the extent to which effortful control and externalizing symptoms share a common developmental trajectory, in addition to examining their associations at a single time point (Durbin & Hicks, 2014; Tackett, 2010). Consistent with previous research in support of the spectrum hypothesis (Fox et al., 2005; Iacono, Malone, & McGue, 2003; Krueger et al., 2002; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Nelson & Trainor, 2007; Vrieze, Perlman, Krueger, & Iacono, 2012; Walton et al., 2008; Young et al., 2009), when we examined effortful control and externalizing symptom *levels* estimated concurrently at age 10, we found that all variables had at least moderately high loadings on a general factor, ranging from .54 (effortful control) to .90 (ADHD). However, when we examined effortful control and externalizing symptom *slopes*, we found that ADHD and ODD symptoms loaded quite highly onto the common developmental trajectory whereas effortful control and CD symptoms had comparatively low loadings on the shared slope factor. Additionally, confirmatory factor analyses of the slopes of the facets of effortful control, ADHD, and ODD, and the overall symptom count for CD showed that a two-factor solution was a better fit to the data, where the effortful control facets load onto one factor and the facets of ADHD and ODD, and the overall CD symptom count load onto the second factor.

In summary, it seems as though the spectrum hypothesis may be a more accurate representation of the data when examining effortful control and externalizing symptoms at one point in time (at age 10), but when extended longitudinally (from age 10 to 16), the evidence is less convincing. It is, of course, possible that effortful control and externalizing symptoms lie on the same continuum, but the part of the continuum related to typical functioning (i.e., effortful control) follows a different developmental course than the part of the continuum related to atypical functioning (i.e., externalizing symptoms). For example, normative socialization processes (e.g., parental monitoring) and normative maturation of the frontal lobes may shape the developmental of healthy levels of effortful control, but

severely dysfunctional environments (e.g., abusive parenting) and neurological problems shape the development of impulse-related attentional and behavioral problems, such as ADHD, ODD, and CD. This possibility suggests that temperamental factors such as effortful control and aspects of psychopathology such as externalizing problems may be more distinct earlier in development (e.g., adolescence), and become progressively more interrelated over time. If so, this pattern would directly contradict Werner's (1957) well-known orthogenetic principle, in that "wherever development occurs it proceeds from a state of relative globality and lack of differentiation to a state of increasing differentiation, articulation, and hierarchic integration" (p. 126). It will be interesting to examine whether extending these trajectories of effortful control and externalizing symptoms into adulthood exhibits the same two-factor structure, as observed in the present study with adolescents. Additionally, future work should aim to collect complete symptom-level data and use IRT methods longitudinally to examine the factor structure of effortful control and externalizing problems over time. More generally, future research would benefit from researchers exploring both developmental and quantitative approaches to the spectrum hypothesis with other aspects of temperament and psychopathology (e.g., neuroticism and internalizing problems). Clearly, there is much room for future work in this area to expand our knowledge of the shared versus unique developmental trajectories of personality and psychopathology across the lifespan.

The Role of Parent ASPD Symptoms in the Development of Effortful Control, ADHD, ODD, and CD Symptoms

In line with a developmental psychopathology approach, we examined the influence of parent ASPD symptoms, which could potentially shape *both* one's capacity to be self-controlled and one's inclination or disinclination to engage in problem behaviors. Presumably, if effortful control and externalizing symptoms lie on the same latent continuum, then they may share some etiological risk factors, as prior work on the genetic, psychophysiological, and neurobiological correlates of personality and psychopathology suggests (Beauchaine & McNulty, 2013; Fox et al., 2005; Iacono, Malone, & McGue, 2003; Krueger et al., 2002; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Nelson & Trainor, 2007; Vrieze, Perlman, Krueger, & Iacono, 2012; Young et al., 2009). Although we were not able to look at these biological correlates of effortful control and externalizing symptoms, we examined parent ASPD symptoms, given that it may serve as both a genetic and environmental proxy for self-regulatory impairments (Krueger et al., 2002; Odgers et al., 2007; Vaughn, Salas-Wright, DeLisi, & Qian, 2015).

