HHS Public Access

Author manuscript

Clin Psychol Sci. Author manuscript; available in PMC 2026 January 10.

Developmental Pathways of Inattention, Hyperactivity/Impulsivity, and Disruptive Behaviors: A Longitudinal, Person-Centered Approach

Jessica N. Smith[1], **Stefany Coxe**[1,2], **Morgan L. Jusko**[1], **Joseph S. Raiker**[3], **Justin Parent**[4], **Elizabeth Nousen**[5], **Jessica Tipsord**[5], **Leeza Maron**[5], **Erica D. Musser**[6]

[1]Department of Psychology, Florida International University

[2]Cancer Institute and Department of Computational Biomedicine, Cedars-Sinai, Los Angeles, California

[3]Joon Health, Orlando, Florida; University of Rhode Island

[4]Department of Psychology, University of Rhode Island

[5]Department of Psychiatry, Oregon Health & Science University

[6]Department of Psychology, Barnard College, Columbia University

Abstract

In the present study, we explore the utility of latent transition analysis to better understand the developmental course of youths' externalizing psychopathology using the Oregon ADHD-1000 data set. Symptomatic latent classes at each age (i.e., 9, 12, and 15 years) were identified, and the corresponding latent transition probabilities and most common latent transition pathways were described. Impairment was examined for clinical validation. The most notable findings included a group of youths with persistent inattention from childhood through adolescence, distinct from youths who decreased in hyperactivity/impulsivity (HI) with age, although HI persisted for some. Oppositional defiant disorder (ODD) occurred only alongside HI and was related to greater HI

Corresponding Author: Jessica N. Smith, Department of Psychology, Florida International University, jessica.n.smith@cuanchutz.edu.

Author Contributions

Jessica N. Smith: Conceptualization; Data curation; Formal analysis; Methodology; Software; Visualization; Writing – original draft; Writing – review & editing.
Stefany Coxe: Conceptualization; Formal analysis; Methodology; Software; Visualization; Writing – review & editing.
Morgan L. Jusko: Conceptualization; Writing – review & editing.
Joseph S. Raiker: Joseph Raiker: Conceptualization; Methodology; Supervision; Writing – review & editing.
Justin Parent: Conceptualization; Methodology; Writing – review & editing.
Elizabeth Nousen: Data curation; Investigation; Resources; Writing – review & editing.
Jessica Tipsord: Data curation; Investigation; Resources; Writing – review & editing.
Leeza Maron: Investigation; Resources; Writing – review & editing.
Erica D. Musser: Conceptualization; Methodology; Supervision; Writing – original draft; Writing – review & editing.

Transparency

Declaration of Conflicting Interests

J. S. Raiker is employed by Joon Health and may have stock options in the company. The authors declare that there were no other conflicts of interest with respect to the authorship or the publication of this article.

Open Practices

This article has received the badge for Open Materials. More information about the Open Practices badges can be found at http://www.psychologicalscience.org/publications/badges.

persistence. HI did not proceed later ODD; rather, HI and ODD were aligned in childhood and diverged slightly in adolescence. Collectively, with the present study's findings, we emphasize the importance of methods that allow for open exploration of which and how many symptoms are relevant at various points in development.

Keywords

comorbidity; heterogeneity; longitudinal methods; developmental psychopathology; attention-deficit/hyperactivity disorder; open materials

Attention-deficit/hyperactivity disorder (ADHD) is one of the most prevalent mental-health diagnoses in childhood (e.g., median point prevalence of 3%; lifetime prevalence as high as ≈10%; Merikangas et al., 2009; Polanczyk et al., 2015) and is associated with academic, social, and familial impairment (Banaschewski et al., 2017). ADHD is a neurodevelopmental disorder that often onsets in early childhood and persists for many into adolescence (i.e., 50%–80%) and adulthood (i.e., 35%–65%; Eme, 2017; Franke et al., 2018; Sibley et al., 2012). In youths, the disorder is highly comorbid with disruptive behavior problems (DBPs), such as oppositional defiant disorder (ODD) and conduct disorder (CD). DBPs occur among 30% to 50% of ADHD cases (Maughan et al., 2004; Reale et al., 2017) and generally exacerbate impairment (Steinberg & Drabick, 2015; Szentiványi & Balázs, 2018). Despite a large body of literature focused on the longitudinal nature of ADHD, numerous questions persist, such as predictors of symptomatic persistence and remittance, continued impairment despite diagnostic remittance, a restricted inattentive subgroup of youths, and late-onset ADHD (Barkley, 2016; Franke et al., 2018; Frick & Nigg, 2012; Karalunas & Nigg, 2020; Sonuga-Barke et al., 2023). In addition, although there is literature focused on the developmental course of ODD/CD, there is surprisingly little work focused on the developmental course of ADHD and DBP comorbidity. In fact, there have been several calls for more nuanced longitudinal work focused on better understanding the developmental sequalae of ADHD and DBPs (e.g., Eme, 2017; Karalunas & Nigg, 2020).

There are two overarching limitations with the extant work documenting the course of ADHD and/or DBPs. First, the literature has largely ignored the role of DBP comorbidity in contributing to ADHD heterogeneity and vice versa (Feczko et al., 2019). That is, the literatures on ADHD heterogeneity and the disorder's comorbidity with DBPs are largely separate (Feczko & Fair, 2020). Second, the current literature that addresses ADHD heterogeneity and its comorbidity with DBPs consists mostly of cross-sectional studies (Karalunas & Nigg, 2020). These two issues will be discussed further in the following sections.

The Interdependent Nature of Heterogeneity and Comorbidity

There is a large literature base focused on ADHD heterogeneity (Luo et al., 2019). Historically, this work focused on the subtypes and presentations of ADHD spanning the third through fifth editions of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; Frick & Nigg, 2012). In particular, this work focused on the degree of similarity between these subtypes/presentations (i.e., differing by as little as the presence or absence

of one symptom from the opposite type) and their developmental instability (Frick & Nigg, 2012; Lahey et al., 2005; Willcutt et al., 2012). Here, the consensus has been that ADHD presentations are best thought of as useful descriptors of current clinical symptoms rather than meaningfully and mechanistically distinct and stable *types*, hence the change to a "presentations" moniker (Frick & Nigg, 2012). More recent work has focused on identifying potentially more meaningful features by which to characterize the disorder's heterogeneity (e.g., executive functioning, temperament; e.g., Fair et al., 2012; Goh et al., 2020; Martel, 2016; Martel et al., 2010; Rajendran et al., 2015); however, this literature has largely not included consideration of DBP comorbidity. Separately, there has been a great deal of work on DBP heterogeneity, such as subdimensions of ODD (e.g., headstrong, irritable, and hurtful; Stringaris & Goodman, 2009) and CD (e.g., with and without callous-unemotional traits; Frick, 2016). However, this work has typically focused on only one of these disorders and/or ODD/CD comorbidity rather than the comorbidity between DBPs and ADHD. Collectively, recruitment of pure samples (e.g., ADHD-only samples to study ADHD heterogeneity), statistical control for comorbidity, and/or exclusion of participants with subthreshold (ST) symptoms (Beauchaine et al., 2010; Caron & Rutter, 1991) limit consideration of the role of comorbidity in giving rise to heterogeneity.

Conversely, the literature focusing explicitly on comorbidity often uses analytic approaches that fail to consider heterogeneity by treating the disorders of interest as discrete, homogeneous entities, often by recruiting and analyzing distinct groups with different diagnoses (e.g., ADHD, ODD; Beauchaine & McNulty, 2013; Feczko et al., 2019). Such group-based analyses often control for common variance such that the ways in which groups are found to differ are likely no longer representative of the disorders in the general population (i.e., the shared externalizing liability between disorders is removed; Beauchaine et al., 2010). Other studies have used continuous approaches to study comorbidity (e.g., factors in a structural equation model with continuous scores for different disorders); although this approach avoids the use of arbitrary thresholds typically used for dichotomizing group membership, it continues to treat disorders as distinct, unitary categories (Feczko et al., 2019) and is still focused on finding differences rather than what may be shared among disorders, including the role of heterogeneity (Beauchaine & McNulty, 2013; Feczko et al., 2019; Lilienfeld & Treadway, 2016).

Heterogeneity and comorbidity are corresponding problems of the field's nosology: It is argued that the *DSM* has parcellated (what could be) single diagnostic entities into multiple excessively comorbid diagnostic labels and/or combined multiple entities under single diagnostic labels that are excessively heterogeneous (Lilienfeld & Treadway, 2016). Despite this theoretical understanding, the literature bases on ADHD heterogeneity and ADHD/DBP comorbidity tend to use analytic methods that limit insights about their convergence. The studies that have examined ADHD and DBP comorbidity with some attention to heterogeneity have focused mostly on associations with ADHD subtypes, especially hyperactivity/impulsivity (i.e., HI) and ODD diagnoses (e.g., Burns & Walsh, 2002; Evans et al., 2020; Harvey et al., 2016; Martel et al., 2010; Vos et al., 2022).[1] Such

[1]. Willcutt et al. (2012) conducted a thorough meta-analysis of 546 studies focused on ADHD subtypes and their associations with impairment, demographics, and comorbid psychopathology, including but not limited to ODD and CD. The metaanalysis was largely

studies commonly neglect to consider ODD heterogeneity (e.g., Burns & Walsh, 2002; Martel et al., 2010; Vos et al., 2022), assign individuals to diagnostic groups a priori, and/or use analytic techniques that control for shared variance (e.g., Burns & Walsh, 2002; Evans et al., 2020; Harvey et al., 2016). Person-centered approaches may be helpful in exploring which symptoms naturally co-occur within and between disorders. The only prior person-centered studies addressing these issues have focused specifically on ADHD heterogeneity (e.g., Fair et al., 2012; Goh et al., 2020; Martel, 2016; Martel et al., 2010; Rajendran et al., 2015) or ODD/CD heterogeneity (e.g., Althoff et al., 2014; Bolhuis et al., 2017; Herzhoff & Tackett, 2016) rather than ADHD/DBP comorbidity, and these studies have not been longitudinal.

A Developmental Understanding of Symptoms Over Time

A second issue is a limited understanding of how ADHD and DBPs present across development because many studies of heterogeneity and comorbidity are cross-sectional. Cross-sectional studies conducted at different points in development may come to very different conclusions regarding the relationships between symptoms within and between disorders that potentially shift over time (Beauchaine & McNulty, 2013). Although there have been longitudinal studies of ADHD and DBPs, much of this literature has focused on only ADHD (Barkley, 2016; Eme, 2017; Franke et al., 2018) or only DBPs (Bolhuis et al., 2017; Rowe et al., 2010; Whelan et al., 2013). Generally, these studies have concluded that ADHD onsets in the early school years (Nigg et al., 2020), that inattention (IN) is relatively stable, and that HI tends to decrease with age (Barkley, 2016; Campbell et al., 2014; Frick & Nigg, 2012). Regarding DBPs, these studies suggest that the onset of ODD tends to occur sometime in middle childhood (Biederman et al., 2008; Nock et al., 2007) and has a median duration of 6 years; furthermore, although most remit by age 18, studies suggest an earlier onset is associated with protracted reduction of symptoms (Nock et al., 2007). Finally, CD typically onsets later than ODD (Rowe et al., 2010) in middle childhood or in adolescence (i.e., child-onset vs. adolescent-onset) but generally increases in prevalence with age in the adolescent years (Biederman et al., 2008; Maughan et al., 2004). These studies and studies that included focus on both ADHD and DBPs have largely examined the extent to which youths continue to meet criteria for the disorders of interest and/ or what later impairments are predicted by the earlier diagnoses (Willoughby, 2003). These studies have yielded insights such as the increased risk for DBPs in ADHD youths, particularly those with HI symptoms (e.g., Beauchaine & McNulty, 2013; Rowe et al., 2010), and that childhood ADHD is associated with increased risk of later deleterious outcomes, especially for youths with ADHD and comorbid ODD/CD (Molina et al., 2009; Steinberg & Drabick, 2015).

