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ADHD-INTERNALIZING DISORDER CO-OCCURRENCE IN CHILDHOOD AND ADOLESCENCE: COMPARING NETWORK AND LATENT VARIABLE CONCEPTUALIZATIONS

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
College of Arts and Sciences
at the University of Kentucky

By
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2021

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ABSTRACT OF DISSERTATION

ADHD-INTERNALIZING DISORDER CO-OCCURRENCE IN CHILDHOOD AND ADOLESCENCE: COMPARING NETWORK AND LATENT VARIABLE CONCEPTUALIZATIONS

Co-occurrence of attention-deficit/hyperactivity disorder (ADHD) with depression or anxiety (i.e., internalizing disorders) is a major route to poor outcomes, with temperament traits presenting as potential shared risk markers that underlie these disorders' development and characterization. Prior work investigating the nature of ADHD-internalizing disorder co-occurrence using structural equation modeling has provided support for both temperament-based common cause (i.e., effortful control and negative affect as liabilities for multiple disorders) and direct causation (i.e., ADHD directly contributing to risk for internalizing disorders) effects separately. Using a network approach, the current study represented the first attempt to integrate these effects into one model while parsing heterogeneity in the trait-symptom and symptom-symptom relations within them. Participants were 799 children and adolescents aged 7-13 years at baseline (61.20% boys, 85.11% White; 59.57% diagnosed with ADHD). Across two measurement points approximately five years apart (i.e., Year 1, Year 6), parents/caregivers provided ratings of participants' ADHD symptoms and temperament traits and participants provided ratings of depressive and anxiety symptoms. Pertaining to ADHD-depression networks, results suggested effortful control and, particularly, negative affect as transdiagnostic risk markers via relations with symptoms of both disorders. Simultaneously, depressive symptoms associated with reductions in perceived self-competence and difficulty making friends were uniquely related to several ADHD symptoms in Year 1, and ADHD inattentive symptoms (i.e., *loses things*; *does not follow through*; *has difficulty sustaining attention*) were uniquely related to depressive symptoms associated with reductions in perceived self-competence, distress/hopelessness, low self-worth, and difficulty making friends in Year 6. Examination of ADHD-anxiety networks suggested limited heterogeneity in symptom-symptom relations, although negative affect emerged as a core transdiagnostic risk marker via relations with inattentive and hyperactive/impulsive ADHD symptoms and anxiety symptoms associated with somatic problems and peer-related fears. Comparison

of network findings with those of structural equation modeling approaches to conceptualizing common cause and direct causation effects suggested consistent and complementary results. No differences were identified in the structure of networks across Years 1 and 6, as well as gender. Continued clarification of specific and unique common cause and direct causation effects in the context of one another may help identify those most influential to the development and characterization of ADHD-internalizing disorder co-occurrence, with a focus on such effects potentially highlighting targets for screening tools and interventions that address and account for symptoms of multiple disorders.

KEYWORDS: Attention-Deficit/Hyperactivity Disorder, Depression, Anxiety, Temperament, Network Analysis, Integrative Framework

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ADHD-INTERNALIZING DISORDER CO-OCCURRENCE IN CHILDHOOD AND ADOLESCENCE: COMPARING NETWORK AND LATENT VARIABLE CONCEPTUALIZATIONS

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DEDICATION

For my family.

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CHAPTER 1. INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a chronic neurodevelopmental disorder characterized by developmentally inappropriate and impairing symptoms of inattention and hyperactivity/impulsivity (American Psychiatric Association, 2013). Estimated to occur in up to 6% of children and adolescents worldwide (Faraone et al., 2021; Polanczyk et al., 2007), ADHD has been associated with high public health costs, impairment in several functional domains (e.g., academic failure, social difficulties), increased discord in the home and risk for drug use, and early death to suicide or accident (Goh, Martel, et al., 2020; Libutzki et al., 2019; Nigg, 2017; Wehmeier et al., 2010). One significant reason for these poor outcomes, in part, may be that youth with ADHD often develop co-occurring disorders (Gnanavel et al., 2019), with much of the prior research in this area focusing on ADHD's overlap with externalizing disorders (Mash & Barkley, 2014).