Similar to previous findings (Farrington et al., 2001; Frisell, Leichenstein, & Langstrom, 2011; Vaughn, Salas-Wright, DeLisi, & Qian, 2015), our results showed that mother and father ASPD symptoms were concurrently associated with effortful control and externalizing symptoms at age 10; specifically, moms and dads with higher levels of ASPD symptoms tended to have children with lower levels of effortful control and higher levels of ADHD, ODD, and CD symptoms. Moreover, extending previous research, we also found that both mother and father ASPD symptoms predicted *change over time* in the child's CD symptoms from age 10 to 16. Father ASPD symptoms also predicted *change over time* in the child's effortful control, but this effect did not replicate with mother ASPD symptoms. Given that mother and father ASPD symptoms only had replicable effects on the trajectory of CD

symptoms, it is possible that there is something unique about how parent ASPD engenders severe externalizing behaviors related to CD that does not extend to the transmission of a basic tendency to be poorly self-regulated (low effortful control), or lower-level externalizing behaviors that involve inattention/hyperactivity (ADHD) and emotion dysregulation/defiance (ODD). This finding is consistent with prior research showing that parent antisocial behavior increases risk for CD symptoms, but not ADHD or ODD symptoms (Biederman et al., 1996; Faraone et al., 2000; Loeber et al., 2000; Petty et al., 2009).

Future research should explore the mechanisms that explain *why* parent ASPD symptoms is concurrently associated with effortful control and externalizing symptoms, and why it might serve as a risk factor for increases *over time* in the child's CD symptoms. Although the continuity of conduct problems is often quite high, especially when displayed early in development (Moffitt, 1993), it is not clear whether the effects of parent ASPD symptoms in our study signify an intergenerational transmission of antisocial behavior (either genetically or environmentally), or whether parent ASPD symptoms are *currently* occurring in the household and the parents are co-offending with their child. Our parent ASPD measure reflects the extent to which the mother and father engaged in antisocial behaviors at any point since age 15, which necessarily complicates our understanding of the time course and mechanisms by which this precursor is influencing the child. Future research would benefit from utilizing more fine-grained measures of parent and child antisocial behavior over time to better understand the pathways through which parents may influence their child's effortful control and externalizing tendencies. Moreover, researchers would ideally use a longitudinal twin design to parse the genetic and environmental influences of parent ASPD symptoms on stability and change in effortful control and ADHD, ODD, and CD symptoms, as well as their co-development among youth. Utilizing this type of study design, researchers would also be able to directly examine the broader environmental influences that may co-occur with genetic risk and parent antisocial behavior such as low socioeconomic status, for example.

Limitations

There are several limitations of the present study. First, although we were able to investigate developmental and co-developmental trajectories of effortful control and externalizing symptoms from age 10 to 16, we do not have data from the youth before age 10. Because externalizing problems can emerge prior to age 10 (APA, 2013), it will be critical for future research to examine how effortful control co-develops with changes in externalizing problems from early childhood through adolescence. Second, we do not yet know which youth in our study desist from, or persist in, externalizing behaviors into young adulthood, or how these externalizing (ADHD, ODD, CD) symptoms connect with the child's ASPD symptoms. Extending these trajectories into young adulthood would illuminate which youth are following an adolescence-limited vs. life-course persistent pathway of externalizing behavior (Moffitt, 1993). Third, given our use of a community sample, there were very low rates of ADHD, ODD, and CD diagnoses (the percentage of participants with a subthreshold or full diagnosis was below 10% for all three disorders at every wave); therefore, we were unable to examine the association between effortful control and externalizing disorder

diagnoses. Nonetheless, understanding how effortful control co-develops with externalizing symptoms in community samples can inform our understanding of how youths' ability to regulate begins to spiral out of control. Fourth, it is important to replicate the present findings in other ethnic groups to establish generalizability of the findings beyond Mexican-origin youth.

Fifth, although we were able to examine parent ASPD symptoms as an antecedent to the development of effortful control and ADHD, ODD, and CD symptoms, it was beyond the scope of the current study to examine the vast array of other risk and protective factors theoretically associated with these constructs. It is possible that different forms of parent psychopathology, such as ADHD symptoms, are more relevant for influencing the development of the child's ADHD and ODD symptoms, compared to parent ASPD symptoms. Moreover, there are many other etiological factors, aside from parent psychopathology, that may influence the development of effortful control and externalizing symptoms, and a more comprehensive analysis of etiological factors would provide better evidence for (or against) their construct similarity (vs. distinctiveness). This is an important avenue for future research. Finally, the association between parent ASPD symptoms and child effortful control/externalizing problems may be due to shared genetic effects. Although not possible to test with data from the California Families Project, longitudinal twin designs would allow future researchers to tease apart genetic and environmental effects, in order to better understand the co-development of effortful control and externalizing problems and their shared etiology.