However, fewer longitudinal studies have focused on symptom trajectories or developmental course (Willoughby, 2003) for ADHD or DBPs alone, and far fewer have considered the course of these symptoms together (e.g., Biederman et al., 2008; Brocki et al., 2007; Burns & Walsh, 2002; Giannotta & Rydell, 2016; Harvey et al., 2016; Jester et al., 2005; Vos et

concerned with the distinction between ADHD subtypes, their predictive utility, and the issue of longitudinal stability to make recommendations for the fifth edition of the *DSM*.

al., 2022). The studies that have done so have one or more limitations, including many of the methodological problems outlined above. For example, many studies have examined one or more disorders (as distinct, homogeneous entities) or groupings, which reifies the field's extant nosology a priori (e.g., Biederman et al., 2008; Brocki et al., 2007; Burns & Walsh, 2002; Harvey et al., 2016; Stringaris et al., 2010; Vos et al., 2022). Several studies that did examine the developmental course of ADHD and DBPs have not included the adolescent period (e.g., have instead studied ages 5–7 years, Brocki et al., 2007; ages 2–4 years, Brown et al., 2022; kindergarten– seventh grade, Burns & Walsh, 2002; kindergarten–fifth grade, Evans et al., 2020; ages 3–6 years, Harvey et al., 2016; 38–91 months, Stringaris et al., 2010), limiting insights about the persistence of ADHD and the development or desistence of DBPs at older ages. Relatedly, some studies have examined children according to wave of recruitment, with each cohort encompassing a range of ages, which limits developmentally sensitive conclusions (e.g., Biederman et al., 2008; Burns & Walsh, 2002). Finally, many large longitudinal studies have used latent class growth analysis (or similar models; e.g., Giannotta & Rydell, 2016; Jester et al., 2005; So et al., 2022; Vos et al., 2022), which often examines different latent trajectories of individual continuous constructs. Even multivariate latent class growth models typically examine the various latent trajectories of two or more separate, continuous scores of different constructs (e.g., ADHD and ODD total scores), sometimes with post hoc examinations of scores on other variables in these latent trajectory classes (e.g., odds or rates of certain outcomes or disorders among a given latent trajectory). Overall, extant longitudinal studies either have the limitations outlined in the previous section (i.e., *DSM* group-based recruitment or analysis that controls for shared variance or obscure understanding of how symptoms relate to one another) and/or lack the necessary sample to form developmentally sensitive insights about the course of externalizing psychopathology.

Accordingly, several questions regarding the longitudinal course of externalizing psychopathology remain, many of which may be better elucidated with attention to these methodological issues. For example, although longitudinal studies of ADHD have identified that certain individuals have symptoms that persist while others remit, questions remain regarding predictors of those that persist (Karalunas & Nigg, 2020) and whether individuals with remission of symptoms may continue to experience significant impairment (Barkley, 2016; Eme, 2017). Some studies have found a pathway from HI to ODD (Beauchaine & McNulty, 2013) and that the presence of ODD may predict greater ADHD symptom persistence (Biederman et al., 2011). However, this association is not fully understood, because approximately half of youths with ODD lack a prior ADHD diagnosis (i.e., not all youths with ADHD develop ODD, and not all youths with ODD had prior ADHD; Nock et al., 2007). In addition, several studies of young children have found associations between HI and ODD, but little is known about the relationship between these symptoms because HI normatively decreases in adolescence (Barkley, 2016; Harvey et al., 2016). Relatedly, some have postulated that the reason for persistent impairment despite symptomatic or diagnostic remittance is that fewer HI symptoms at older ages are nevertheless impairing because these behaviors become less normative with increasing age (Barkley, 2016), although this remains largely unexplored. Furthermore, questions remain regarding a persistently restricted inattentive type (Frick & Nigg, 2012) and if it is meaningfully distinct from

individuals who later present with IN in adolescence. In addition, some research has identified a potentially meaningful subset of individuals who experience a later (i.e., after the age specified by the *DSM*) onset of ADHD symptoms (Sonuga-Barke et al., 2023). Collectively, there have been calls for more research to consider what the key symptoms are at various points across development and whether it is the same individuals in these profiles over time (Karalunas & Nigg, 2020). Gaining a greater understanding of the developmental course of externalizing psychopathology will not only advance basic research, but also help progress early identification and intervention efforts.

A Longitudinal, Person-Centered Approach

In sum, there is a need for the use of methodological approaches that allow for examination of comorbidity's contribution to heterogeneity and vice versa. In addition, longitudinal studies are crucial for understanding which symptoms co-occur and when. An analytic approach that is both person-centered and longitudinal addresses both limitations, such as latent transition analysis (LTA). The only LTAs focused on the course of child psychopathology to date have focused on internalizing and externalizing psychopathology and largely found separate latent classes for these two dimensions (Blok et al., 2022; Göbel et al., 2022; Isdahl-Troye et al., 2022; McElroy et al., 2017; Van Zalk et al., 2020; Willner et al., 2016). One LTA of externalizing problems identified "well-adjusted," "hyperactive/oppositional," and "aggressive/rulebreaking" in youths ages 4, 8, and 12 and was focused on how these groups are related to adverse childhood experiences (Villodas et al., 2015). The only other studies focused on the developmental course between and within ADHD and DBPs are those discussed above with one or more limitations that this study seeks to address. The present study, to our knowledge, is the first LTA of individual externalizing symptoms (including ADHD, ODD, and CD) spanning middle childhood (i.e., age 9) through midadolescence (i.e., age 15). In addition, in the present study, we examined how latent classes and latent transition pathways relate to academic, social, familial, and leisure impairment for clinical validation.

Regarding hypotheses, it is expected that certain symptoms will form a class akin to the *DSM* nosology (e.g., an IN class), whereas other classes will reflect comorbidity and contain symptoms from multiple *DSM* disorders (e.g., a class characterized by both HI and ODD; Burns & Walsh, 2002; Wood et al., 2009), which will be more impaired. Regarding change over time, it is hypothesized that IN will be generally stable, HI will decrease, some youths will demonstrate persistence of ODD symptoms, some will transition out of ODD classes, and any CD symptoms that are present will become more prevalent in adolescence, although relatively less common overall (Biederman et al., 2008; Döpfner et al., 2015; Maughan et al., 2004).

Transparency and Openness

Preregistration

The present study was not preregistered.

Data, materials, code, and online resources

Code and syntax that was used for the present study's data management and analyses is included in the OSF registration, https://doi.org/10.17605/OSF.IO/2KB7N, and associated OSF project, https://osf.io/6d7wc/?view_only=d402530f7107490d9ba27a3f9c691e02. Furthermore, we have provided instructions on how to access materials that are publicly available (i.e., excluding proprietary materials) in the Measures section below. Finally, additional methods, tables, and figures are available in the Supplemental Material available online. For further information about data, code, or materials, contact J. N. Smith.

Reporting

We believe that our methods are rigorous, were conducted with integrity, and are presented transparently. In the present study, we used data from the Oregon ADHD-1000 data set (Nigg et al., 2023). Note that the specific information provided throughout the Method section reflects only the data relevant to the present study at the time the data were acquired and was based, in part, on direct personal communication with the study team at Oregon Health & Science University regarding the most recent information because data collection and processing were still ongoing at the time this study was conducted. For full information about the finalized, publicly available Oregon ADHD-1000 data set, see Nigg et al. (2023). We report how we determined the subsample used in the present study, the specific measures used in our secondary data analysis, and all manipulations to used variables.

Ethical approval

The protocol from which the present study derived its data and the secondary data analysis were each approved by an institutional review board and was carried out in accordance with the provisions of the World Medical Association Declaration of Helsinki.

Method

Procedures

The data described herein were obtained through multiple National Institutes of Mental Health grants, including 1R01-MH59105 (principal investigator [PI]: Nigg), R01MH099064 (PI: Nigg), 1R37MH59105 (PI: Nigg), 2R56MH086654 (PI: Nigg), R01MH115357 (PIs: Fair and Nigg), K99 MH091238 (PI, Fair); and R00 MH091238 (PI: Fair). Secondary data analysis was approved by the institutional review board at Florida International University (IRB-21–0520-AM02).

In brief, families were recruited from the community, and ADHD was deliberately oversampled. Symptoms of ODD and CD were free to vary. Families were recruited via mass mailings to all families with children in the target age range within a 50-mile radius of Oregon Health & Science University. A sample of 2,144 inquiries were screened by phone to establish initial eligibility and interest, and an on-site clinical evaluation was conducted for 1,450, after which, best-estimate research diagnoses and final eligibility were established by a team of two clinicians. At this time, 528 youths were excluded (for further detail, see Nigg et al., 2023 and the Supplemental Methods in the Supplemental Material). After the diagnostic screen, 104 eligible participants withdrew because of lack of further interest, and

31 excluded participants ultimately participated, either for substudy needs or for rapport (i.e., sibling of an included participant). Ultimately, the present study sample includes 849, a subset of which (n = 610) were followed up (i.e., planned attrition) in Waves 2 through 8. In addition to planned attrition, some annual waves were missed for some individuals. The data used in the present study were collected between 2009 and 2022.

Participants

The proportions of primary race endorsed in the total Wave 1 sample were 85.4% White/Middle Eastern, 6.7% Black, 5.1% Asian/East Indian, 1.8% American Indian/Alaska Native/Eskimo, 0.7% Native Hawaiian/Pacific Islander, and 0.4% declined to answer/not known. In addition, approximately 10% endorsed second and third additional racial identities. The sample was 6.1% Hispanic/Latino/a. Regarding biological sex, the Wave 1 sample was 61.8% male.

Regarding the highest education obtained by the participant's parents, 34.0% had a bachelor's degree; 26.4% had a master's, law, or other 2- to 3-year degree; 16.1% had some college education but no degree; 9.9% had a doctorate, PhD, or medical degree; 9.8% had an associate's degree; 2.5% had a high school degree or equivalent; and 0.1% had some high school education but no degree (1.2% of participants did not respond). Regarding income, 8.1% earned < $25,000, 5.9% earned < $35,000, 10.5% earned < $50,000, 20.1% earned < $75,000, 21.0% earned < $100,000, 14% earned < $130,000, 5.4% earned < $150,000, and 8.1% earned > $150,000 (6.8% declined to answer or did not know).

Measures

Kiddie Schedule for Affective Disorders and Schizophrenia. Individual ADHD, ODD, and CD items from the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS; Kaufman et al., 1997) parent interview were dichotomized (i.e., symptom endorsed = 1, symptom not endorsed = 0, such that ST and threshold responses count as endorsement) and used as latent class indicators. No "rule out" or "not otherwise specified" responses occurred in the present study's data. Symptoms were dichotomized because of the infrequency of subthreshold endorsements (≈0.5%–2% across most items), which limits the ability of an analytic model to determine thresholds and complicates interpretation (DiStefano et al., 2021). This measure is proprietary and cannot be shared by study authors.

Strengths and Difficulties Questionnaire impact items. Impairment in friendships, learning, leisure, and family settings were assessed using impact items of the Strengths and Difficulties Questionnaire (SDQ), which has satisfactory reliability (Goodman, 2001). Parents reported whether their child's difficulties impair everyday life in these domains from 0 (*not at all*) to 3 (*a great deal*). The SDQ is open access and can be found on sdqinfo.org. Specifically, navigate to English language (USA) questionnaires and select the version that includes the impact supplement for parents of 4- to 10-year-olds and parents of 11- to 17-year-olds.

Data management and data-analytic plan: The first aim of the present study was to conduct an LTA using individual symptoms of ADHD, ODD, and CD at ages 9, 12, and 15.