Conversely, the nature of ADHD's overlap with internalizing disorders (i.e., depression and anxiety), has remained relatively understudied. This remains a critical gap in the research literature, as evidence supporting elevated prevalence of internalizing disorders in those with ADHD is now fairly substantial. Past epidemiological work has suggested youth with ADHD develop depressive disorders at up to a five-fold rate, as well as anxiety disorders at up to a three-fold rate, compared to typically-developing youth (Angold et al., 1999). Additionally, studies have suggested that 20-30% and 13-51% of children and adolescents with ADHD may also develop depressive or anxiety disorders, respectively (Mash & Barkley, 2014; Mitchison & Njardvik, 2019; Mohammadi et al., 2021; Reale et al., 2017; Tsang et al., 2015), with risk for these

disorders beginning in childhood and then rising sharply after puberty particularly in girls (Gnanavel et al., 2019; Kessler et al., 2005; Thapar et al., 2012).

Co-occurrence of depression in those with ADHD has been associated with greater functional impairment, longer and more severe depressive episodes, and higher rates of suicidality and hospitalizations than either disorder in isolation (Biederman et al., 2008; Chronis-Tuscano et al., 2010; Daviss, 2008; Reid et al., 2015). Similarly, though there is some evidence that anxiety may reduce impulsivity in those with ADHD, co-occurrence of ADHD and anxiety during childhood and adolescence has been associated with differential response to ADHD-focused interventions, higher severity of inattentive symptoms, negative affect, and social difficulties, decreased self-esteem, and some increased cognitive difficulties (i.e., greater attention and working memory issues but improved response inhibition compared to those with ADHD only; Maric et al., 2018; Melegari et al., 2018; Pliszka, 2000; Schatz & Rostain, 2006; Tannock, 2009; van der Meer et al., 2018). Such seemingly additive effects of ADHD and internalizing disorders on subsequent impairment and quality of life highlight the importance of understanding reasons for these disorders' co-occurrence, particularly in the transition periods of middle-to-late childhood and adolescence when risk is highest. Yet, though some research has been conducted in this area, additional exploration is needed, with the development of an integrative model incorporating multiple explanations simultaneously potentially serving as a meaningful step forward.

1.1 Heterogeneity in ADHD and Internalizing Disorders' Presentations

1.1.1 Age-Based Heterogeneity

Complicating efforts to conceptualize ADHD-internalizing disorder co-occurrence is the fact that these disorders have been characterized by significant heterogeneity throughout childhood and adolescence. Pertaining to ADHD, research has suggested that hyperactive/impulsive symptoms may be most prominent in childhood and decline throughout development while inattentive symptoms increase in prominence and persist into young adulthood (Franke et al., 2018; Leopold et al., 2016; Willcutt et al., 2012). Age-based heterogeneity has also been identified in the expression of internalizing disorders: similar to inattentive symptoms, some limited work has suggested that depression during childhood may be characterized most by feelings of helplessness and loneliness, with difficulties with self-esteem, sadness, suicidal ideation, decreased concentration, and sleep problems becoming more prominent throughout development (Fu-I & Wang, 2008; Sørensen et al., 2005). Analogously, prior studies have suggested that anxiety disorders during childhood may be more characterized by separation anxiety and some phobia, with social phobia, panic disorder, agoraphobia, and generalized anxiety disorder having their core periods in adolescence or afterward (Beesdo et al., 2009; Lijster et al., 2017).

1.1.2 Gender-Based Heterogeneity[1]

Gender differences have also been suggested as contributing to heterogeneity in ADHD phenotypes during childhood and adolescence, with boys at least twice as likely

[1] Participants were categorized as boys or girls during data collection, so these two groups were retained for the current study. However, prior work has suggested that youth identifying as transgender and/or gender non-conforming may exhibit higher levels of ADHD and internalizing disorders than their cisgender peers

to be diagnosed with ADHD (American Psychiatric Association, 2013; Skogli et al., 2013). This discrepancy in prevalence has been suggested to be attributable to the fact that girls typically present with greater inattentive symptoms and fewer hyperactive/impulsive symptoms compared to boys, with this phenotype being more difficult to diagnose (although hyperactive/impulsive symptom severity may decrease in boys with age; Franke et al., 2018; Mowlem et al., 2019). Interestingly, it has also been suggested that girls with ADHD may develop and utilize better coping strategies than boys to compensate for ADHD-related difficulties, such as working hard to maintain classroom performance, which may also contribute to gender-based heterogeneity in the expression of ADHD and lower rates of diagnosis in girls (Quinn & Madhoo, 2014).