Conclusion

Aligned with a developmental psychopathology perspective, we examined the extent to which deviations from normative personality development are related to the escalation of psychopathological symptoms. Generally, our research suggests that greater decreases in effortful control are related to greater increases in externalizing symptoms from late childhood through adolescence. We also found evidence to suggest that ADHD and ODD symptoms (and CD to a lesser extent) develop through a shared externalizing pathway, raising more questions about the need to study developmental processes common and unique to each disorder. However, there is less evidence to suggest that effortful control and externalizing symptoms lie on the same spectrum developmentally, given that the contributions of effortful control and CD symptoms to the shared trajectory were much weaker than those of ADHD and ODD symptoms (and that a factor analysis of longitudinal trajectories indicated that the effortful control facets form a separate factor from the externalizing facets). Additionally, parent ASPD symptoms may increase the risk of children experiencing greater *increases* in CD symptoms during adolescence, which suggests that there may be something about more severe forms of externalizing behavior that is uniquely transmitted apart from the basic tendency for poor effortful control. Although the present findings provide an interesting window into the ways that effortful control and externalizing problems co-develop, they raise a number of questions to be explored in future research. There are a vast number of opportunities for future research to explore how cognitive, affective, and behavioral systems across multiple levels of analysis lead to the development of externalizing problems, especially among ethnic minority youth.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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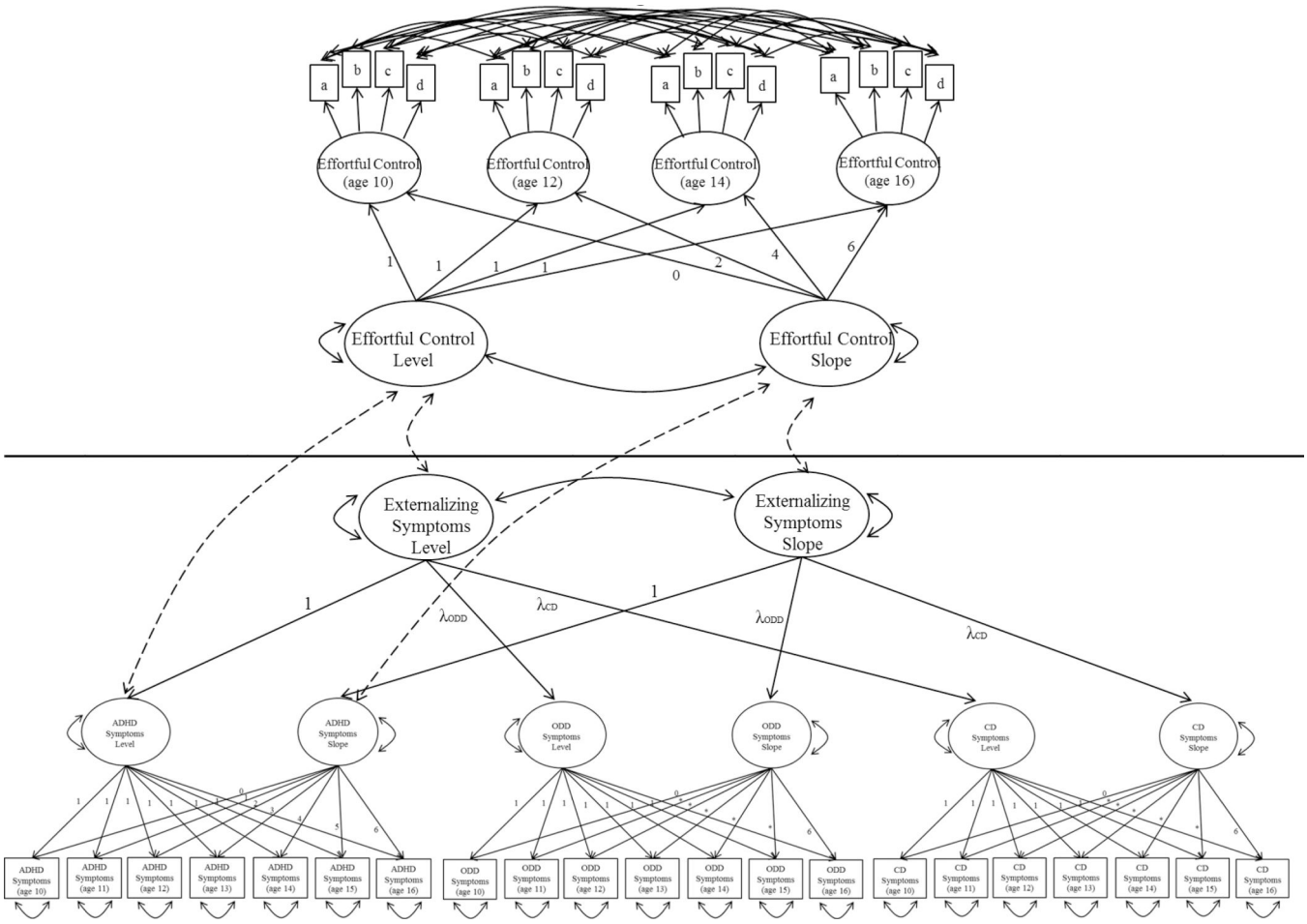