To further characterize these classes, the descriptive statistics for the specific items endorsed in each class at each age were examined. The second aim was to consider the extent to which latent classes at each age predict concurrent functional impairment. To examine SDQ scores and descriptive statistics, latent class and latent transition pathway membership assignments were hard-classified (for further detail, see Supplemental Methods in the Supplemental Material). Latent class assignments were dummy-coded, and one multiple linear regression model was conducted for each area of impairment (i.e., friendships, learning, leisure, and family) at each age. All data management was conducted in SPSS; the latent class analysis (LCA), LTA, and multiple regressions were conducted in Mplus Version 8.9 (Muthén & Muthén, 2017); and the hard-classified classes were exported back to SPSS, in which descriptive statistics by class (e.g., see Tables 1 and 2) were conducted. Finally, the Benjamini-Hochberg false discovery rate (FDR) procedure was conducted for multiplicity control (Benjamini & Hochberg, 1995) on SDQ regression analyses using a freely available online resource (Weinkauf, 2012). The procedure compares the smallest p value with a p value of FDR / m, where FDR is the desired FDR and m is the number of tests. This is the most stringent test. The next smallest p value is compared with a p value of 2 × FDR / m, the third-smallest p value is compared with a p value of 3 × FDR / m, and so on; testing stops when a test is not significant. We used a desired FDR of .05 and entered p values by age (i.e., each p-value column of Table 3, such that m = 16 for age 9, m = 12 for age 12, and m = 16 for age 15). The largest adjusted p values for which a finding survived comparison was .0344, .0292, and .0156 for ages 9, 12, and 15, respectively.

Before conducting analyses, all available data (i.e., including the baseline n = 849 and all follow-up data) were converted from a by-wave to a by-age format. Given that participants were ages 7 to 14 years at the first wave and ages 14 to 21 years by the last wave of data collection, examination of latent classes at various specific waves would have limited developmental utility. Ages 9 (n = 398), 12 (n = 265), and 15 (n = 179) years were chosen to balance developmental considerations, equal distance between chosen ages, and largest available sample sizes after restructuring data. To reformat data by age, a series of variables was created in SPSS using syntax to aid in restructuring. Specifically, variables were created to identify the wave at which participants were a given age in years, and then, data from that wave were copied into a new age-based variable for each age and variable of interest. For more information, see Supplemental Methods in the Supplemental Material. Before LCAs were conducted, KSADS items were dichotomized, and items with n < 5% endorsement were removed to prevent problems with model estimation (Collins & Lanza, 2009; DiStefano et al., 2021; Flora et al., 2012). A threshold of 5% was chosen to roughly approximate the prevalence of these symptoms in the general population (Maughan et al., 2004; Merikangas et al., 2009; Nock et al., 2006, 2007; Polanczyk et al., 2015). As a result, all 18 ADHD items were retained at all ages, seven of eight ODD items were retained at all ages (excluding only ODD No. 8: “spiteful/vindictive” at all ages), and only one out of 15 CD items was retained at ages 9 and 12 (CD No. 1: “bully”).[2]

[2]Authors did not assess measurement invariance in the LTA model. Whether or not to assess for measurement invariance is an issue of trade-off between parsimony, model fit, model identification, and interpretation (Collins & Lanza, 2009). We opted to allow classes to differ across ages to prioritize identification and interpretation of classes as they naturally occur at each age.

Results

Fit statistics and entropy of LCAs and LTAs

Considering Akaike information criterion (AIC), Bayesian information criterion (BIC), sample-size-adjusted BIC, entropy, likelihood ratio tests, theoretical rationale, parsimony, and minimum class sizes to avoid overfitting (> 5%), we found that the ideal class solutions at each age were identified as five classes at age 9 (entropy = 0.944), four classes at age 12 (entropy = 0.941), and five classes at age 15 (entropy = 0.976). For entropy and fit statistics, see Figure 1; for chosen class solutions, see Figure 2; and for all class solutions, see Figures S1 to S15 in the Supplemental Material. A corresponding LTA solution was ultimately chosen. The overall entropy for the LTA (0.709) was lower than the entropy for the individual LCAs because of a lower entropy for the estimated solution of the LTA at age 15 (age 9 = 0.800, age 12 = 0.740, age 15 = 0.518), which is likely due to substantial developmental shifts and perhaps a smaller sample size in adolescence that made it more difficult for the model to predict class membership longitudinally (see Figs. S16–S20 in the Supplemental Material). However, these developmental changes are of interest to the present study.

LCA solutions by age

At age 9, Class 1 (C1) consisted of typically developing (TD) individuals (n = 151, 37.9%). Class 2 (C2) consisted of youths likely to have symptoms of IN and HI (i.e., IN+HI; n = 64, 16.1%). Class 3 (C3) consisted of youths represented by similar IN and HI to C2 and ODD symptoms (i.e., IN+HI+ODD; n = 75, 18.8%). Although CD items were rarely above the threshold (5%), the CD item “bully” occurred only in the IN+HI+ODD class. Class 4 (C4) included youths likely to have symptoms of IN but not HI or ODD (i.e., n = 54, 13.6%). Finally, Class 5 (C5) consisted of youths with a moderate probability of IN and HI symptoms, especially items “fidgets/squirms,” “difficulty remaining in seat,” “talks excessively,” and “interrupts/intrudes” (i.e., ST; n = 54, 13.6%). TD and ST youths had higher IQ than IN+HI, IN+HI+ODD, and IN youths, and TD youths were proportionally more female than IN+HI, IN+HI+ODD, and ST youths. The omnibus test for parent education level was significant, but no post hoc comparisons emerged. Classes did not differ in race/ethnicity or combined family income. See Tables S1 and S2 in the Supplemental Material.

At age 12, C1 consisted of TD individuals (i.e., n = 88, 33.2%). C2 consisted of youths most likely to have symptoms of IN and with a moderate (mod) probability of HI symptoms (i.e., IN+modHI; n = 60, 22.6%). C3 included youths with similar IN and HI to C2 and ODD symptoms (i.e., IN+modHI+ODD; n = 38, 14.3%). Again, the CD item “bully” occurred only alongside this class. Finally, C4 consisted of individuals with a moderate likelihood of having IN symptoms and “interrupts/ intrudes” (i.e., an ST, predominantly IN class [ST/IN]; n = 79, 29.8%). TD youths had higher IQ than IN+modHI, IN+modHI+ODD, and ST/IN youths, and TD youths were more female than IN+modHI+ODD youths. Classes did not differ in parent education, income, or race/ ethnicity; see Tables S3 and S4 in the Supplemental Material.

At age 15, C1 consisted of TD individuals (i.e., $n = 74$, 41.3%). C2 consisted of youths most likely to have symptoms of IN and HI (i.e., $n = 20$, 11.2%). C3 included youths with high levels of IN, moderate HI, and ODD symptoms (i.e., $n = 19$, 10.6%). The CD item "bully" was not above the threshold for inclusion at age 15. C4 consisted of individuals who were predominantly IN (i.e., $n = 53$, 29.6%). Finally, C5 included individuals with moderate levels of HI and some ODD symptoms, most notably, moderate levels of "blames others" and "easy to annoy" (i.e., $n = 13$, 7.3%). TD youths had higher IQ than IN+HI youths. Classes did not differ in biological sex. Omnibus tests for parent education and combined family income were significant, but post hoc comparisons did not emerge. Classes did not differ in ethnicity but did differ in racial identity; see Tables S5 and S6 in the Supplemental Material.

Descriptive statistics of symptoms within latent classes

To further characterize the symptomatic profiles of these latent classes, descriptive statistics for individual symptoms endorsed by class at each age were explored. Only the most noteworthy statistics are provided herein for brevity; for complete information, see Table 1. IN+HI, IN+HI+ODD, and IN classes endorsed a similar number of IN symptoms at age 9 (IN+HI class: $M = 8.19$; IN+HI+ODD class: $M = 7.72$; IN class: $M = 7.44$). However, the IN class decreased to an average of $M = 4.57$ and $M = 5.79$ IN symptoms at ages 12 and 15, respectively, while the IN+HI class continued to have a similar number of IN symptoms at later ages (age 12: $M = 7.98$; age 15: $M = 8.35$). The IN+HI+ODD class decreased slightly in IN symptoms at age 15 ($M = 6.68$). Regarding HI, the IN+HI and IN+HI+ODD classes had similar means at age 9 (IN+HI class: $M = 7.30$; IN+HI+ODD: class $M = 6.91$) and age 12 (IN+HI class: $M = 5.28$; IN+HI+ODD class: $M = 5.66$); however, the IN+HI+ODD class decreased in HI levels at age 15 ($M = 3.32$) compared with the IN+HI class at age 15 ($M = 6.05$). Nevertheless, IN+HI+ODD and IN+HI classes had higher HI than TD ($M = 0.10$), IN ($M = 0.91$), and to a lesser extent, ST ($M = 2.92$) classes at age 15. The IN class had an average of 2.11, 1.53, and 0.91 HI symptoms at ages 9, 12, and 15, respectively.

Frequency of HI symptoms in latent classes

Finally, Table 2 shows the frequency of individual HI items by age in each class. Most notably, nearly 52% of the IN class at age 9 endorsed items "fidgets/squirms," "difficulty remaining in seat," and "runs/climbs." The remaining items were less common in IN youths (i.e., approximately 4%−28%) but were very common in IN+HI and IN+HI+ODD classes (i.e., approximately 69%−94%), including items "driven by motor," "talks excessively," "blurts answers," "difficulty waiting turn," and "interrupts/intrudes." In classes with marked HI (i.e., C2 and C3), the items "fidgets/squirms," "difficulty playing quietly," "talks excessively," "blurts answers," and "interrupts/intrudes" had relatively stable frequencies over time (i.e., less than 10% decrease from ages 9 to 15, or an increase), whereas the items "difficulty remaining in seat," "runs/climbs," "driven by motor," and "difficulty waiting turn" became less frequent over time. Finally, although the classes with ODD at all ages (i.e., IN+HI+ODD at age 9 and IN+modHI+ODD at age 12 and 15) had moderate to high HI, these classes decreased in frequency of HI items compared with IN+HI and IN+modHI, even for the items that remained more common with age.

Latent transition probabilities

The LTA solutions for each age are nearly identical to the separate LCA solutions at each individual age; for LTA solutions, see Figures S16 to S18 in the Supplemental Material. The latent transition probabilities between classes were estimated. From age 9 to age 12, 91.8% of TD individuals remained TD, and 8.2% became ST. Furthermore, 51.6% of IN+HI individuals at age 9 remained both IN and HI at age 12 (i.e., IN+modHI), whereas 48.4% transitioned to ST/IN at age 12. In addition, 62.6% of HI and ODD youths maintained these symptoms from age 9 to 12 (i.e., IN+HI+ODD to IN+modHI+ODD), whereas 26.0% transitioned to IN+modHI, and 11.3% transitioned to ST/IN. From age 9 to 12, 73.3% of IN youths transitioned to the most similar class at age 12 (i.e., to C4, the ST/IN class). Finally, 63.7% of ST individuals at age 9 transitioned to develop greater ADHD symptoms at age 12 (i.e., transitioned to IN+modHI), and 35.4% transitioned to ST/IN. From ages 12 to 15, 82.2% of TD individuals remained TD, and 13.0% transitioned to IN. From ages 12 to 15, 60.9% of IN+modHI individuals transitioned to a predominantly IN presentation, 20.4% transitioned to a class with less representation of IN symptoms but relatively greater ODD symptoms (i.e., transitioned to modHI+lowODD), and 10.8% remained purely ADHD (i.e., IN+HI). Conversely, for IN+modHI+ODD individuals, 61.3% maintained those symptoms, 14.5% became IN only, 14.4% maintained ODD symptoms but did not retain symptoms of IN (modHI+lowODD), and 9.8% desisted in ODD symptoms but retained ADHD symptoms (IN+HI) from 12 to 15. Finally, 37.2% of ST/IN individuals maintained and developed greater IN symptoms, 31.5% developed greater HI (i.e., IN+HI), 14.6% desisted (i.e., transitioned to TD), and 14.0% developed greater oppositionality (i.e., IN+modHI+ODD) from 12 to 15. See Figure 3.