Pertaining to depression, results of some studies have indicated that symptoms of depression in girls may peak earlier and at higher levels of severity compared to boys (13.7 years in girls and 16.4 years in boys; Kwong et al., 2019; Salk et al., 2017). Additionally, studies have suggested gender-based heterogeneity in the expression of individual symptoms, with girls endorsing higher levels of guilt, body image dissatisfaction, self-blame, self-disappointment, feelings of failure, concentration problems, difficulty working, sadness/depressed mood, sleep problems, fatigue, and health worries and boys exhibiting higher levels of anhedonia and irritability (Bennett et al., 2005; Rucklidge, 2010; Zahn-Waxler et al., 2008). Gender differences have also been identified with respect to anxiety disorders, with girls at higher risk of experiencing anxiety problems although findings have been more mixed compared to depression (Kessler et al., 2012; Ohannessian et al., 2017). One idea is that these differences may

(Connolly et al., 2016; Veale et al., 2017), so future studies should include youth across the gender identity spectrum to facilitate more a comprehensive understanding of co-occurrence phenotypes.

stem from higher levels of rumination in girls compared to boys, although additional work is needed comparing anxiety-based phenotypes (Nolen-Hoeksema & Girgus, 1994). Overall, ADHD and internalizing disorders have both been characterized by significant gender- and age-based heterogeneity, with continued clarification of this heterogeneity needed particularly in the context of these disorders' co-occurrence.

1.2 Current Conceptualizations of Co-Occurrence

Perhaps the most prominent hypothesis for ADHD's overlap with depression and anxiety has been a "common cause" model suggesting that covariation between these disorders results due to a group of transdiagnostic liabilities that contribute to the characterization of multiple disorders (Smith et al., 2020). This model has typically been evaluated by specifying symptoms of disorders as loading onto one or a few latent variable entities (e.g., a superordinate "p" factor), which are then correlated with hypothesized shared risk markers using structural equation modeling (SEM) techniques (Caspi et al., 2014). Pertaining to ADHD's overlap with depression and anxiety, previously explored common causes have included genetics, early life stressors and parental factors, emotion dysregulation, weak executive function, deficits in attentional control, and intrusive and task-irrelevant thoughts (Brooker et al., 2020; Chronis-Tuscano et al., 2010; Cole et al., 2009; Fenesy & Lee, 2019; Humphreys et al., 2013; Jarrett et al., 2016; Jarrett, 2016; Meinzer et al., 2014; Ostrander & Herman, 2006; Pennington & Ozonoff, 1996).

Separately, "direct causation" has been offered as an explanation for ADHD's co-occurrence with depression and anxiety, with ADHD directly contributing to increased

risk for the development of these co-occurring internalizing disorders.[2] Pertaining to depression, one idea is that cumulative effects of inattentive and, perhaps secondarily, hyperactive/impulsive symptoms in social and scholastic domains fuels the development of depression, with recent studies using path analysis (a specific application of SEM) and related techniques accommodating causal relations (e.g., Mendelian randomization) to provide support for this idea (Riglin et al., 2020; Stern et al., 2020; Ward et al., 2019). Similarly, longitudinal studies in children and adolescents using similar statistical methods have provided some evidence for ADHD as a risk factor for the development of an anxiety disorder via peer rejection, academic failure, sporting failure, parenting practices, and decreased self-esteem (D'Agati et al., 2019; Tai et al., 2013). Interestingly, there has also been evidence suggesting a reciprocal relationship between ADHD and anxiety (Murray et al., 2020), with one idea being that primarily inattentive symptoms contribute to intrusive worry and hypervigilance which, in turn, alter the expression of ADHD by reducing impulsivity while increasing inattention via decreases in attentional control.

1.3 Temperament Traits as Common Causes

It was noted above that that prior common cause approaches have examined multiple different possible candidates. However, one under-utilized logic is that ADHD and internalizing disorders may share roots in temperment (i.e., individual differences in

[2] A growing body of work has also proposed a group of problems characterized by "sluggish cognitive tempo" that are distinct from yet overlap with inattentive symptoms of ADHD and contribute to internalizing disorders, particularly depression (Penny et al., 2009; Schatz & Rostain, 2006; Ward et al., 2019). However, no measures of sluggish cognitive tempo were administered for the current study, so additional work is needed to determine its relevance.

reactivity and self-regulation; Rothbart & Bates, 2006). Temperament in early life acts as a liability for multiple conditions including ADHD, with recent studies supporting temperament as a risk factor (Forbes et al., 2017; Nigg, 2017; Rutter & Arnett, 2020) rather than an analog (i.e., continuum hypothesis) or exacerbator of psychopathology (see Martel et al., 2014).