Figure 1.

Factor of Curves Model (with extension).

Note. Below the bold, horizontal line is a visual depiction of the FOCUS model with ADHD, ODD, and CD trajectories. Above the bold line demonstrates an extended FOCUS model with correlations among the level and slope of effortful control and the FOCUS model levels and slopes. Specifically, the dotted covariances demonstrate an example of the extended FOCUS analysis for examining the associations between effortful control and externalizing symptoms and ADHD symptoms, simultaneously.

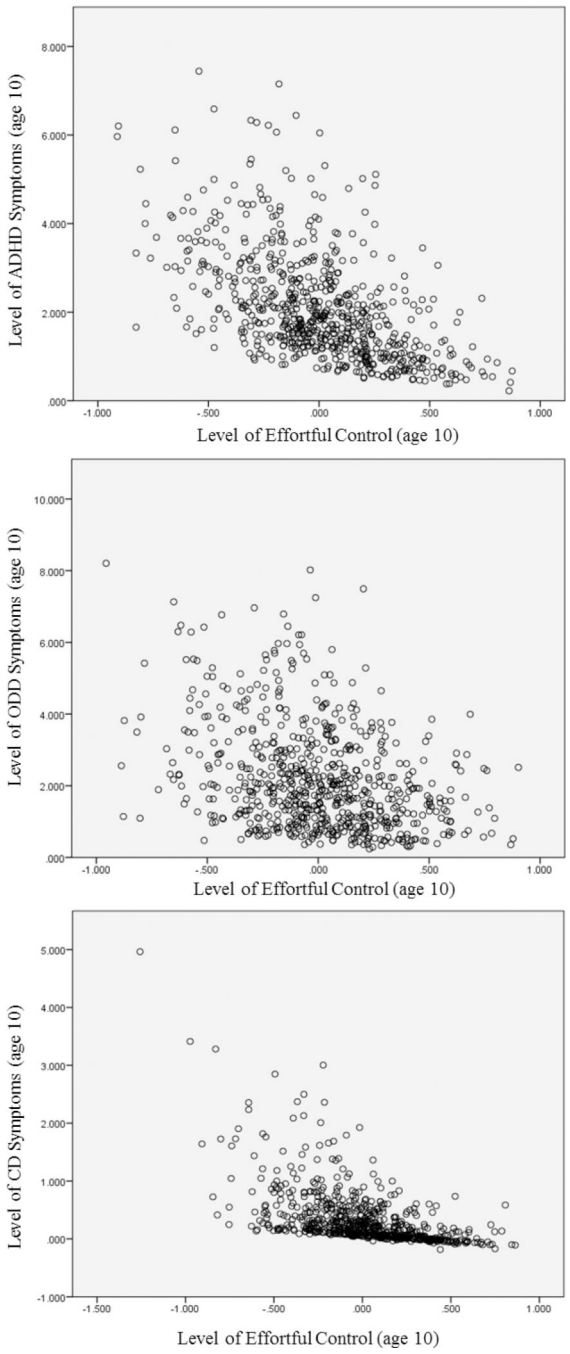

Figure 2.

Scatterplots of the correlation between the levels of effortful control and ADHD, ODD, and CD symptoms at age 10.

Note. ADHD = Attention-Deficit/Hyperactivity Disorder, ODD = Oppositional Defiant Disorder, CD = Conduct Disorder.

