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Links between within-person fluctuations in hyperactivity/attention problems and subsequent conduct problems

Anne B. Arnett¹, Bruce F. Pennington¹, Jami F. Young², and Benjamin L. Hankin¹

¹Department of Psychology, University of Denver, Denver, CO, USA

²Department of Clinical Psychology, Rutgers University, Piscataway, NJ, USA

Abstract

Background—The onset of hyperactivity/impulsivity and attention problems (HAP) is typically younger than that of conduct problems (CP), and some research supports a directional relation wherein HAP precedes CP. Studies have tested this theory using between-person and between-group comparisons, with conflicting results. In contrast, prior research has not examined the effects of within-person fluctuations in HAP on CP.

Method—The current study tested the hypothesis that within-person variation in HAP would positively predict subsequent within-person variation in CP, in two population samples of youth (N=620) who participated in identical methods of assessment over the course of 30 months. Three-level, hierarchical models were used to test for within-person, longitudinal associations between HAP and CP, as well as moderating effects of between-person and between-family demographics.

Results—We found a small but significant association in the expected direction for older youth, but the opposite effect in younger and non-Caucasian youth. These results were replicated across both samples.

Conclusions—The process by which early HAP relates to later CP may vary by age and racial identity.

Keywords

ADHD; conduct problems; disruptive behavior; within-person; longitudinal

Introduction

Comorbidity between symptoms of hyperactivity/impulsivity and attention problems (HAP) and conduct problems (CP) has been a topic of interest in the field of child psychology within the last two decades (Beauchaine, Hinshaw, & Pang, 2010; Loeber, Burke, Lahey, Winters, & Zera, 2000). These symptom clusters are core features of common childhood disorders, namely attention deficit hyperactivity disorder (ADHD), which is prevalent in

Correspondence Anne B. Arnett, Department of Psychology, University of Denver, 2155 S. Race St., Denver, CO 80208, USA; arnett.anne@gmail.com.

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about 6 - 11 percent of school age children (Willcutt, 2012) and oppositional defiant disorder (ODD) and conduct disorder (CD), which are estimated to affect 10 percent of the population (Nock, Kazdin, Hiripi, & Kessler, 2006; Nock, Kazdin, Hiripi, & Kessler, 2007). HAP and CP share phenotypic and genotypic latent traits as well as common negative outcomes, including peer problems, early adult arrest, substance abuse, and antisocial personality disorder (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Beauchaine et al., 2010; Loeber et al., 2000; Pennington, 2002; Tuvblad, Zheng, Raine, & Baker, 2009; Waschbusch, 2002). Yet, despite their high rates of comorbidity, overlapping etiological factors, and shared sequelae, HAP and CP are distinct symptom clusters, and only a proportion of youth with high levels of HAP also have elevated CP (Biederman, Newcorn, & Sprich, 1991).

One theory of comorbidity, the developmental cascades model (Masten & Cicchetti, 2010), describes the longitudinal relations between two symptom clusters as encompassing both direct and indirect effects. In line with this model, some research has indicated a developmental progression in which CP follows onset of HAP, specifically in the presence of harsh parenting (Beauchaine et al., 2010; Lahey, McBurnett, & Loeber, 2000). Patterson's proposed coercive cycle (Patterson, 1976; Patterson, DeGarmo, & Knutson, 2000) further specifies this pattern by describing transactions in which HAP elicits inconsistent and coercive parenting practices, which in turn increase the child's propensity toward coercive and antisocial behaviors, which further elicits coercive parenting, and so on.

Although widely accepted as viable, Patterson's coercive theory has not been adequately tested. For example, using structural equation modeling to test their own hypothesis, Patterson and colleagues (2000) found that a latent hyperactivity factor in fourth grade did not predict delinquency by ninth grade when early antisocial behaviors were included in the model. However, this analysis used only a few, distant time points and depended on latent traits across a group of youth. This approach could have masked acute temporal relations between early hyperactivity and antisocial behaviors, as well as individual variability in HAP-CP relational patterns.