Although various temperament taxonomies have been proposed, three broad higher order traits have consistently been identified and are thought to be moderately stable across childhood and adolescence (Kopala-Sibley et al., 2018; Rothbart, 2011). *Effortful control* connotes the ability to suppress a prepotent or dominant response and purposefully resist interference to achieve a goal. It is closely related to the concept of executive functioning and hence of obvious relevance to ADHD liability, particularly that of the inattentive symptom domain (Martel, 2009; Nigg, 2017), but also to affect regulation. *Negative affect* connotes a predisposition to frequently experience negative emotions like sadness, fear, and anger, of likely relevance to internalizing disorders and to the emotional dysregulation associated with ADHD's hyperactive/impulsive symptom domain (De Pauw & Mervielde, 2010; Forbes et al., 2017; Nigg, 2006). *Surgency* is characterized by high activity levels, high-intensity pleasure seeking, low shyness, and impulsivity; it is also associated with positive affect such as excitement and exuberance. It thus connotes associations with ADHD that are seen developmentally (Miller et al., 2019) as well as inverse relations with depression and anxiety (Oldehinkel et al., 2004).

Many children with ADHD have difficulties with negative affect or negative emotional reactivity (Goh, Lee, et al., 2020; Karalunas et al., 2019; Smith & Martel, 2019). Developmentally, these difficulties may disrupt the consolidation of effortful

control, which, in turn, serve as a liability for additional deficits in self-regulation commonly associated with ADHD (Gagne & Goldsmith, 2011; Miller et al., 2019; Nigg et al., 2020). Further, low effortful control and high negative affect have been associated with internalizing symptoms in children with ADHD, with one idea being that low effortful control may contribute to decreased resilience to high negative affect and increase vulnerability to internalizing problems while also increasing one's perceived ineffectiveness due to an inability to mitigate or regulate distress (Muris et al., 2007; Rutter & Arnett, 2020).

1.4 Limitations of Past Conceptualizations

Though parsimonious and statistically supported, common cause and direct causation models examined through SEM approaches have precluded an examination of unique relations among symptoms of commonly co-occurring disorders and their risk markers (i.e., symptoms of ADHD and internalizing disorders being differentially related to one another, as well as various shared risk markers) as these relations have largely been conflated within latent or composite entities (Cramer et al., 2010). In some cases, the ability to capture shared variance across indicators presents as a strength, as it provides a means to reduce measurement error and test relations between latent constructs as conceptualized by overlapping information in multiple indicators. Yet, a parsing of unique relations between individual symptoms of different disorders, as well as exploration of whether such relations differ based on gender or age range, may be critical for identifying relations that are key to characterizing the ADHD-internalizing disorder relationship (e.g., ADHD-related difficulties sustaining attention may demonstrate a particularly strong relation with depression-related decreases in perceived

self-competence), as well as elucidating clinically relevant relations that are masked when conflating symptoms within composite factors (e.g., negative affect may be uniquely related to hyperactive/impulsive and depressive symptoms only after partialing out the role of inattention). Recent studies have provided support for these ideas: ADHD symptoms have been shown to exhibit distinct and heterogeneous relations with clinically relevant external correlates (e.g., impairment domains; Goh, Martel, et al., 2020; Martel et al., 2020). Similarly, prior work on depression and anxiety has suggested that individual symptoms may differ in their unique relations with etiological correlates, impairment domains, and common comorbidities (Beard et al., 2016; Fried, 2017).

Another limitation is the fact that past studies investigating co-occurring internalizing disorders in those with ADHD have generally explored direct causation and common cause effects separately (see Meinzer et al., 2014), leading to a striking need to examine the two conceptualizations in the context of one another to isolate their unique contributions to co-occurrence. Such exploration may be critical for determining the relevance of these effects when conceptualizing ADHD-internalizing disorder co-occurrence (i.e., do direct causation effects persist once accounting for common cause effects, and vice versa?), as well as informing the development of treatment tools aimed at the strongest unique relations to potentially facilitate additive benefits. Yet, such an integration has been relatively inaccessible through an SEM framework, as assumptions underlying these models have generally necessitated that they be examined in isolation. Specifically, when common causes have been included in SEM frameworks, covariation among symptoms of co-occurring disorders has been thought to result from the common cause, with an examination of direct relations among these symptoms being generally

inaccessible (Cramer et al., 2010). Alternatively, studies examining direct causation effects have sometimes included common causes as mediators (e.g., Humphreys et al., 2013), with directional hypotheses inherent in mediation contrasting with theory positing shared risk markers that contribute to the characterization of multiple disorders.