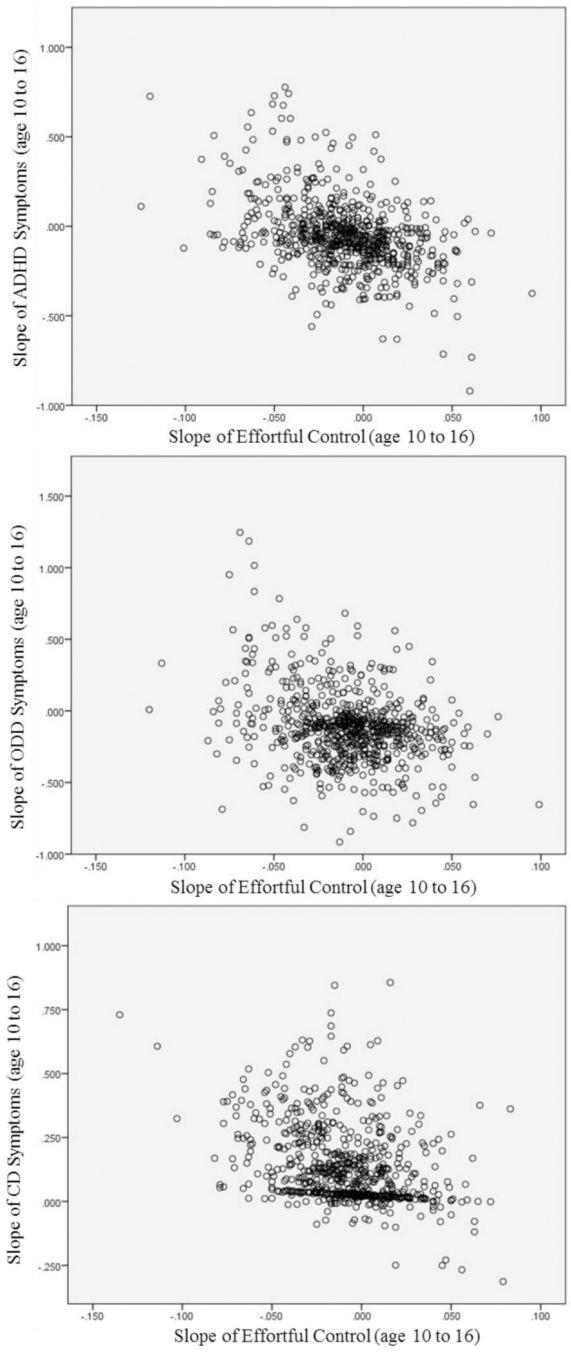

Figure 3.

Scatterplots of the correlation between the slopes of effortful control and ADHD, ODD, and CD symptoms from age 10 to 16.

Note. ADHD = Attention-Deficit/Hyperactivity Disorder, ODD = Oppositional Defiant Disorder, CD = Conduct Disorder.