Latent transition pathways

Out of 100 possible latent transition pathways (i.e., 5 × 4 × 5 classes at each age), only 31 contained any participants. For all LTA pathways, see Table S7 in the Supplemental Material. Only the eight LTA pathways with a frequency over 1% are discussed further and used in subsequent SDQ analyses (three-digit numbers indicate the class assignment at ages 9, 12, and 15, respectively, in the latent transition pathway, with each class as described above): 111 (i.e., “persistently TD”; $n = 227$, 26.7%), 114 (i.e., TD at ages 9 and 12 and IN at age 15, or “adolescent IN”; $n = 9$, 1.1%), 224 (i.e., IN+HI at ages 9 and 12 and IN only at 15, or “typical ADHD”; $n = 51$, 6.0%), 324 (i.e., all symptoms at age 9, IN+HI at 12, and IN only at age 15, or “desisters”; $n = 14$, 1.6%), 333 (i.e., a most severe group with persistence of all symptoms, or “persistently oppositional”; $n = 93$, 11.0%), 442 (i.e., a group that was IN in childhood but developed HI at age 15, or “worsening”; $n = 12$, 1.4%), 444 (i.e., a “persistently IN” group; $n = 83$, 9.8%), and 524 (i.e., a class that began ST, developed greater IN and HI at age 12, and desisted to IN only by 15, or a “mixed trajectory” class; $n = 66$, 7.8%).

Regressions for functional impairment predicted by latent class

Finally, dummy-coded latent classes and latent transition pathways were used in multiple linear regression to consider impairment in friendships, learning, leisure, and family using the SDQ impact items. Regarding friendships, only IN+HI+ODD at age 9 ($b = 0.708$, *SE*

= 0.158, p = .000) and IN+modHI+ODD at age 12 (b = 0.911, SE = 0.212, p = .000) were significantly impaired. Regarding learning, all classes at all ages except modHI+lowODD at age 15 were significantly impaired (see Table 3; all ps = .000). Regarding leisure, IN+HI at age 9, IN+modHI at age 12, IN+HI+ODD at age 9, and IN+modHI+ODD at age 12 were significantly impaired, especially classes with ODD (age 9 IN+HI: b = 0.461, SE = 0.145, p = .002; age 9 IN+HI+ODD: b = 0.679, SE = 0.142, p = .000; age 12 IN+modHI: b = 0.417, SE = 0.159, p = .009; age 12 IN+modHI+ODD: b = 0.746, SE = 0.184, p = .000). All classes were significantly impaired with family at age 9 (see Table 3; all ps < .001); at age 12, only IN+modHI+ODD youths were significantly impaired (b = 0.919, SE = 0.176, p = .000). At age 15, both IN+modHI+ODD and IN classes had significant familial impairment, although IN+modHI+ODD youths had the largest magnitude effect (IN+modHI+ODD: b = 0.889, SE = 0.278, p = .001; IN: b = 0.370, SE = 0.141, p = .009). See Table 3.

Discussion

First, the findings from our study support the presence of a meaningfully distinct restricted IN type from childhood through adolescence. In fact, the persistently IN path comprised a substantial portion of the total sample (≈10%). This persistently IN path is unique from the other paths with IN implicated in adolescence, such as the typical ADHD (224) path, the desisters (324) path, and the adolescent IN (114) path (which may resemble one pathway to late-onset ADHD, or youths that present with IN for another reason not captured by this study, or youths whose symptoms were not detected until demands exceeded abilities; Solanto, 2019). Individuals in the IN class decreased to approximately five IN symptoms at ages 12 and 15, which is below the threshold indicated by the *DSM* (using *DSM* criteria of six symptoms, these youths would be categorized as ST or control). This suggests that there are potentially some decreases in IN levels over time, even among groups who primarily present with clinically significant IN (i.e., our IN class was distinct from ST and TD classes and associated with impairment), consistent with other work (e.g., Frick & Nigg, 2012; Sibley et al., 2012, 2017). Clinically, this suggests that in addition to the well-established normative decreases in HI with age (Barkley, 2016; Campbell et al., 2014; Frick & Nigg, 2012), IN symptoms may also normatively decrease in adolescence for some ADHD youths. This may indicate that the symptoms of ADHD are not well captured by the *DSM* criteria at older ages—even for IN—and/or that fewer symptoms are necessary to meet criteria and experience meaningful functional impairment at older ages. Furthermore, our findings are consistent with that reviewed by Frick and Nigg (2012) such that our IN class had two HI symptoms at age 9 (i.e., Frick and Nigg discussed that youths with two or fewer HI symptoms are distinct from youths with three or more symptoms). Furthermore, our IN class had zero to one HI symptoms at ages 12 to 15, which may inform normative levels of HI symptoms at older ages for groups without significant HI presentations. Relatedly, it is noteworthy that certain HI items were better at distinguishing potentially normative HI in the IN class from the significant HI in the IN+HI and IN+HI+ODD classes at age 9 (i.e., items that were common for these classes and rare for the IN class; see Table 2).

Furthermore, our findings support that certain individuals continue to have significant HI in adolescence and also suggest that these classes with high levels of HI had greater and more stable IN. Although there were normative decreases in HI over time and many individuals

transitioned into the IN class at age 15, the individuals that remained in the IN+HI class still had threshold levels of HI symptoms (i.e., an average of approximately six) at age 15. The items that remained more prevalent at later ages were not strictly impulsivity items, as some literature would suggest (Cabral et al., 2020; Faraone et al., 2006; see Table 2). Furthermore, HI occurred only in the presence of high levels of IN at all ages, excluding C5 at age 15 (i.e., modHI+lowODD youths). Specifically, the IN+HI class at age 15 had an average of eight IN symptoms, compared with the IN class at age 15, which endorsed approximately six. This could be a product of measurement, that is, that parents may attribute inattentiveness to observable HI behavior, whereas inattention only may go undetected (Wolraich et al., 2005). Alternatively, it is possible that the presence of HI is related to greater severity overall (Smith et al., 2013; Willcutt et al., 2012). Nevertheless, findings replicate that the ADHD-HI presentation is rare and may lack clinical significance (Gibbins et al., 2010; Valo & Tannock, 2010).

Results of the present study also yield several insights about the co-occurrence of ADHD and ODD. First, ODD and HI largely co-occurred over time, with a slight divergence at age 15. Specifically, ODD symptoms occurred only in the presence of moderate to high levels of HI symptoms (i.e., never with IN only or independently). Furthermore, the presence of ODD was related to greater stability of HI. Specifically, although a minority (10%) of IN+HI youths at age 12 remained IN+HI at age 15 (i.e., most transitioned to IN), the *majority* (61%) of IN+modHI+ODD youths at age 12 continued into the IN+modHI+ODD class at age 15, and an additional 10% transitioned to IN+HI at age 15. Between these two paths, the 71% of youths in the IN+modHI+ODD class at age 12 continued to have some degree of HI symptoms at age 15, compared with only 10% of youths with HI and without ODD at age 12. However, there was a slight divergence at age 15 such that youths in C2 (IN+HI) had higher levels of HI than C3 (IN+modHI+ODD), even for the HI items that were more resilient to development (see Table 2). Nevertheless, the levels of HI in the IN+modHI+ODD class were still greater than other classes at age 15. Second, our study did not support the idea that earlier HI predicts later ODD (Beauchaine & McNulty, 2013; Harvey et al., 2016). Rather, it was more common that ODD occurred *with* HI in childhood, followed by either persistence of ODD (e.g., the persistently oppositional path comprised 11% of the sample) or desistence of ODD (e.g., as evidenced by the desisters path and consistent transitions out of classes with ODD over time). Collectively, the findings of this study suggest that HI and ODD symptoms may be very aligned in middle childhood and diverge somewhat in adolescence. Clinically, these findings suggest that children with ODD are likely to have more stable HI behavior than their peers without ODD and may require more long-standing interventions for these symptoms; however, they may desist in ODD. Conversely, children with ADHD alone are unlikely to develop later ODD in adolescence and are more likely to desist in HI compared with their peers with ODD. By comparison, these youths may require fewer supports for ADHD symptoms in adolescence.

Regarding impairment, findings broadly validate the clinical significance of Classes 2, 3, and 4 at all ages (i.e., all but TD and ST classes). Despite recent literature positing that less than threshold levels of HI in adolescence may continue to create functional impairment given the extent to which it is no longer normative (Barkley, 2016), these findings suggest that ODD is what contributes most greatly to friendship and familial impairment if it

occurs at older ages. All classes at age 9 were significantly impaired with family, perhaps attributable to greater familial involvement at younger ages. Furthermore, all classes at all ages except C5 at age 15 were significantly impaired in learning, likely attributable to the shared IN between these classes (Garner et al., 2013). The modHI+lowODD class at age 15 was not impaired in any domain; although this class is small, it may represent a nonclinical impulsive and oppositional group that is somewhat normative in adolescence. Clinically, findings emphasize the importance of full-threshold levels of ODD (≈3.0–4.5 symptoms) for contributing substantially to interpersonal impairment.

Several key insights are offered by our choice to examine individual items and avoid examination of groupings by diagnosis or ADHD presentation in the present study. While the use of thresholds for diagnosis and/or dichotomous categorization of ADHD presentations would have likely mischaracterized youths with fewer than six symptoms of IN or HI, the methodological approach of the present study allowed these youths to remain included in an otherwise relatively stable and clinically significant latent class at individual ages and/or in a longitudinal trajectory of related symptoms. For example, persistently IN youths had, on average, fewer than six symptoms of IN at ages 12 and 15 but were meaningfully grouped with youths who were best characterized by relatively elevated IN and demonstrated related functional impairment (see Tables 1 and 3). In addition, the IN+modHI+ODD classes at ages 12 and 15 had below-threshold HI symptoms but threshold-level ODD symptoms, on average. With use of diagnostic thresholds, various individual participants in this class may have been characterized as TD, IN only, or ODD only, which would have contributed to heterogeneity within and similarity across these groupings. Moreover, the detection of this IN+modHI+ODD latent class was critical to understanding longitudinal trajectories because these youths had greater persistence of HI symptoms over time than youths without ODD symptoms. An approach that controlled for or omitted ODD among ADHD youths may have detected only a decrease in HI overall with time (i.e., the distinction in persistence vs. desistence of HI between youths with and without ODD symptoms would have been diluted). This is an especially important insight because most studies on the relationship between HI and ODD are in samples of young children (Brocki et al., 2007; Brown et al., 2022; Evans et al., 2020; Harvey et al., 2016; Stringaris et al., 2010), and less is known about the relationship between these symptoms as HI decreases in adolescence.

There are various other benefits to the present study's unique methodological approach. The longitudinal nature of the present study allowed for consideration of which symptoms at what frequency are meaningful because information is gleaned about their trajectory—and with diagnostic categorization, youths may have had greater rates of transitioning in and out of groups over time, obscuring the true nature of their symptomatic pathway. Moreover, the examination of individual items provided richer information than diagnostic-retention studies. As one example, we were able to investigate that certain HI symptoms were better at distinguishing potentially normative HI in otherwise primarily IN youths from youths with significant HI in other classes (see Table 2). In addition, albeit with only the presence/absence of one symptom from each diagnostic category, it is possible that our methodological approach, which used individual symptoms as latent class indicators, allowed us to investigate the contribution of heterogeneity to comorbidity such that the one

CD item that was above the frequency threshold for inclusion (No. 8: “bully”) occurred only alongside ODD items and without other CD items and only at ages 9 and 12 when ODD is more prevalent (Biederman et al., 2008; Nock et al., 2007), whereas the one ODD item that was below the frequency threshold for inclusion (No. 8: “spiteful/vindictive”) may have been more related to the CD construct, which was largely absent in the present sample. Finally, our findings highlight the importance of including individuals with ST symptoms. For example, youths in the ST class at age 9 were more likely to develop later symptoms than TD youths (see Fig. 3).