1.5 Integrating Models using Network Analysis

One way to move toward integration while also parsing heterogeneity in relations among symptoms of ADHD and internalizing disorders is to use a network framework (Borsboom & Cramer, 2013; Cramer et al., 2010). This proposed reformulation represents a potentially transformative approach to understanding psychopathology, as it assumes symptoms as active components of psychological disorders that demonstrate direct, dynamic, and potentially reciprocal relationships both with one another and various risk markers (Borsboom & Cramer, 2013). As an extension, co-occurrence between disorders is also radically reconceptualized through the network approach as resulting from a series of associations between symptoms of different disorders and their shared risk markers, rather than solely resulting from *either* a general liability or a correlation between disorder composites. In other words, the conceptual frame of the network approach suggests a process where co-occurrence may occur due to direct, heterogeneous, and unique relations between risk markers and symptoms of different disorders (i.e., common causes), *as well as* direct relations between symptoms themselves (i.e., direct causation).

Importantly, network models make use of partial correlations, thus allowing for a quantifying of the most robust relations among elements after controlling for others in a network. Additionally, network analysis provides a means to statistically explore whether

these relations may differ depending on age range or gender and thus potentially contribute to differences in ADHD-internalizing disorder co-occurrence phenotypes (i.e., "Network Comparison Test"). Hence, the network approach may provide a means to clarify unique trait-symptom and symptom-symptom relations underlying common cause and direct causation effects simultaneously, thus facilitating insights that are generally consistent with *and* complementary to those gleaned from SEM approaches (See Figure 1.1; Bringmann & Eronen, 2018).

1.5.1 Bridge Symptoms Linking ADHD, Internalizing Disorders, and Traits

Given the idea that symptoms demonstrate differential relations with risk markers and symptoms of other disorders, it is possible that a subset of ADHD symptoms may be particularly likely to be accompanied by increasingly severe levels of internalizing disorders, and vice versa. Similarly, it is also possible that specific symptoms of ADHD and internalizing disorders may be robustly related to certain temperament traits (and vice versa). Network theory accommodates the first statistical exploration of these "bridge elements" that may be key to conceptualizing a disorder's relation with another construct (i.e., another disorder or risk marker), as well as the unique relations through which bridge symptoms' importance may derive (Jones et al., 2019). For instance, past studies have suggested the inattentive symptom domain to be more robustly associated with depression and anxiety than the hyperactive/impulsive symptom domain (Bowen et al., 2008; Fenesy & Lee, 2019). Network analysis techniques could readily accommodate a more specific identification of relations between ADHD bridge symptoms associated with difficulties concentrating and staying organized with internalizing disorder symptoms associated with reductions in perceived competence. Similarly, although

effortful control and negative affect may exhibit some relation with all symptoms of ADHD and internalizing disorders (as reflected by SEM approaches), effortful control's relations with ADHD and internalizing disorders may be most accurately characterized via relations with ADHD inattentive bridge symptoms, whereas negative affect may be more associated with hyperactive/impulsive and internalizing disorder bridge symptoms associated with restlessness and associated distress. Ultimately, exploration of bridge symptoms and risk markers' roles in ADHD-internalizing disorder co-occurrence, as well as potential variation in bridge elements based on different factors (e.g., age range; gender), could further highlight specific relations through which co-occurrence may best be characterized and suggest a constellation of the most efficient indicators that clinicians could use to efficiently assess and intervene on risk for co-occurring internalizing disorders in youth with ADHD.