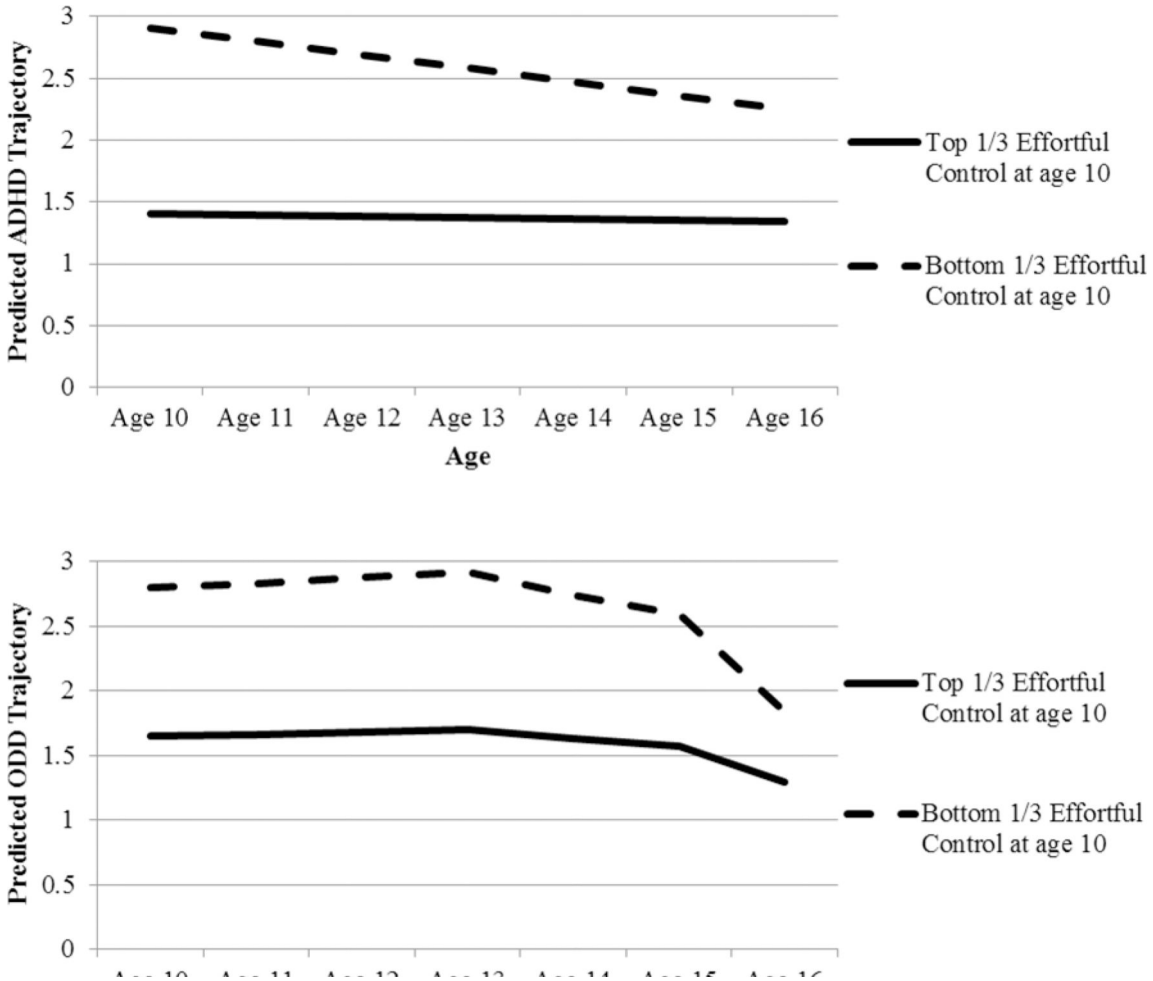

Figure 4.

Predicted ADHD and ODD trajectories based on initial levels of effortful control at age 10. *Note*. ADHD = Attention-Deficit/Hyperactivity Disorder, ODD = Oppositional Defiant Disorder.

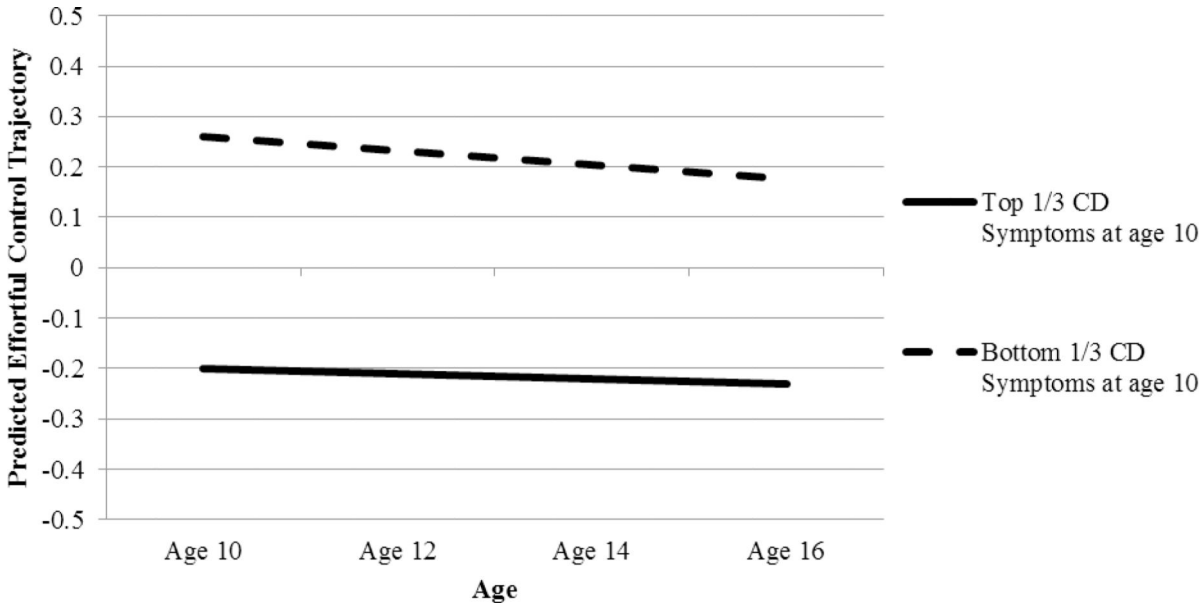

Figure 5.