No study is without limitations. Contrary to expectations, symptoms largely clustered according to the *DSM*, with some slight variation in likelihood of specific items within a set of related items (e.g., see Fig. 2). Ultimately, recruitment procedures and the characteristics of the sample drive the latent classes that are found. Although ODD/CD were free to vary and ST youths were included, ADHD youths were the primary recruitment target in the present study; in particular, CD symptoms were less prevalent than expected. Despite this, we were able to observe ODD’s co-occurrence with ADHD, specifically, only in the presence of HI, which likely adds to ADHD heterogeneity if ODD is not measured or is statistically controlled for in analyses. However, we did not have sufficient CD symptoms to meaningfully examine the construct; rather, the one CD item included at ages 9 and 12 (i.e., “bully”) appears especially related to ODD. Although CD may not have been meaningfully assessed in the present study, the item was retained to be consistent with the study’s motivation to avoid exclusion or statistical control according to *DSM* diagnosis. Future studies using larger, more symptomatically heterogeneous samples may yield more nuanced insights, including larger samples of ODD and CD (which may reveal more about ODD/CD comorbidity and heterogeneity) and other comorbidities commonly observed in ADHD (e.g., anxiety, autism, learning disorders). Relatedly, in the present study, we used a sample that is primarily White, non-Hispanic, and middle-class, which influences symptomatic trajectories found (including potentially lower rates of CD) and limits generalizability (Noordermeer et al., 2017; Sharp et al., 2021). Further still, in the present study, we did not examine the role of treatment participation on latent classes or longitudinal pathways. Another limitation of the present study is that the earliest age was middle childhood (i.e., age 9), and it is possible that we missed meaningful classes and transitions in early childhood. Specifically, it is possible that studies examining the preschool-age period may find relationships from earlier HI to later ODD. Furthermore, although the present study had excellent entropy in individual LCAs at all ages (all > .90), we did also have poorer entropy for age 15 in the LTA solution. This may be inevitable because of the longitudinal model’s inability to predict changes into adolescence using the class indicators provided and with the sample size available. However, LTA findings for age 15 should be interpreted with caution. Finally, the sample sizes of many LTA paths were too small to investigate how LTA paths predict outcomes, which would be a unique opportunity for larger data sets to explore. In addition, future research should examine the etiological mechanisms potentially shared and unique across these LCAs and LTA paths. Collectively, we believe that the present study’s analytic approach is theoretically promising, but was limited because of the very large and heterogeneous data sets required to answer such questions. Future studies with access to such samples should consider a similar analytic approach.

In the present study, we explored the potential utility of data-driven, person-centered, developmentally sensitive models, such as LTA, to better understand heterogeneity and comorbidity in externalizing psychopathology from childhood through adolescence. Findings revealed that for youths with persistence of HI, it was a mix of hyperactive and impulsive items that remained more prevalent into adolescence (i.e., not just impulsivity items). Findings also revealed that youths with ODD in childhood have greater HI persistence at older ages than youths without. However, youths who do persist in HI *without* ODD have greater HI symptoms than youths with ODD in adolescence, suggesting divergence of HI and ODD over time. Relatedly, ODD primarily occurred alongside HI in childhood rather than subsequent to earlier HI. The insights offered by this study emphasize the importance of using analytic methods consistent with theory surrounding the nature of heterogeneity and comorbidity, a lack of which obscures insights about the true developmental course of psychopathology. Thus, future studies should choose methods that do not rely on a priori *DSM* groupings and do not control for commonly comorbid symptoms in recruitment or in analyses, with the spirit of openly exploring which symptoms are most relevant at what points in development.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Thank you to Joel Nigg, Andy Pham, Elisa Trucco, Lauren McGrath, Eliza Kramer, and Tyler W. Mason for their support on this project.

Funding

Authors received grant support from the National Institute of Mental Health (J. N. Smith: F31 MH129054; E. D. Musser: K23 MH117280) and the National Institute of Child Health and Human Development (J. Parent: L40HD103048).

References

Althoff RR, Kuny-Slock AV, Verhulst FC, Hudziak JJ, & van der Ende J (2014). Classes of oppositionaldefiant behavior: Concurrent and predictive validity. Journal of Child Psychology and Psychiatry, 55(10), 1162–1171. [PubMed: 24673629]

Banaschewski T, Becker K, Döpfner M, Holtmann M, Rösler M, & Romanos M (2017). Attention-deficit/ hyperactivity disorder: A current overview. Deutsches Ärzteblatt International, 114(9), 149–159. 10.3238/arztebl.2017.0149 [PubMed: 28351467]

Barkley RA (2016). Recent longitudinal studies of childhood attention-deficit/hyperactivity disorder: Important themes and questions for further research. Journal of Abnormal Psychology, 125(2), 248–255. [PubMed: 26854509]

Beauchaine TP, Hinshaw SP, & Pang KL (2010). Comorbidity of attention-deficit/hyperactivity disorder and early-onset conduct disorder: Biological, environmental, and developmental mechanisms. Clinical Psychology: Science and Practice, 17(4), 327–336.

Beauchaine TP, & McNulty T (2013). Comorbidities and continuities as ontogenic processes: Toward a developmental spectrum model of externalizing psychopathology. Development and Psychopathology, 25(4, Pt. 2), 1505–1528. 10.1017/S0954579413000746 [PubMed: 24342853]

Benjamini Y, & Hochberg Y (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. Journal of the Royal Statistical Society Series B: Methodological, 57(1), 289–300.

Biederman J, Petty CR, Clarke A, Lomedico A, & Faraone SV (2011). Predictors of persistent ADHD: An 11-year follow-up study. Journal of Psychiatric Research, 45(2), 150–155. 10.1016/j.jpsychires.2010.06.009 [PubMed: 20656298]

Biederman J, Petty CR, Dolan C, Hughes S, Mick E, Monuteaux MC, & Faraone SV (2008). The long-term longitudinal course of oppositional defiant disorder and conduct disorder in ADHD boys: Findings from a controlled 10-year prospective longitudinal follow-up study. Psychological Medicine, 38(7), 1027–1036. [PubMed: 18205967]

Blok E, de Mol CL, van der Ende J, Hillegers MH, Althoff RR, Shaw P, & White T (2022). Stability and change of psychopathology symptoms throughout childhood and adolescence. Child Psychiatry & Human Development, 53(6), 1330–1339. 10.1007/s10578-021-01212-8 [PubMed: 34184159]

Bolhuis K, Lubke GH, van der Ende J, Bartels M, van Beijsterveldt CE, Lichtenstein P, Larsson H, Jaddoe VWV, Kushner SA, Verhulst FC, Boomsma DI, & Tiemeier H (2017). Disentangling heterogeneity of childhood disruptive behavior problems into dimensions and subgroups. Journal of the American Academy of Child & Adolescent Psychiatry, 56(8), 678–686. [PubMed: 28735697]

Brocki KC, Nyberg L, Thorell LB, & Bohlin G (2007). Early concurrent and longitudinal symptoms of ADHD and ODD: Relations to different types of inhibitory control and working memory. Journal of Child Psychology and Psychiatry, 48(10), 1033–1041. [PubMed: 17915004]

Brown HR, Laws HB, & Harvey EA (2022). Early development of ADHD and ODD symptoms from the toddler to preschool years. Journal of Attention Disorders, 26(10), 1335–1346. [PubMed: 34996308]

Burns GL, & Walsh JA (2002). The influence of ADHD– hyperactivity/impulsivity symptoms on the development of oppositional defiant disorder symptoms in a 2-year longitudinal study. Journal of Abnormal Child Psychology, 30(3), 245–256. [PubMed: 12041710]

Cabral MDI, Liu S, & Soares N (2020). Attentiondeficit/hyperactivity disorder: Diagnostic criteria, epidemiology, risk factors and evaluation in youth. Translational Pediatrics, 9(Suppl. 1), S104–S113. 10.21037/tp.2019.09.08 [PubMed: 32206588]

Campbell SB, Halperin JM, & Sonuga-Barke EJ (2014). A developmental perspective on attention-deficit/hyperactivity disorder (ADHD). In Lewis M & Rudolph KD (Eds.), Handbook of developmental psychopathology (pp. 427–448). Springer.

Caron C, & Rutter M (1991). Comorbidity in child psychopathology: Concepts, issues and research strategies. Journal of Child Psychology and Psychiatry, 32(7), 1063–1080. [PubMed: 1787137]

Collins LM, & Lanza ST (2009). Latent class and latent transition analysis: With applications in the social, behavioral, and health sciences (Vol. 718). John Wiley & Sons.

DiStefano C, Shi D, & Morgan GB (2021). Collapsing categories is often more advantageous than modeling sparse data: Investigations in the CFA framework. Structural Equation Modeling: A Multidisciplinary Journal, 28(2), 237–249.

Döpfner M, Hautmann C, Görtz-Dorten A, Klasen F, & Ravens-Sieberer U (2015). Long-term course of ADHD symptoms from childhood to early adulthood in a community sample. European Child & Adolescent Psychiatry, 24(6), 665–673. [PubMed: 25395380]

Eme R (2017). A review of the most recent longitudinal studies of ADHD. Journal of Memory Disorders and Rehabilitation, 2(1), Article 1004. 10.47739/2578-319X/1004

Evans SC, Cooley JL, Blossom JB, Pederson CA, Tampke EC, & Fite PJ (2020). Examining ODD/ADHD symptom dimensions as predictors of social, emotional, and academic trajectories in middle childhood. Journal of Clinical Child & Adolescent Psychology, 49(6), 912–929. [PubMed: 31454272]

Fair DA, Bathula D, Nikolas MA, & Nigg JT (2012). Distinct neuropsychological subgroups in typically developing youth inform heterogeneity in children with ADHD. Proceedings of the National Academy of Sciences, 109(17), 6769–6774.

Faraone SV, Biederman J, & Mick E (2006). The agedependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. Psychological Medicine, 36(2), 159–165. [PubMed: 16420712]

Feczko E, & Fair DA (2020). Methods and challenges for assessing heterogeneity. Biological Psychiatry, 88(1), 9–17. [PubMed: 32386742]

Feczko E, Miranda-Dominguez O, Marr M, Graham AM, Nigg JT, & Fair DA (2019). The heterogeneity problem: Approaches to identify psychiatric subtypes. Trends in Cognitive Sciences, 23(7), 584–601. [PubMed: 31153774]

Flora DB, LaBrish C, & Chalmers RP (2012). Old and new ideas for data screening and assumption testing for exploratory and confirmatory factor analysis. Frontiers in Psychology, 3, Article 55. 10.3389/fpsyg.2012.00055

Franke B, Michelini G, Asherson P, Banaschewski T, Bilbow A, Buitelaar JK, Cormand B, Faraone SV, Ginsberg Y, Haavik J, Kuntski J, Larsson H, Lesch K, Quiroga AR, Réthelyi JM, Ribases M, & Reif A (2018). Live fast, die young? A review on the developmental trajectories of ADHD across the lifespan. European Neuropsychopharmacology, 28(10), 1059–1088. [PubMed: 30195575]

Frick PJ (2016). Current research on conduct disorder in children and adolescents. South African Journal of Psychology, 46(2), 160–174.

Frick PJ, & Nigg JT (2012). Current issues in the diagnosis of attention deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder. Annual Review of Clinical Psychology, 8, 77–107.

Garner AA, O'Connor BC, Narad ME, Tamm L, Simon J, & Epstein JN (2013). The relationship between ADHD symptom dimensions, clinical correlates and functional impairments. Journal of Developmental and Behavioral Pediatrics, 34(7), 469–477. 10.1097/DBP.0b013e3182a39890 [PubMed: 24042078]

Giannotta F, & Rydell AM (2016). The prospective links between hyperactive/impulsive, inattentive, and oppositional-defiant behaviors in childhood and antisocial behavior in adolescence: The moderating influence of gender and the parent–child relationship quality. Child Psychiatry & Human Development, 47, 857–870. [PubMed: 26680210]

Gibbins C, Weiss MD, Goodman DW, Hodgkins PS, Landgraf JM, & Faraone SV (2010). ADHD-hyperactive/ impulsive subtype in adults. Mental Illness, 2(1), 41–45.