Other studies have also reported that early childhood antisocial and aggressive behaviors mediate the association between early HAP and later CP (Cadoret & Stewart, 1991; Patterson, DeGarmo, & Knutson, 2000; Young, Heptinstall, Sonuga-Barke, Chadwick, & Taylor, 2005). Further, Lahey et al. (2009) found that early hyperactivity did not uniquely relate to later CP once genetic and environmental factors influencing early CP were controlled. This suggests that the longitudinal relation between the behavioral constructs may be accounted for by underlying genetic and environmental risk for CP.

Conflicting results with regard to the joint developmental progression of HAP and CP likely relate to previous studies' use of few time points and/or between-person analyses. As Masten and Cicchetti (2010) described, in order to truly model developmental cascades, multiple, repeated assessments over time are necessary. Similarly, Patterson's coercive cycle implies that an individual's fluctuations in HAP severity at any given time point will relate to the same individual's subsequent CP just a short time later. In other words, if a youth is having a 'severe HAP' month, then according to Patterson's theory, severe CP should follow.

Thus, studies of associations between HAP and CP across only a few, distant time points do not adequately test these theories.

Additionally, most studies that have targeted this question have focused on high-risk, clinically-referred boys. These restricted samples limit the amount of behavioral variance, which would underestimate the correlation between HAP and CP longitudinally. Further, most research indicates that HAP exists along a continuum of behavioral severity (e.g. Arnett et al., 2012). Thus, it is important to test these associations across the full spectrum of behaviors.

The current study aimed to fill a gap in the disruptive behavior literature by examining associations between within-person fluctuations in HAP and CP in a population sample from three salient age cohorts. The study design included ten repeated assessments over the course of 30 months. We used hierarchical linear modeling to test three-level, repeated measures models of HAP and CP. We hypothesized that within-person fluctuations in HAP (wpHAP) would significantly predict within-person variation in CP (wpCP) at the subsequent time point, but not vice versa. WpHAP and wpCP were defined as deviations from the youth's average HAP or CP severity, respectively.

Given that the influence of family environment on neuropsychological development is greater for younger youth (Nisbett, 2009), we proposed that Patterson's coercive cycle would be supported if the effect of wpHAP on later wpCP was stronger in younger youth. We further expected that the effect of wpHAP on subsequent wpCP would be stronger in the context of previously established risk covariates: more negative parenting, lower SES, and more severe average HAP (i.e. across all time points) (Barkley, Fisher, Edelbrock, & Smallish, 1991; Chronis et al., 2007; Loeber & Keenan, 1994; Patterson et al., 2000; Waschbusch, 2002). Additionally, we predicted that the association between wpHAP and later wpCP would be stronger for females, due to a paradoxical gender effect wherein fewer females exhibit HAP, but among those who do, there are relatively higher rates of comorbid CP compared to males with HAP (Loeber & Keenan, 1994).

Prior literature has also documented a weaker association between harsh parenting and disruptive behavior outcomes in African American youth relative to Caucasian youth (Hill & Bush, 2001). The current samples were representative of the U.S. with regard to ethnic and racial backgrounds; non-Caucasian youth encompassed a broad range of racial identities, including many youth who identified as multi-racial, which limited our power to test for race-specific effects. Thus, non-Caucasian youth were grouped together under the category of 'minority'. Given the crudeness of this measure, we did not expect to find significant effects of minority race on the lagged wpHAP-wpCP association.

Finally, we repeated all analyses testing for the opposite directional effect of wpCP on subsequent wpHAP, as a measure of discriminant validity for our hypotheses. We did not expect to find a significant effect of wpCP on subsequent wpHAP.