Predicted effortful control trajectory based on initial levels of CD symptoms at age 10.

Note. CD = Conduct Disorder.

Table 1

Descriptive Statistics of Study Variables

	Effortful Control	ADHD		ODD		CD	
	M (SD)	M (SD)	Range	M (SD)	Range	M (SD)	Range
Age 10 (5th grade)	2.96 (.34)	2.23 (2.50)	0–17	2.17 (2.28)	0–11	.38 (.97)	0–8.5
Age 11 (6th grade)	--	1.72 (2.28)	0–15	1.94 (2.40)	0–11	.36 (1.01)	0–12
Age 12 (7th grade)	3.00 (.34)	2.02 (2.47)	0–14	2.13 (2.49)	0–11	.49 (1.04)	0–11
Age 13 (8th grade)	--	2.18 (2.60)	0–14	2.38 (2.65)	0–11	.77 (1.35)	0–9
Age 14 (9th grade)	2.94 (.35)	2.03 (2.47)	0–16	2.29 (2.62)	0–11	.97 (1.58)	0–10
Age 15 (10th grade)	--	1.92 (2.51)	0–16	2.22 (2.52)	0–11	1.19 (1.70)	0–9
Age 16 (11th grade)	2.94 (.33)	1.63 (2.35)	0–12	1.56 (2.09)	0–11	1.26 (1.77)	0–9

Note. Values are descriptive statistics of the observed variables. ADHD=Attention-Deficit Hyperactivity Disorder, ODD=Oppositional Defiant Disorder, CD=Conduct Disorder. M=Mean, SD=Standard deviation.

Table 2

Co-Development of Effortful Control and ADHD, ODD, and CD Symptoms

	r (Level$_1$, Level$_2$)	r (Slope$_1$, Slope$_2$)	r (Level$_1$, Slope$_2$)	r (Level$_2$, Slope$_1$)
1. Effortful Control & 2. ADHD	−.47* [−.58, −.36]	−.36* [−.56, −.17]	.20* [.06, .35]	.15 [−.02, .32]
1. Activation Control & 2. ADHD	−.50* [−.63, −.36]	−.41* [−.68, −.15]	.13 [−.04, .30]	.11 [−.11, .33]
1. Inhibitory Control & 2. ADHD	−.49* [−.65, −.32]	−.38* [−.69, −.06]	.32* [.13, .50]	.13 [−.13, .40]
1. Attention & 2. ADHD	−.67* [−.82, −.51]	−.45* [−.72, −.19]	.27* [.08, .46]	.13 [−.09, .34]
1. Effortful Control & 2. Inattention	−.55* [−.67, −.42]	−.48* [−.69, −.28]	.21* [.06, .36]	.21 [−.02, .43]
1. Effortful Control & 2. Hyperactivity	−.34* [−.46, −.22]	−.12 [−.33, .08]	.08 [−.06, .24]	.09 [−.08, .28]
1. Effortful Control & 2. ODD	−.30* [−.40, −.21]	−.21* [−.35, −.06]	.19* [.08, .29]	−.08 [−.22, .07]
1. Activation Control & 2. ODD	−.34* [−.46, −.22]	−.23* [−.42, −.04]	.18* [.06, .31]	−.12 [−.30, .07]
1. Inhibitory Control & 2. ODD	−.34* [−.47, −.21]	−.21* [−.42, −.003]	.25* [.12, .38]	−.10 [−.31, .10]
1. Attention & 2. ODD	−.22* [−.34, −.10]	−.10 [−.25, .05]	.11 [−.01. .23]	−.03 [−.18, .12]
1. Effortful Control & 2. Emotion Dys.	−.26* [−.37, −.15]	−.28* [−.43, −.14]	.18* [.08, .29]	.09 [−.07, .25]
1. Effortful Control & 2. Defiance	−.39* [−.49, −.28]	−.22* [−.36, −.08]	.18* [.07, .28]	.03 [−.12, .19]
1. Effortful Control & 2. CD	−.45* [−.56, −.34]	−.28* [−.45, −.11]	−.06 [−.18, .06]	.22* [.06, .39]
1. Activation Control & 2. CD	−.39* [−.52, −.27]	−.27* [−.49, −.05]	−.08 [−.21, .05]	.13 [−.08, .33]
1. Inhibitory Control & 2. CD	−.44* [−.58, −.30]	−.28* [−.53, −.03]	.00 [−.14, .15]	.11 [−.13, .35]
1. Attention & 2. CD	−.42* [−.55, −.29]	−.22* [−.40, −.04]	.00 [−.13, .14]	.21* [.03, .38]