Göbel K, Ortelbach N, Cohrdes C, Baumgarten F, Meyrose AK, Ravens-Sieberer U, & Scheithauer H (2022). Co-occurrence, stability and manifestation of child and adolescent mental health problems: A latent transition analysis. BMC Psychology, 10(1), Article 267. 10.1186/s40359-022-00969-4

Goh PK, Lee CA, Martel MM, Karalunas SL, & Nigg JT (2020). Subgroups of childhood ADHD based on temperament traits and cognition: Concurrent and predictive validity. Journal of Abnormal Child Psychology, 48(10), 1251–1264. [PubMed: 32666315]

Goodman R (2001). Psychometric properties of the strengths and difficulties questionnaire. Journal of the American Academy of Child and Adolescent Psychiatry, 40, 1337–1345. [PubMed: 11699809]

Harvey EA, Breaux RP, & Lugo-Candelas CI (2016). Early development of comorbidity between symptoms of attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). Journal of Abnormal Psychology, 125(2), 154–167. 10.1037/abn0000090 [PubMed: 26854502]

Herzhoff K, & Tackett JL (2016). Subfactors of oppositional defiant disorder: Converging evidence from structural and latent class analyses. Journal of Child Psychology and Psychiatry, 57(1), 18–29. [PubMed: 25907358]

Isdahl-Troye A, Villar P, Domínguez-Álvarez B, Romero E, & Deater-Deckard K (2022). The development of co-occurrent anxiety and externalizing problems from early childhood: A latent transition analysis approach. Research on Child and Adolescent Psychopathology, 50(4), 505–519. 10.1007/s10802-021-00865-2 [PubMed: 34499292]

Jester JM, Nigg JT, Adams K, Fitzgerald HE, Puttler LI, Wong MM, & Zucker RA (2005). Inattention/ hyperactivity and aggression from early childhood to adolescence: Heterogeneity of trajectories and differential influence of family environment characteristics. Development and Psychopathology, 17(1), 99–125. [PubMed: 15971762]

Karalunas SL, & Nigg JT (2020). Heterogeneity and subtyping in attention-deficit/hyperactivity disorder— Considerations for emerging research using person- centered computational approaches. Biological Psychiatry, 88(1), 103–110. [PubMed: 31924323]

Kaufman J, Birmaher B, Brent D, Rao UMA, Flynn C, Moreci P, Williamson D, & Ryan N (1997). Schedule for affective disorders and schizophrenia for schoolage children-present and lifetime

version (K-SADS-PL): Initial reliability and validity data. Journal of the American Academy of Child & Adolescent Psychiatry, 36(7), 980–988. [PubMed: 9204677]

Lahey BB, Pelham WE, Loney J, Lee SS, & Willcutt E (2005). Instability of the DSM-IV subtypes of ADHD from preschool through elementary school. Archives of General Psychiatry, 62(8), 896–902. [PubMed: 16061767]

Lilienfeld SO, & Treadway MT (2016). Clashing diagnostic approaches: DSM-ICD versus RDoC. Annual Review of Clinical Psychology, 12, 435–463.

Luo Y, Weibman D, Halperin JM, & Li X (2019). A review of heterogeneity in attention deficit/hyperactivity disorder (ADHD). Frontiers in Human Neuroscience, 13, Article 42. 10.3389/fnhum.2019.00042

Martel MM (2016). Dispositional trait types of ADHD in young children. Journal of Attention Disorders, 20(1), 43–52. [PubMed: 23239785]

Martel MM, Goth-Owens T, Martinez-Torteya C, & Nigg JT (2010). A person-centered personality approach to heterogeneity in attention-deficit/hyperactivity disorder (ADHD). Journal of Abnormal Psychology, 119(1), 186–196. 10.1037/a0017511 [PubMed: 20141255]

Maughan B, Rowe R, Messer J, Goodman R, & Meltzer H (2004). Conduct disorder and oppositional defiant disorder in a national sample: Developmental epidemiology. Journal of Child Psychology and Psychiatry, 45(3), 609–621. [PubMed: 15055379]

McElroy E, Shevlin M, & Murphy J (2017). Internalizing and externalizing disorders in childhood and adolescence: A latent transition analysis using ALSPAC data. Comprehensive Psychiatry, 75, 75–84. [PubMed: 28334631]

Merikangas KR, Nakamura EF, & Kessler RC (2009). Epidemiology of mental disorders in children and adolescents. Dialogues in Clinical Neuroscience, 11(1), 7–20. [PubMed: 19432384]

Molina BS, Hinshaw SP, Swanson JM, Arnold LE, Vitiello B, Jensen PS, Epstein JN, Hoza B, Hechtman L, Abikoff HB, Elliott GR, Greenhill LL, Newcorn JH, Wells KC, Wigal T, Gibbons RD, Hur K, & Houck PR, & MTA Cooperative Group. (2009). The MTA at 8 years: Prospective follow-up of children treated for combined-type ADHD in a multisite study. Journal of the American Academy of Child & Adolescent Psychiatry, 48(5), 484–500. [PubMed: 19318991]

Muthén LK, & Muthén BO (2017). Mplus user's guide (8th ed.).

Nigg JT, Sibley MH, Thapar A, & Karalunas SL (2020). Development of ADHD: Etiology, heterogeneity, and early life course. Annual Review of Developmental Psychology, 2(1), 559–583. 10.1146/annurev-devpsych-060320-093413

Nigg JT, Karalunas SL, Mooney MA, Wilmot B, Nikolas MA, Martel MM, Tipsord J, Nousen EK, Schmitt C, Ryabinin P, Musser ED, Nagel BJ, & Fair DA (2023). The Oregon ADHD-1000: A new longitudinal data resource enriched for clinical cases and multiple levels of analysis. Developmental Cognitive Neuroscience, 60, Article 101222. 10.1016/j.dcn.2023.101222

Nock MK, Kazdin AE, Hiripi E, & Kessler RC (2006). Prevalence, subtypes, and correlates of DSM-IV conduct disorder in the National Comorbidity Survey Replication. Psychological Medicine, 36(5), 699–710. [PubMed: 16438742]

Nock MK, Kazdin AE, Hiripi E, & Kessler RC (2007). Lifetime prevalence, correlates, and persistence of oppositional defiant disorder: Results from the National Comorbidity Survey Replication. Journal of Child Psychology and Psychiatry, 48(7), 703–713. [PubMed: 17593151]

Noordermeer SD, Luman M, Weeda WD, Buitelaar JK, Richards JS, Hartman CA, Hoekstra PJ, Franke B, Heslenfeld DJ, & Oosterlaan J (2017). Risk factors for comorbid oppositional defiant disorder in attention-deficit/hyperactivity disorder. European Child & Adolescent Psychiatry, 26, 1155–1164. [PubMed: 28283834]

Polanczyk GV, Salum GA, Sugaya LS, Caye A, & Rohde LA (2015). Annual research review: A metaanalysis of the worldwide prevalence of mental disorders in children and adolescents. Journal of Child Psychology and Psychiatry, 56(3), 345–365. [PubMed: 25649325]

Rajendran K, O'Neill S, Marks DJ, & Halperin JM (2015). Latent profile analysis of neuropsychological measures to determine preschoolers' risk for ADHD. Journal of Child Psychology and Psychiatry, 56(9), 958–965. [PubMed: 26053870]

Reale L, Bartoli B, Cartabia M, Zanetti M, Costantino MA, Canevini MP, Termine C, & Bonati M (2017). Comorbidity prevalence and treatment outcome in children and adolescents with ADHD. European Child & Adolescent Psychiatry, 26, 1443–1457. [PubMed: 28527021]

Rowe R, Costello EJ, Angold A, Copeland WE, & Maughan B (2010). Developmental pathways in oppositional defiant disorder and conduct disorder. Journal of Abnormal Psychology, 119(4), 726–738. 10.1037/a0020798 [PubMed: 21090876]

Sharp W, Mangalmurti A, Hall C, Choudhury S, & Shaw P (2021). Associations between neighborhood, family factors and symptom change in childhood attention deficit hyperactivity disorder. Social Science & Medicine, 271, Article 112203. 10.1016/j.socscimed.2019.02.054

Sibley MH, Pelham WE Jr., Molina BS, Gnagy EM, Waschbusch DA, Garefino AC, Kuriyan AB, Babinski DE, & Karch KM (2012). Diagnosing ADHD in adolescence. Journal of Consulting and Clinical Psychology, 80(1), 139–150. 10.1037/a0026577 [PubMed: 22148878]

Sibley MH, Swanson JM, Arnold LE, Hechtman LT, Owens EB, Stehli A, Abikoff H, Hinshaw SP, Molina BSG, Mitchell JT, Jensen PS, Howard AL, Lakes KD, & Pelham WE (2017). Defining ADHD symptom persistence in adulthood: Optimizing sensitivity and specificity. Journal of Child Psychology and Psychiatry, 58(6), 655–662. [PubMed: 27642116]

Smith LC, Tamm L, Hughes CW, & Bernstein IH (2013). Separate and overlapping relationships of inattention and hyperactivity/impulsivity in children and adolescents with attention-deficit/hyperactivity disorder. ADHD Attention Deficit and Hyperactivity Disorders, 5, 9–20. [PubMed: 22996914]

So FK, Chavira D, & Lee SS (2022). ADHD and ODD dimensions: Time varying prediction of internalizing problems from childhood to adolescence. Journal of Attention Disorders, 26(6), 932–941. [PubMed: 34632828]

Solanto MV (2019). The prevalence of “late-onset” ADHD in a clinically referred adult sample. Journal of Attention Disorders, 23(9), 1026–1034. [PubMed: 29629842]

Sonuga-Barke EJ, Becker SP, Bölte S, Castellanos FX, Franke B, Newcorn JH, Nigg JT, Rohde LA, & Simonoff E (2023). Annual research review: Perspectives on progress in ADHD science–From characterization to cause. Journal of Child Psychology and Psychiatry, 64(4), 506–532. [PubMed: 36220605]

Steinberg EA, & Drabick DA (2015). A developmental psychopathology perspective on ADHD and comorbid conditions: The role of emotion regulation. Child Psychiatry & Human Development, 46(6), 951–966. [PubMed: 25662998]

Stringaris A, & Goodman R (2009). Longitudinal outcome of youth oppositionality: Irritable, headstrong, and hurtful behaviors have distinctive predictions. Journal of the American Academy of Child & Adolescent Psychiatry, 48(4), 404–412. [PubMed: 19318881]

Stringaris A, Maughan B, & Goodman R (2010). What’s in a disruptive disorder? Temperamental antecedents of oppositional defiant disorder: Findings from the Avon longitudinal study. Journal of the American Academy of Child & Adolescent Psychiatry, 49(5), 474–483. [PubMed: 20431467]

Szentiványi D, & Balázs J (2018). Quality of life in children and adolescents with symptoms or diagnosis of conduct disorder or oppositional defiant disorder. Mental Health & Prevention, 10, 1–8.

Valo S, & Tannock R (2010). Diagnostic instability of DSM–IV ADHD subtypes: Effects of informant source, instrumentation, and methods for combining symptom reports. Journal of Clinical Child & Adolescent Psychology, 39(6), 749–760. [PubMed: 21058123]

Van Zalk N, Tillfors M, & Mörtberg E (2020). Social anxiety-impulsivity subgroups and links to later emotional adjustment in adolescence: A latent transition analysis. The Journal of Early Adolescence, 40(9), 1397–1426.