Methods

Participants

Youth in third, sixth, and ninth grades were recruited from schools in the greater Denver, CO and New Brunswick, NJ areas for enrollment in a multi-site, longitudinal study of mood disorders. Exclusionary criteria included autism spectrum or psychotic disorders, IQ less than 70, and non-English speaking. Recruitment procedures have been described in detail in previously published studies (Cohen, Young, Gibb, Hankin, & Abela, 2014; Hankin, Jenness, Abela, & Smolen, 2011). The current study only used data from participants who had completed at least three of the relevant time points. The final samples included 105 third grade, 119 sixth grade, and 104 ninth grade youth ($n = 328$) recruited from Denver and 82 third grade, 108 sixth grade, and 102 ninth grade youth ($n = 292$) from New Brunswick (total $n = 620$). Participants' ages ranged from 7 to 16 years at baseline; means and standard deviations are detailed in Table 1.

Sample demographics are listed in Table 1. A minority of participants in each sample also had a sibling enrolled in the study. The Denver and New Brunswick samples did not differ with regard to percent female, minority ethnicity, average SES, or HAP or CP severity.

Procedures

The current study performed secondary analyses on disruptive behavior symptoms in a sample of youth recruited for a study of internalizing symptoms. Although the goal of the parent study was to examine internalizing symptoms, participants were recruited from the community and symptom rates were representative of the general population; thus, the purpose of the parent study had no effect on our results. Each youth and a parent participated in a baseline laboratory visit. Parents provided written consent for themselves and their youth. The youth provided written assent. Thereafter, every three months following the baseline visit, participants completed behavioral questionnaires for a total of 10 waves of follow-up assessment over 30 months. An additional laboratory visit took place at the 18-month follow-up. All procedures were approved by the University of Denver and Rutgers University institutional review boards.

Measures

Disruptive behavior symptoms—Child HAP and CP symptoms were measured using the Strengths and Difficulties Questionnaire, Parent Report (SDQ; Goodman, 1997) every three months, from the 3- through 30-month follow-ups, for a total of 10 waves. The SDQ was not collected at the 18-month follow-up at the Denver site. The SDQ scales are highly correlated with other established parent-report measures of CP and HAP symptoms, including the Rutter scales (HAP $r=.88$; CP $r=.82$) (Goodman, 1997). Internal validity for the SDQ has been established for a large ($N=9,878$) sample of children age 4-17 (Bourdon, Goodman, Rae, Simpson, & Koretz, 2005). The SDQ HAP and CP scales comprise five items each (see Table 2). Parents rate the items on a scale of 0 'not true' to 2 'certainly true'. Items were reverse coded when necessary such that higher scores reflected more symptoms. Although the original measure was designed to assess behavior over the past six months, participants in this study were asked to rate behavior over the course of the past three

months. Overall reliabilities of the scales across all time points were high (HAP $\alpha = .96$; CP $\alpha = .94$) and average correlations between consecutive time points, or three months (HAP $r = .73$; CP $r = .68$) were similar to those across two time points, or six months (HAP $r = .70$; CP $r = .64$). Moreover, these coefficients were very similar to those reported by Goodman (2001), who reported test-retest reliability of the SDQ scales across 4-6 months in a sample of 10,438 British youth.

Socioeconomic status (SES)—SES was ascertained at baseline using a demographic parent report. Information about both parents' education levels and specific occupations was used to calculate the Hollingshead Four-Factor Index (Adams & Weakliem, 2011).

Race—Minority status was defined as non-Caucasian, and included participants who identified as 'mixed race' or 'other.' The child's race and ethnicity were reported by the parent as part of the baseline demographic questionnaire.

Negative parenting—Negative parenting was measured at baseline using the Alabama Parenting Questionnaire (APQ), which is a parent self-report. Three negative parenting subscales measuring poor monitoring/supervision, inconsistent discipline, and corporal punishment were created following guidelines published by Shelton, Frick & Wootton (1996). Scales were averaged in this study to create a single measure of negative parenting practices. The scales showed moderate internal validity in both the Denver and New Brunswick samples (*Chronbach's alphas* = .51 and .56, respectively).

Hierarchical linear modeling analysis plan

Three-level, nested hierarchical linear models were tested using HLM Version 6.08 (Raudenbush, Bryk, Cheong, & Cogdon, 2004). Variance was divided into within-person, time varying effects at level-1; between-person effects at level-2; and between-family effects at level-3.