Note. ADHD = Attention-Deficit/Hyperactivity Disorder. ODD = Oppositional Defiant Disorder. CD = Conduct Disorder. r = Correlation. Values in brackets are the 95% confidence intervals. The '1.' and '2.' in the row labels correspond to the subscript numbers in the column headers, in order to distinguish which constructs are the levels/slopes in the analysis of correlation coefficients.

* $p < .05$

Table 3

The Associations between Effortful Control and Higher-Order and Lower-Order Externalizing Problems in the FOCUS Model

	r (Level$_1$, Level$_2$)	r (Slope$_1$, Slope$_2$)
1. Effortful Control & 2. Externalizing Symptoms	−.52* [−.63, −.41]	−.47* [−.67, −.27]
1. Effortful Control & 2. ADHD Symptoms	−.12 [−.29, .04]	−.45 [−1.68, .78]
1. Effortful Control & 2. Externalizing Symptoms	−.63* [−.74, −.52]	−.56* [−.77, −.34]
1. Effortful Control & 2. ODD Symptoms	.43* [.12, .74]	.15 [−.12, .42]
1. Effortful Control & 2. Externalizing Symptoms	−.53* [−.63, −.42]	−.51* [−.69, −.32]
1. Effortful Control & 2. CD Symptoms	−.17* [−.28, −.06]	−.15 [−.30, .003]

Note. ADHD = Attention-Deficit/Hyperactivity Disorder. ODD = Oppositional Defiant Disorder. CD = Conduct Disorder. r = Correlation. Values in brackets are the 95% confidence intervals. The '1.' and '2.' in the row labels correspond to the subscript numbers in the column headers, in order to distinguish which constructs are the levels/slopes in the analysis.

* $p < .05$.

Table 4

Loadings of Effortful Control and Externalizing Symptom Trajectories on Factors

	Standardized Factor Slopes	
	Factor 1 Loadings	**Factor 2 Loadings**
ODD: Defiance	**.67**	.06
ODD: Emotion Dysregulation	**.64**	.06
ADHD: Inattention	**.61**	.01
ADHD: Hyperactivity	**.49**	−.12
Conduct Disorder	**.44**	−.04
Effortful Control: Attention Control	.05	**.76**
Effortful Control: Activation Control	−.03	**.55**
Effortful Control: Inhibitory Control	−.02	**.41**

Note. Values in the table are rotated factor loadings from a principal axis factor analysis (with direct oblimin rotation) of slope factors estimated from univariate latent growth curve models. ODD = Oppositional Defiant Disorder. ADHD = Attention-Deficit/Hyperactivity Disorder.

Table 5

Influence of Mother and Father ASPD Symptoms on Initial Level and Change Over Time

	Mother ASPD → Level (Age 10 Symptoms) β [95% CI]	Mother ASPD → Slope (Change Over Time) β [95% CI]
Effortful Control	−.10* [−.20, −.004]	−.09 [−.22, .04]
ADHD Symptoms	.15* [.04, .25]	.02 [.10, .14]
ODD Symptoms	.10* [.02, .19]	.01 [−.08, .10]
CD Symptoms	.10* [.01, .20]	.13* [.03, .22]
	Father ASPD → Level (Age 10 Symptoms) β [95% CI]	**Father ASPD → Slope (Change Over Time) β [95% CI]**
Effortful Control	−.15* [−.27, −.03]	.21* [.05, .37]
ADHD Symptoms	.22* [.10, .35]	−.04 [−.19, .11]
ODD Symptoms	.16* [.05, .26]	.01 [−.11, .12]
CD Symptoms	.10 [−.04, .24]	.13* [.002, .26]

Note. ADHD = Attention-Deficit/Hyperactivity Disorder. ODD = Oppositional Defiant Disorder. CD = Conduct Disorder. ASPD = Antisocial Personality Disorder. β=Standardized regression coefficient; CI=Confidence interval.

* $p < .05$.