1.6 The Current Study

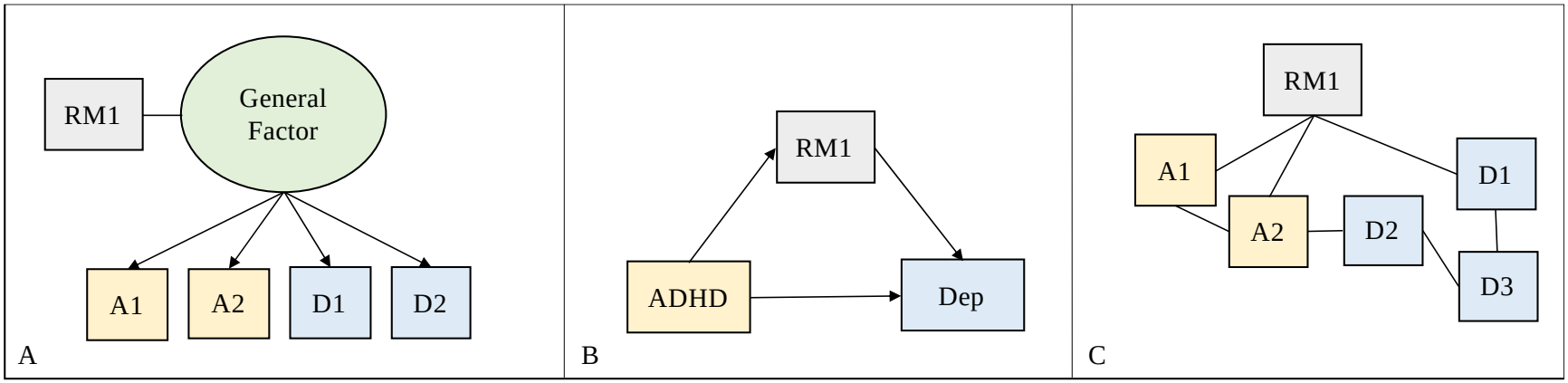
By allowing for a simultaneous investigation of common cause and direct causation effects, while also parsing the strongest unique relations within these effects, the network approach demonstrates potential for extending upon the findings of past SEM conceptualizations of ADHD-internalizing disorder co-occurrence during childhood and adolescence. Yet, such potential has yet to be empirically explored. The current study thus sought to use the network approach to identify key trait-symptom and symptom-symptom relations underlying ADHD-internalizing disorder co-occurrence, and then assessed whether such findings were consistent with and complementary to those obtained from SEM approaches to co-occurrence. Analyses were conducted using data from a longitudinal sample of youth across two measurement points approximately five

years apart to facilitate an exploration of co-occurrence in childhood and adolescence, with ADHD-depression and ADHD-anxiety co-occurrence examined separately.

First, two sets of bridge elements in networks were identified at each measurement point: those pertaining to disorders' relations with temperament traits, and those pertaining to ADHD-internalizing disorder relations. Pertaining to trait-disorder relations, it was hypothesized that effortful control and negative affect would both demonstrate relations with symptoms of ADHD and internalizing disorders that were relatively consistent in strength overall, with low levels of effortful control being primarily related to increased severity of ADHD inattentive bridge symptoms and increases in negative affect being primarily related to increased severity of hyperactive/impulsive and internalizing disorder bridge symptoms. Pertaining to ADHD-internalizing disorder relations, it was hypothesized that ADHD bridge symptoms associated with difficulties concentrating and staying organized would also demonstrate direct and positive relations with internalizing disorder bridge symptoms associated with decreased perceived self-competence and self-esteem. Second, network findings, as pertaining to the unique relations between disorders and traits, were qualitatively compared to those obtained when common cause and direct causation effects were conceptualized separately via SEM. It was hypothesized that, across measurement points, results of the two approaches would broadly be consistent (i.e., inattention related to internalizing disorders, traits related to both disorders). However, it was also hypothesized that the network approach would facilitate insights pertaining to the strongest relations among traits and symptoms that complemented those obtained from SEM models.

Finally, relations between symptoms and traits within ADHD-depression and ADHD-anxiety networks were compared between childhood versus adolescence, as well as between girls versus boys. It was hypothesized that ADHD-internalizing disorder relations would be significantly stronger in adolescence than childhood, though ADHD bridge symptoms would primarily be from the hyperactive/impulsive symptom domain during childhood and the inattentive symptom domain during adolescence. Further, it was hypothesized that ADHD symptoms in girls would be more strongly related, overall, to symptoms of depression and anxiety compared to boys, with ADHD bridge symptoms falling within the inattentive symptom domain in girls and the hyperactive/impulsive symptom domain in boys.

Figure 1.1 Simplified Depictions of Alternative Models of Co-Occurrence



Note. Model A depicts a latent variable approach to the Common Cause Model, with the circle representing a latent general factor underlying symptoms of ADHD (i.e., A1, A2) and depression (i.e., D1, D2). This factor is then correlated with a theorized risk marker (i.e., RM1). Model B depicts a composite variable approach to the Direct Causation Model, with an ADHD composite score specified as predicting a depression composite score. Inattentive and hyperactive/impulsive symptom domain scores are sometimes separated, latent factors are sometimes used instead of composite scores to reduce measurement error, and risk markers are sometimes included as mediators. Model C depicts co-occurrence conceptualized through the network approach, where symptoms of ADHD and depression (i.e., A2, D2) demonstrate unique relations with one another. Under this approach, co-occurrence occurs when the "activation" of specific symptoms of one disorder are directly associated with the activation of those of another. Further, the network approach accommodates an exploration of a risk marker's unique relations with various symptoms of co-occurring disorders without directional restrictions.