At level-1, we tested our hypothesis that wpCP at each time point would be predicted by wpHAP severity at the previous time point (i.e. lagged wpHAP), controlling for the contemporaneous association with wpHAP and the autoregressive association with lagged wpCP. We tested both a one-time point lag and two-time point lag (three and six months, respectively) in wpHAP to allow for the possibility of a sleeper effect. Additionally, we included the interaction of lagged wpHAP and age to test our hypothesis that if coercive parenting mediated the wpHAP to wpCP progression, the association would be stronger in younger youth.

At level-2, we tested our hypothesis that the longitudinal association between within-person variation in HAP and CP would be even stronger for youth with more negative parenting and higher average HAP severity, as well as for females. Negative parenting was included at this level (between-person) rather than level-3 (between-family) because parents were asked to complete the APQ with a specific child in mind, and parenting practices can vary with each child, particularly in the presence of externalizing behavior.

At level-3, we tested our theory that higher family stress in the form of lower SES would increase the magnitude of the longitudinal association between within-person HAP and CP at the first level, and that minority ethnicity would not have an effect on this association. There were no intercept moderators included at levels -2 or -3 because the outcome variable, wpCP, represented a time-varying deviation from an individual's average CP severity; thus, there were no between-person or between-family differences on the intercept.

Next, we tested the opposite model with wpHAP as the outcome, and lagged wpCP as the level-1 predictor of interest. We hypothesized that there would not be a statistically significant effect of lagged wpCP on wpHAP.

Model specification was done incrementally (Snijders & Bosker, 2012), by adding predictors and random slopes one level at a time. All variables and random slopes that were significant at $p < .05$ at initial entry into the model were retained in the final models, along with level-1 predictors of primary interest. Lastly, sample was added as a level-3 moderator of all significant level-1 and level-2 effects in order to test for replication of results across the Denver and New Brunswick samples.

Results

Preliminary analyses

Data transformation—All variables were initially examined for normality. SDQ HAP and CP scores were winsorized to within three standard deviations of the mean at each time point, within sample. All variables then demonstrated skew and kurtosis values in the acceptable range (absolute value < 1.6).

HAP and CP severities—Participants' clinical severities for HAP and CP symptoms were estimated using sex-specific normative data collected on 9,878 7-17 year old youth from the National Health Interview Survey (Bourdon et al., 2005), available on the SDQ website (www.sdqinfo.org/USnorm.html). Results are reported by sample in Table 1. Altogether, 5% of participants scored at least 1.5 standard deviations above the sex norm for average HAP severity across all time points; 6% scored in this range for average CP severity.

Within-person CP outcomes

When all level-1 variables were included in the model, the association of interest between lagged wpHAP and wpCP showed an interaction with age. While wpCP at a given time point was negatively predicted by wpHAP lagged by one time point ($b = -.07, p = .018$), and age was not a significant predictor of wpCP on its own ($b = .00, p = .564$), there was a positive effect of the interaction between age and the lagged wpHAP score on subsequent wpCP ($b = .02, p < .001$). This interaction effect indicated that the association between lagged wpHAP and wpCP became more positive with age. Additional analyses using dummy variables for the age cohorts at level-2 indicated that the sixth and ninth grade cohorts had comparable, positive associations between lagged wpHAP and wpCP, while the third grade cohort showed a significantly more negative association relative to the ninth graders ($b = -.07, p = .011$). As expected, contemporaneous wpHAP was positively

associated with wpCP ($b = .19, p < .001$). The autoregressive effect of lagged wpCP on subsequent wpCP was negative ($b = -.06, p = .006$), likely due to regression to the mean.

As planned, we also checked for a sleeper effect by testing the predictive effect of wpHAP lagged by two time points (i.e. six months prior). This model resulted in non-significant beta values for the two-time point lagged wpHAP ($b = .03, p = .250$), age ($b = .00, p = .666$), and their interaction ($b = .00, p = .745$). Thus, we reverted to the one-time point lagged level-1 model for the subsequent analyses.