Villodas MT, Litrownik AJ, Thompson R, Jones D, Roesch SC, Hussey JM, Block S, English DJ, & Dubowitz H (2015). Developmental transitions in presentations of externalizing problems among boys and girls at risk for child maltreatment. Development and Psychopathology, 27(1), 205–219. [PubMed: 25045912]

Vos M, Rommelse NN, Franke B, Oosterlaan J, Heslenfeld DJ, Hoekstra PJ, Klein M, Faraone SV, Buitelaar K, & Hartman CA (2022). Characterizing the heterogeneous course of inattention

and hyperactivity-impulsivity from childhood to young adulthood. European Child & Adolescent Psychiatry, 31(8), 1–11. 10.1007/s00787-021-01764-z

Weinkauf M (2012). BenjaminiHochberg.xlsx [Excel sheet]. MARUM – Center for Marine Environmental Sciences. https://www.marum.de/Binaries/Binary2518/BenjaminiHochberg.xlsx

Whelan YM, Stringaris A, Maughan B, & Barker ED (2013). Developmental continuity of oppositional defiant disorder subdimensions at ages 8, 10, and 13 years and their distinct psychiatric outcomes at age 16 years. Journal of the American Academy of Child & Adolescent Psychiatry, 52(9), 961–969. [PubMed: 23972698]

Willcutt EG, Nigg JT, Pennington BF, Solanto MV, Rohde LA, Tannock R, Loo SK, Carlson CL, McBurnett K, & Lahey BB (2012). Validity of DSM-IV attention deficit/hyperactivity disorder symptom dimensions and subtypes. Journal of Abnormal Psychology, 121(4), 991–1010. 10.1037/a0027347 [PubMed: 22612200]

Willner CJ, Gatzke-Kopp LM, & Bray BC (2016). The dynamics of internalizing and externalizing comorbidity across the early school years. Development and Psychopathology, 28(4, Pt. 1), 1033–1052. [PubMed: 27739391]

Willoughby MT (2003). Developmental course of ADHD symptomatology during the transition from childhood to adolescence: A review with recommendations. Journal of Child Psychology and Psychiatry, 44(1), 88–106. [PubMed: 12553414]

Wolraich ML, Wibbelsman CJ, Brown TE, Evans SW, Gotlieb EM, Knight JR, Clarke Ross E, Shubiner HH, Wender EH, & Wilens T (2005). Attention-deficit/ hyperactivity disorder among adolescents: A review of the diagnosis, treatment, and clinical implications. Pediatrics, 115(6), 1734–1746. [PubMed: 15930238]

Wood AC, Rijsdijk F, Asherson P, & Kuntsi J (2009). Hyperactive-impulsive symptom scores and oppositional behaviours reflect alternate manifestations of a single liability. Behavior Genetics, 39(5), 447–460. [PubMed: 19633943]

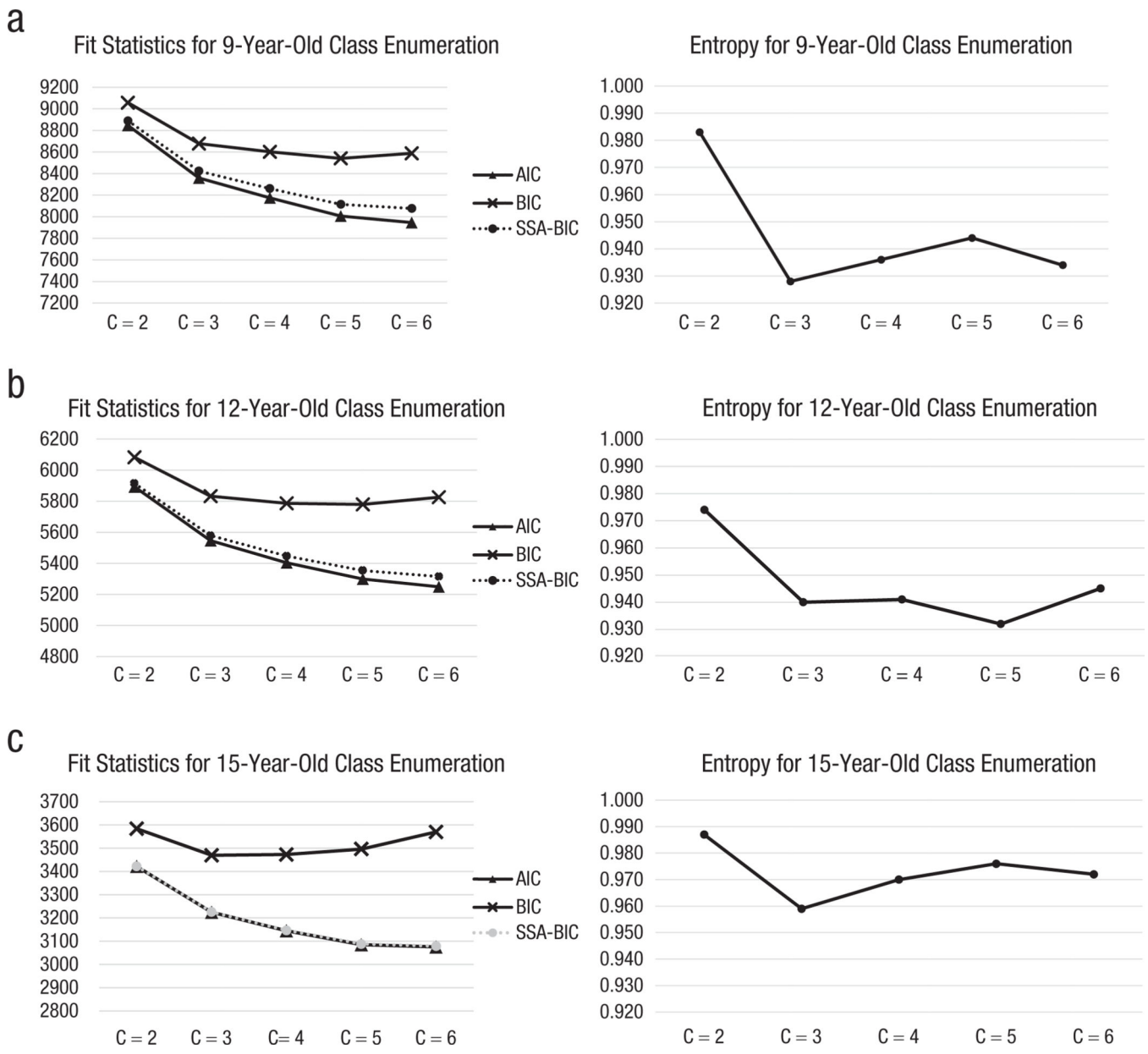

Fig. 1.

Fit statistics and entropy for latent class enumeration (Classes 2–6) at ages 9, 12, and 15 (Rows A, B, and C, respectively). C = class; AIC = Akaike information criterion; BIC = Bayesian information criterion; SSA-BIC = sample-size adjusted BIC.

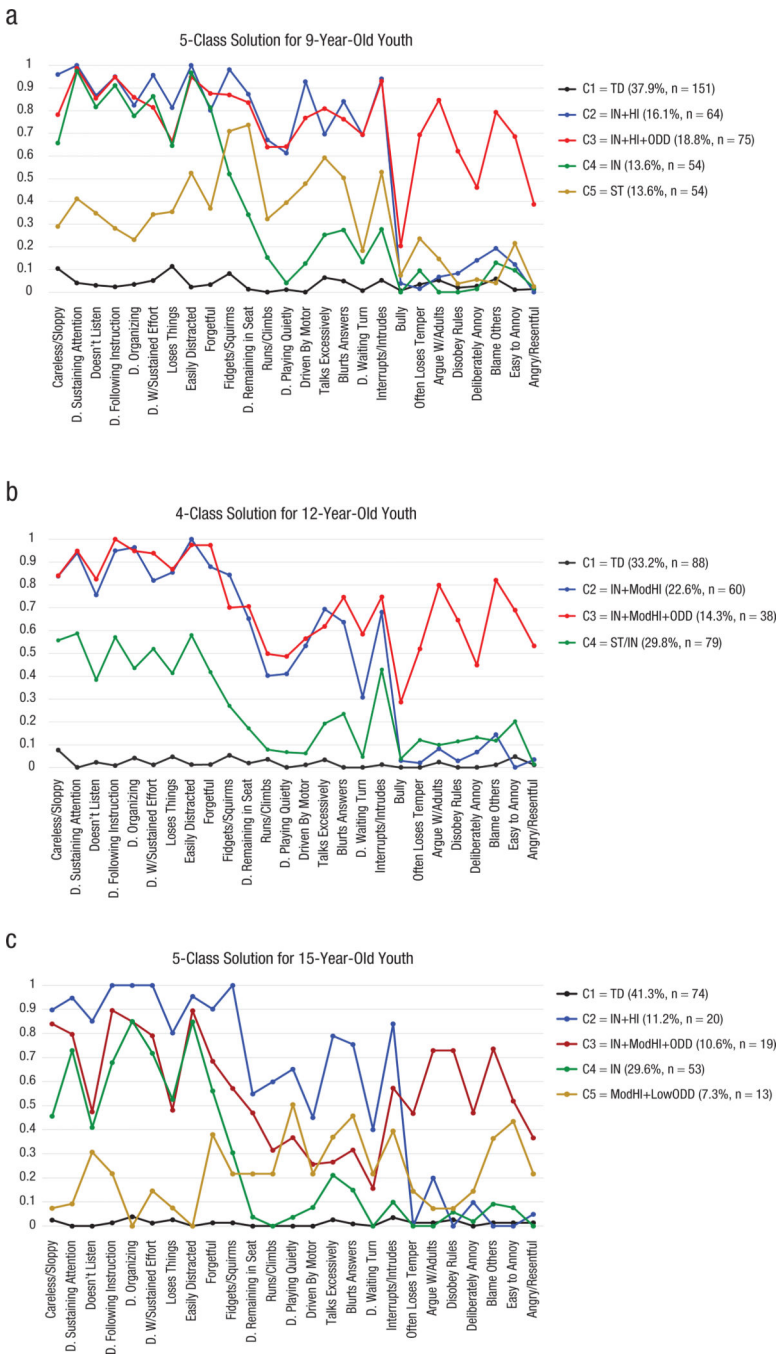

Fig. 2.

Final latent-class-analysis solutions at ages 9, 12, and 15 (Rows A, B, and C, respectively). C = class; D = difficulty.