CHAPTER 2. METHODS

2.1 Participants

Participants were drawn from the Oregon ADHD-1000, a well-characterized child cohort for which the community-based recruitment and enrollment procedures have been published in detail elsewhere (Karalunas et al., 2017; Musser et al., 2016). Data are reported for 799 participants at the first ("Year 1") and 377 participants at the sixth ("Year 6") measurement points of that longitudinal data set to facilitate an examination of middle-to-late childhood and adolescent periods of development, respectively (Year 1: *M* = 9.65 years, 61.7% boys, 84.6% White, mean age difference between Years 1 and 6 = 5.09 years, SD = 0.17). Preliminary assessment of pubertal stage generally supported a distinction between measurement points, with 93% of participants' parents/caregivers indicating prepubertal to early pubertal stage in Year 1 and 73% indicating mid- to post-pubertal in Year 6. Ethics approval was obtained from the Institutional Review Board at Oregon Health & Science University. A parent/legal guardian provided written informed consent and children provided written assent. Among eligible children with ADHD (Year 1: n = 476; Year 6: n = 123), 39% reported prescribed stimulant medications in Year 1 and 59% in Year 6. Detailed demographic information is available in Table 1.1.

2.1.1 Recruitment

Volunteers were recruited via mass mailings, using commercial mailing lists, to all families with children in the target age range (7-13 years in Year 1) within the geographic radius of 50 miles from a Northwest University in the United States. The mailing made clear that the study was looking for children with possible or definite ADHD, as well as typically developing children with no history of learning or attention problems. In

response to mailings, 2144 inquiries were received. During an initial screening phone call, nearly half of the initial inquiries were excluded because of prescribed non-stimulant psychotropic medications, a history of non-febrile seizure, head injury with loss of consciousness > 60 seconds, autism spectrum disorder or intellectual disability, or any other major medical conditions that precluded completion of testing sessions. Children with ADHD taking stimulant medications were included in the study. Those who were excluded at this stage did not differ from the final sample on sex (p = .11) or race (p = .22) but reported marginally lower family income (p = .06) and were slightly younger (p = .06). Behavioral ratings data were collected and managed using REDCap electronic data capture tools hosted locally, which provided a secure web-based and intuitive interface and export capabilities (Harris et al., 2009).

For remaining participants (n = 1449), an in-person "diagnostic" visit was scheduled. Parents and teachers of participants, as well as participants themselves, completed multiple assessments, including those pertaining to ADHD (ADHD Rating Scale and Kiddie Schedule for Affective Disorders and Schizophrenia; DuPaul et al., 1998; Puig-Antich & Ryan, 1986) and IQ (WISC-IV Vocabulary, Block Design, and Information subtests; Sattler & Dumont, 2004; Wechsler, 2003). Among eligible children with ADHD, 154 (35%) were prescribed stimulant medications and needed to complete a washout, only slightly lower than rates in community surveys for pre-adolescent children. Parents/caregivers were instructed to rate children as if not taking medication. All clinical interviewers and psychometric testers were trained to a reliability of kappa > .80 for all diagnoses on the Kiddie Schedule for Affective Disorders and Schizophrenia and had

videotapes viewed by a supervisor to prevent procedural drift. Participants were contacted once per year for the following seven years to complete the same assessments.

2.1.2 Diagnostic Assignment

All materials were scored and presented to a clinical diagnostic team comprising a board-certified child psychiatrist and a licensed child neuropsychologist. Implementing a best estimate procedure (Kosten & Rounsaville, 1992), each clinician independently assigned diagnoses based on parent and teacher ratings, parent clinical interview, IQ and achievement testing, and behavioral observations. Their agreement rate for all diagnoses was satisfactory (ADHD: $\kappa = 0.88$; all other disorders with at least 5% base rate: $\kappa > 0.68$). Disagreements were conferenced to consensus or excluded.

To count ADHD symptoms, clinicians used the following rule: if both parent and teacher ratings exceeded a t-score of 60 on at least one ADHD scale and both rated at least three symptoms as "often" or "very often" on the ADHD rating scale (or for parents, were counted present on the Kiddie Schedule for Affective Disorders and Schizophrenia), the "or" algorithm could be employed (i.e., a symptom is present if either the parent OR the teacher endorses a specific symptom; Lahey et al., 1994). When either informant fell below this mark, and clinicians judged that this was not explained by successful medication treatment during the school day, then the case was rejected as failing to meet the DSM requirement of substantial symptoms present in more than one setting. In addition, it was required that all other DSM criteria were met, including (a) impairment (determined through clinical interview and questionnaires), (b) onset prior to age 7, (c) sustained impairing symptoms > 1 year, and (d) symptoms of ADHD were not better accounted for by comorbid conditions, trauma history, or other confounds.