We added random slopes for wpHAP, lagged wpHAP, the interaction term, and lagged wpCP; all four variances were statistically significant ($p < .01$), indicating between-person variability. To compare effect sizes of the significant level-1 predictors, we removed them one at a time from the level-1 random model, and calculated a pseudo- R^2 as a proportion of the change in variance of the level-1 intercept. The association with concurrent wpHAP accounted for the greatest proportion of variance at 13%, followed by lagged wpCP (6%), lagged wpHAP (2%) and a negligible effect size for the interaction term.

Next, we retained these random slopes at level-1 and added our proposed level-2 moderators. Contrary to our predictions, none of the proposed between-person variables (negative parenting, average HAP or sex) significantly moderated the association between lagged wpHAP and wpCP, which remained negative and statistically significant. Thus, all of the level-2 moderators were dropped from the model.

Next, we tested for an effect of lower SES at the between-family level on the within-person association between lagged wpHAP and wpCP. We also tested for a moderating effect of minority status, although we proposed that our non-Caucasian population was too diverse to result in a significant interaction. Contrary to our prediction, when both SES and minority status were included in the model, SES was not significant ($b = .00, p = .929$), but non-Caucasian youth demonstrated a weaker (more negative) association between lagged wpHAP and wpCP outcomes ($b = -.10, p = .010$) relative to Caucasian youth. For Caucasian youth, the effect of lagged wpHAP on subsequent wpCP was not statistically significant ($b = -.05, p = .146$); however, the random slope was still significant ($\sigma^2 = .11, p < .001$), indicating that minority status did not explain all of the between-person variance in the effect. Finally, we added sample as a moderator of all of the significant level-1 effects. The purpose of this final analysis was to replicate our results across both samples. As predicted, sample was not a significant moderator of any of the level-1 associations, indicating that the results replicated across both the Denver and New Brunswick samples.

Results of the final, three-level hierarchical linear model with lagged wpHAP predicting subsequent wpCP are reported in Table 3. As seen in Figure 1, the association between lagged wpHAP and wpCP was positive for older youth. However, minority and youngest youth demonstrated a negative association. The final model accounted for 22% of the within-person variance in CP severity.

Within-person HAP outcomes

As expected, wpHAP was not predicted by the one-time point lagged wpCP ($b = .02, p = .729$). Further, neither age ($b = -.01, p = .137$) nor the interaction between age and lagged wpCP ($b = -.00, p = .942$) was significantly related to wpHAP outcomes. When the interaction term was removed, the lagged wpCP effect remained non-significant. Effect sizes as calculated by pseudo R^2 were negligible for all level-1 predictors. As planned, we next tested the same level-1 model using wpCP lagged by two time points. Again, the effect was not significant. These results were consistent with our original hypothesis that we would not see an association between lagged wpCP and later wpHAP, as well as with literature supporting HAP as a developmental precursor of CP, but not vice versa. Thus, we did not continue to evaluate this comparison model beyond level -1.

Discussion

We tested the hypothesis that individual fluctuations in HAP severity would precede and predict individual fluctuations in CP, consistent with the developmental pattern described by Patterson's coercive cycle. Our study differed from prior research (e.g. Gittelman, 1985; Loeber et al., 2000; Mannuzza et al., 2004) in that we tested the effects of within-person differences in behavioral severity over multiple time points, rather than executing between-person comparisons over just a few time points. As predicted, wpHAP did predict subsequent wpCP variation, but not vice versa; however, the predicted effect was only evident in older youth, and the variance explained by the association was minimal. Among the youngest youth, a negative association was found. As the youngest cohort did not have more variance than the older cohorts, it is unlikely that this is due to regression to the mean. Following our initial hypotheses, our results suggest that the HAP to CP behavioral developmental pattern may depend on environmental mediators that are more salient in older youth, such as deviant peer relations or academic stress. Alternately, lack of measurement invariance across ages may explain the difference, if the behavioral items were less appropriate for the youngest youth.