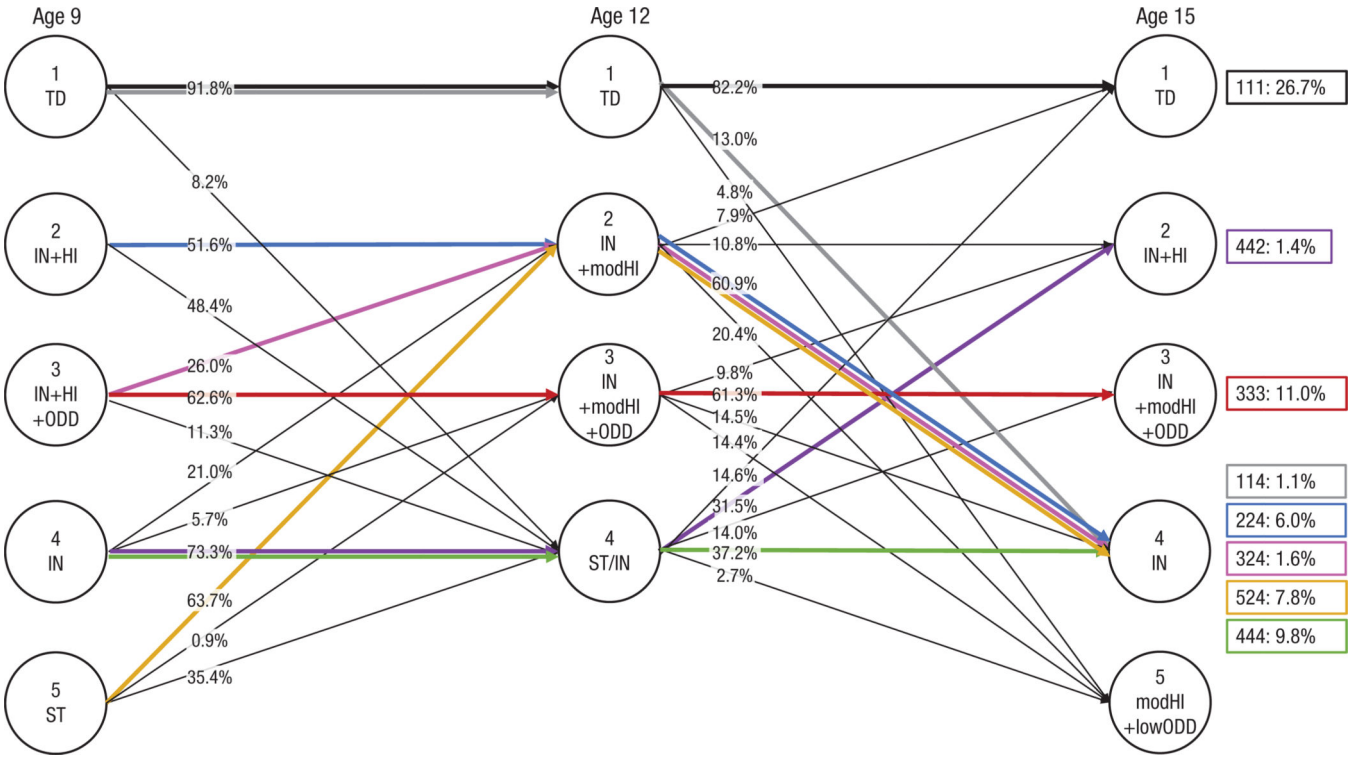

Fig. 3.

Diagram of all latent transition probabilities between latent classes at ages 9, 12, and 15 and most common latent transition analysis paths. Numbers within each circle correspond to latent class (e.g., “1” = class 1) at the age indicated at the top of the column above it. Percentages displayed on lines between the columns for each age represent the percentage of individuals from within the prior (i.e., left) latent class who transitioned to the indicated (i.e., right) latent class. Percentages indicated on the right side of the figure within rectangles indicate the percentage of the total sample in a given latent transition path from age 9 through age 15, as depicted by the pair of lines in the indicated color and the corresponding numbers. Three-digit numbers indicate the class assignments at ages 9, 12, and 15 in the latent transition pathway. 111 = persistently TD; 114 = adolescent IN; 224 = typical ADHD; 324 = desisters; 333 = persistently oppositional; 442 = worsening; 444 = persistently IN; 524 = mixed trajectory; TD = typically developing; IN = inattention; ADHD = attention-deficit/hyperactivity disorder; HI = hyperactivity/impulsivity; ST = subthreshold; mod = moderate.

Table 1.

Descriptive Statistics for Number of Inattention, Hyperactivity/Impulsivity, and Oppositional Defiant Disorder Symptoms Endorsed by Latent Class at Ages 9, 12, and 15

	Age 9		Age12		Age 15	
	***M* (*SD*)**	**Minimum–maximum**	***M* (*SD*)**	**Minimum–maximum**	***M* (*SD*)**	**Minimum–maximum**
			Inattention symptoms endorsed			
C1	0.463 (0.922)	0–4	0.250 (0.611)	0–2	0.135 (0.338)	0–2
C2	8.188 (1.052)	6–9	7.983 (1.269)	4–9	8.350 (0.813)	7–9
C3	7.720 (1.429)	2–9	8.351 (1.033)	4–9	6.684 (1.701)	4–9
C4	7.444 (1.423)	4–9	4.570 (2.505)	0–9	5.793 (1.822)	3–9
C5	3.111 (1.787)	0–7	—	—	1.308 (1.437)	0–4
			Hyperactivity/impulsivity symptoms endorsed			
C1	0.265 (0.550)	0–2	0.171 (0.530)	0–3	0.095 (0.376)	0–2
C2	7.297 (1.305)	5–9	5.283 (2.187)	1–9	6.050 (2.012)	3–9
C3	6.905 (1.753)	2–9	5.658 (2.724)	0–9	3.316 (2.311)	0–8
C4	2.111 (1.313)	0–5	1.532 (1.475)	0–6	0.906 (1.114)	0–3
C5	4.519 (1.979)	1–9	—	—	2.923 (2.498)	0–7
			Oppositional defiant disorder symptoms endorsed			
C1	0.219 (0.598)	0–4	0.091 (0.360)	0–2	0.250 (0.670)	0–3
C2	0.641 (0.861)	0–3	0.383 (0.666)	0–2	0.625 (0.916)	0–2
C3	4.581 (1.508)	2–7	4.553 (1.224)	2–7	3.000 (2.449)	0–7
C4	0.352 (0.756)	0–3	0.810 (1.311)	0–6	0.821 (1.492)	0–5
C5	0.741 (1.169)	0–6	—	—	0.250 (0.500)	0–1

Note: Conduct disorder is omitted in this table because of limited variability (i.e., only one symptom was endorsed at ages 9 and 12; thus, the mean number of symptoms and range would be between 0 and 1). The latent class solution at age 12 included only four classes; thus, no data for the fifth class placeholder (i.e., for ages 9 and 15) are shown. Age 9: C1 = TD, C2 = IN+HI, C3 = IN+HI+ODD, C4 = IN, C5 = (ST). Age 12: C1 = TD, C2 = IN+modHI, C3 = IN+modHI+ODD, C4 = ST/IN. Age 15: C1 = TD, C2 = IN+HI, C3 = IN+modHI+ODD, C4 = IN; C5 = modHI+lowODD. C = class; IN = inattention; HI = hyperactivity/impulsivity; ODD = oppositional defiant disorder; TD = typically developing; ST = subthreshold; mod = moderate.

Table 2.

Frequency of Item Endorsement for All Hyperactivity/Impulsivity Symptoms by Latent Class at Ages 9, 12, and 15

	Age 9					Age 12				Age 15				
Item	**C1**	**C2**	**C3**	**C4**	**C5**	**C1**	**C2**	**C3**	**C4**	**C1**	**C2**	**C3**	**C4**	**C5**
1. Fidgets/squirms	7.9	**98.4**	86.7	51.9	72.2	5.7	**86.7**	71.1	25.3	1.4	**100**	57.9	30.2	23.1
2. Difficulty remaining in seat	7.9	98.4	86.7	51.9	72.2	2.3	66.7	71.1	16.5	0.0	55.0	47.4	3.8	23.1
3. Runs/climbs	7.9	98.4	86.7	51.9	72.2	3.4	41.7	50.0	7.6	0.0	60.0	31.6	0.0	23.1
4. Difficulty playing quietly	0.7	**62.5**	64.0	3.7	40.7	0.0	**41.7**	50.0	6.3	0.0	**65.0**	36.8	3.8	53.8
5. Driven by motor	0.0	93.8	76.0	13.0	48.1	1.1	53.3	57.9	6.3	0.0	45.0	26.3	7.5	23.1
6. Talks excessively	6.6	**68.8**	81.3	25.9	59.3	3.4	**71.7**	60.5	19.0	2.7	**80.0**	26.3	20.8	38.5
7. Blurts answers	4.6	**84.4**	75.7	27.8	51.9	0.0	**65.0**	73.7	24.1	1.4	**75.0**	31.6	15.1	16.2
8. Difficulty waiting turn	0.7	71.9	69.3	13.0	16.7	0.0	31.7	57.9	5.1	0.0	40.0	15.8	0.0	23.1
9. Interrupts/intrudes	5.3	**93.8**	93.3	27.8	53.7	1.1	**70.0**	73.7	43.0	4.1	**85.0**	57.9	9.4	38.5

Note: Values shown are percentages of participants who endorsed the indicated item. Highlighted columns indicate the latent classes with notable HI, C2 and C3. Bold text indicates the items that had relative stability over time compared with other items. Item descriptions are paraphrased for brevity from the Kiddie Schedule for Affective Disorders and Schizophrenia. Age 9: C1 = TD, C2 = IN+HI, C3 = IN+HI+ODD, C4 = IN, C5 = ST. Age 12: C1 = TD, C2 = IN+modHI, C3 = IN+modHI+ODD, C4 = ST/IN. Age 15: C1 = TD, C2 = IN+HI, C3 = IN+modHI+ODD, C4 = IN, C5 = modHI+lowODD. C = class; IN = inattention; HI = hyperactivity/impulsivity; ODD = oppositional defiant disorder; TD = typically developing; ST = subthreshold; mod = moderate.

Table 3.

Strengths and Difficulties Questionnaire Impact Items for Friendship, Learning, Leisure, and Family Predicted by Dummy-Coded Latent Class (Compared With Class 1, Typically Developing Youths) at Ages 9, 12, and 15

	9-year-olds			12-year-olds			15-year-olds		
	b	***SE***	***p***	***b***	***SE***	***p***	***b***	***SE***	***p***
Friendships									
Class 2	0.334	0.167	.045[a]	0.368	0.190	.052	0.400	0.265	.131
Class 3	**0.708**	**0.158**	**.000**	**0.911**	**0.212**	**.000**	0.489	0.247	.048[a]
Class 4	0.088	0.174	.612	0.385	0.182	.034[a]	0.187	0.220	.395
Class 5	−0.116	0.172	.499	—	—	—	−0.233	0.322	.468
Learning									
Class 2	**1.658**	**0.144**	**.000**	**1.541**	**0.165**	**.000**	**1.400**	**0.235**	**.000**
Class 3	**1.341**	**0.156**	**.000**	**1.798**	**0.172**	**.000**	**1.411**	**0.275**	**.000**
Class 4	**1.289**	**0.148**	**.000**	**1.168**	**0.181**	**.000**	**1.126**	**0.192**	**.000**
Class 5	**0.753**	**0.170**	**.000**	—	—	—	0.300	0.345	.385
Leisure									
Class 2	**0.461**	**0.145**	**.002**	**0.417**	**0.159**	**.009**	0.150	0.191	.432
Class 3	**0.679**	**0.142**	**.000**	**0.746**	**0.184**	**.000**	0.372	0.211	.078
Class 4	0.208	0.152	.172	0.215	0.157	.171	−0.154	0.169	.361
Class 5	0.048	0.147	.744	—	—	—	−0.072	0.246	.769
Family									
Class 2	**0.901**	**0.150**	**.000**	0.269	0.155	.083	0.250	0.233	.284
Class 3	**1.167**	**0.144**	**.000**	**0.919**	**0.176**	**.000**	**0.889**	**0.278**	**.001**
Class 4	**0.723**	**0.163**	**.000**	0.247	0.139	.076	**0.370**	**0.141**	**.009**
Class 5	**0.567**	**0.150**	**.000**	—	—	—	−0.222	0.232	.338

Note: Strengths and Difficulties Questionnaire items are scored as *not at all* = 0, *only a little* = 1, *quite a lot* = 2, *a great deal* = 3. All regression paths within an age were conducted simultaneously in a structural equation model; all ages are shown together for readability. Statistically significant results ($p < .05$) are shown in bold. Age 9: Class 2 = IN+HI, Class 3 = IN+HI+ODD, Class 4 = IN, Class 5 = ST. Age 12: Class 2 = IN+modHI, Class 3 = IN+modHI+ODD, Class 4 = ST/IN. Age 15: Class 2 = IN+HI, Class 3 = IN+modHI+ODD, Class 4 = IN, Class 5 = modHI+lowODD. C = class; IN = inattention; HI = hyperactivity/impulsivity; ODD = oppositional defiant disorder; TD = typically developing; ST = subthreshold; mod = moderate.

[a]Findings were not statistically significant after Benjamini-Hochberg false discovery rate adjustment (and thus are not in bold).