Among non-Caucasian youth, the association between lagged wpHAP and wpCP was weaker than that for Caucasian youth (across all ages). Although our youth were highly diverse, approximately 10 percent of the parents who completed the questionnaires were of Hispanic/Latino descent. Parents in this ethnic group have previously been found to over-report problem behaviors (e.g. Gross et al., 2007), which could have affected the results of the non-Caucasian youth. Further, prior research has reported a weaker association between harsh parenting and CP in African American relative to European American youth (Hill & Bush, 2001), which would mitigate the transactions proposed by Patterson. Although normative clinical values for the SDQ were established using a U.S. sample that was overselected for African American and Hispanic youth (Bourdon et al., 2015), measurement invariance across these racial groups has not been extensively tested. Minority youth are generally underrepresented in research on disruptive behavioral problems and deserve specific attention in future research.

Our results partially supported the developmental cascades model; however, our study did not clarify the process by which HAP influences CP. Frameworks such as Jessor's Problem

Behavior Theory (Jessor, 1992) suggest that deviant behavior itself can place youth at risk for additional negative psychosocial symptoms; for example, a youth who steals or uses illegal drugs may be more likely to experience subsequent social isolation, academic failure, and anxiety. On the other hand, Patterson theorized that transactions between disruptive behavior and environmental risk were crucial for eliciting more severe behavior. In the case of HAP and CP, both direct and indirect associations are plausible, and not mutually exclusive. An example of a direct effect of HAP on CP would be a youth whose impulsivity and impaired social skills associated with HAP (Laird, Jordan, Dodge, Pettit, & Bates, 2001) led him to engage in mildly antisocial behaviors, such as stealing or fighting, which acted as gateway behaviors to more frequent and severe forms of CP.

One limitation of our study was that our sample did not include a very young cohort of children. Mediation by negative parenting may occur in children prior to third grade, with a transition to peer and other non-familial influences as youth get older. However, this does not explain the negative association between lagged wpHAP and later wpCP in the third grade cohort. Future examination of age as a moderator of HAP and CP comorbidity is warranted, preferably using mediation models and repeated measures of disruptive behaviors and environmental risk.

Importantly, the effect size for the association between lagged wpHAP and later wpCP was very small in the current study, even when age was taken into account. Our community sample had a low base rate of disorder, and we may have seen a larger effect among youth with greater within-person variance in behavioral severity over time. The lack of significant moderation by average behavioral severity at the second level suggests that our results applied to youth with extreme (i.e. disordered) behavior, but this result would be more conclusive in a sample with higher rates of disordered youth. This is likewise true for the lack of moderation by negative parenting, which suggested that the HAP to CP progression was not more likely to occur in highly dysfunctional parenting environments. However, it would be helpful to replicate this finding in a population sample that is enhanced for behavioral disorders and family risk, to insure adequate variance exists at both the typical and maladaptive ends of the behavioral and parenting continua.

We were somewhat limited by the use of the SDQ to measure disruptive behaviors in this study. Although a reliable instrument, the number of items comprising the HAP and CP scales was small, so we could not investigate specific associations among subtypes of symptom clusters, such as predominantly inattentive versus hyperactive/impulsive HAP, and aggressive versus covert CP. Previous literature suggests that the HAP to CP progression may be specific to the hyperactivity/impulsivity cluster of ADHD symptoms (Babinski, Hartsough, & Lambert, 1999). Thus, use of an expanded HAP measure might result in different findings for individual symptom subtypes. An additional limitation of the SDQ was that the scales measured only the dysfunctional ends of the symptom spectrums, and did not capture variance at the favorable ends of the distributions (e.g. good impulse control, high empathy). This limited variance in the behavioral severities and may have resulted in underestimated regression coefficients. Future research should aim to test our hypotheses using balanced and more in depth measurement tools at each time point.

Conclusion

Within-person variability in HAP predicted within-person variability in CP at the subsequent time point for older, Caucasian youth. This was the first study that we know of to test relations between within-person variability in disruptive behaviors. The results are consistent with both direct and indirect developmental relations between HAP and CP. However, our results do not clarify the process by which HAP potentiates to CP, and suggest that the association may vary as a function of age.

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Key Points

- HAP is thought to precede CP in some youth.
- Prior studies have focused on between group differences, which do not capture direct effects of HAP on CP within individuals.
- The current study found a unique effect of within-person fluctuations in HAP on subsequent within-person fluctuations in CP in older, Caucasian youth.
- The effect of HAP on CP may depend on peer stress and other environmental mediators that are salient to adolescents.

Figure 1.

The effect of within-person variation in HAP on subsequent within-person variation in CP is more positive for older youth

Table 1

Sample Demographics

	<u>Denver</u>	<u>New Brunswick</u>
<i>n</i>	328	292
Female	185 (56%)	160 (55%)
Minority Race	104 (32%)	108 (37%)
Age 3 rd Grade Cohort (SD)	8.76 (.61)	8.45 (.76)
Age 6 th Grade Cohort (SD)	11.63 (.52)	11.31 (.59)
Age 9 th Grade Cohort (SD)	14.39 (.55)	14.34 (.68)
Siblings	76 (23%)	66 (23%)
SES (SD)	48.65 (11.26)	49.19 (11.74)
HAP Severity (SD)	2.75 (1.97)	2.74 (1.95)
CP Severity (SD)	1.15 (1.25)	1.16 (1.15)
Elevated HAP	15 (4.6%)	17 (5.8%)
Elevated CP	21 (6.4%)	14 (4.8%)

Minority Race = non-Caucasian. Age = age in years at baseline. Siblings = participants with at least one sibling also in the study. SES = Hollingshead 4 factor index (Adams & Weakliem, 2011). Elevated HAP/CP = average SDQ *z*-score greater than 1.5 for HAP/CP scales.

Table 2

Items in the HAP and CP Scales of the Strengths and Difficulties Questionnaire

<u>HAP</u>	<u>CP</u>
1 Restless, overactive, cannot stay still for long	1 Often has temper tantrums or hot temper
2 Constantly fidgeting or squirming	2 Generally obedient, usually does what adults request *
3 Easily distracted, concentration wanders	3 Often fights with other children or bullies them
4 Thinks things out before acting *	4 Often lies or cheats
5 Sees tasks through to the end, good attention span *	5 Steals from home, school, or elsewhere

* Reverse coded.

Table 3

Three-Level Hierarchical Linear Model with Lagged wpHAP Predicting wpCP

Fixed Effects	Parameter	B (SE)	t (df)
Intercept	Intercept δ_{000}	-.03 (.04)	-.74 (410)
Age	Intercept δ_{100}	.00 (.01)	.63 (2053)
Concurrent wpHAP	Intercept δ_{200}	.18 (.02)***	10.11 (53)
Lagged wpHAP	Intercept δ_{300}	-.05 (.03)	-1.47 (53)
	Minority δ_{301}	-.10 (.04)**	-2.70 (53)
Lagged wpHAP $\times$ Age	Intercept δ_{400}	.01 (.00)**	3.23 (53)
Lagged wpCP	Intercept δ_{500}	-.08 (.03)**	-3.17 (53)

Variance Components	σ^2	χ^2 (df)
Level 1, σ^2_e	.46	
Level 2, σ^2_0	0	Too few df
Level 3, u^2_{00}	0	248.04 (410)
Concurrent wpHAP, σ^2_2	.03***	460.34 (262)
Lagged wpHAP, σ^2_3	.11***	427.62 (262)
Lagged wpHAP $\times$ Age, σ^2_4	.00**	432.44 (262)
Lagged wpCP, σ^2_5	.06***	472.36 (262)

*
 $p < .05$,**
 $p < .01$,***
 $p < .001$.

Sample = New Brunswick sample.