2.1.3 Longitudinal Retention

After the diagnostic session, 103 participants withdrew due to lack of further interest (e.g., only wanted the diagnostic screen), and 497 were ruled out for the following reasons: excess teacher-parent rating discrepancy (situational problems; 35%), subthreshold symptom count (not control or ADHD, 17%), psychosis, mania, current severe depressive episode, Tourette's syndrome, or head injury (10%), autism (7%), other health condition (7%), ineligible medication (2%), IQ < 80 (n = 1), unknown (n = 1), or multiple rule outs. This resulted in a final sample of 849 children, from whom 610 were selected for long term follow-up study. Of those 610, resource limitations mandated a planning missing design from among those youth such that 413 children were seen in Year 6 (data collection is still ongoing, and some children were excluded from the current study because of incomplete data).

2.2 Measures

2.2.1 ADHD Symptoms

The parent-reported version of the ADHD-Rating Scale (ADHD-RS) was used to assess ADHD symptomatology (DuPaul et al., 1998). This scale contained nine inattentive and nine hyperactive/impulsive symptom items consistent with DSM-IV criteria. Parents responded to all 18 items (Year 1: $\alpha = .7$; Year 6: $\alpha = .96$) on a 0 (i.e., "never or rarely") to 3 (i.e., "very often") scale. These items, referred to subsequently as symptoms, were included in networks. Additionally, all symptoms were included in SEM models examining common cause and direct causation effects separately.

2.2.2 Depressive Symptoms

The child-report Children's Depression Inventory (CDI) was used as an assessment of depression (the study was initiated prior to the publication of the CDI-II; measures were retained to facilitate other studies of change over time; Kovacs, 1985, 1992). Children responded to items assessing different aspects of depression on a 0 to 2 scale, with some responses reversed to ensure that 2 represented the severe form of an aspect (i.e., "I hate myself" versus "I do not like myself" versus "I like myself; symptoms are labeled below using the most severe option). To constrain the number of items due to statistical power considerations in networks, 10 items from the validated short version of the CDI were utilized (CDI-S; Kovacs, 2003; Year 1: $\alpha = .72$; Year 6: $\alpha = .85$). These items, referred to subsequently as symptoms, were also included in SEM models.

2.2.3 Anxiety Symptoms

The child-report Multidimensional Anxiety Scale for Children (MASC) was used as an assessment of anxiety (the study was initiated prior to the publication of the MASC-2; March, 1998). Children responded to items assessing aspects of anxiety (e.g., "I feel tense or uptight") on a 0 ("Never true about me") to 3 ("Often true about me") scale. Like depression, 10 items that have been specified by the MASC as making up an anxiety disorder index (ADI) were included in analyses (Year 1: $\alpha = .59$; Year 6: $\alpha = .69$). The ADI has been suggested to demonstrate a strong association with and exhibit high diagnostic efficiency with respect to anxiety disorders (Ivarsson, 2006; March, 1998). The 10 items, referred to subsequently as symptoms, were also included in SEM models.

2.2.4 Temperament Traits

The 157-item Temperament in Middle Childhood Questionnaire (TMCQ; Simonds, 2006) was used to assess traits in Year 1, and the 62-item Early Adolescent Temperament Questionnaire - Revised (EATQ-R; Ellis & Rothbart, 2001) was used in Year 6. The use of the two measures was driven by recommendations made by their authors and based on the age range of participants at each measurement point. For both measures, parents/caregivers rated items assessing participants' temperament-related behaviors on a 1 ("Almost always untrue") to 5 ("Almost always true") scale. Scores were then summed to form lower-order scales (e.g., activity level, affiliation, inhibitory control). After these scales were created, and in line with prior work (Ellis & Rothbart, 2001; Simonds, 2006), activation control, attention, and inhibitory control scales (+ low intensity pleasure and perceptual sensitivity for the TMCQ) were averaged to obtain effortful control composite scores (Year 1: $\alpha = .71$; Year 6: $\alpha = .89$), and frustration, depressed mood, and aggression scales (for the TMCQ: anger/frustration, discomfort, fear, sadness, and soothability) were averaged to obtain negative affect scores (Year 1: $\alpha = .87$; Year 6: $\alpha = .80$).

Scores on effortful control and negative affect derived from the EATQ have been linked to internalizing and externalizing behaviors (Snyder et al., 2015), and have been found to differ in youth with versus without ADHD (De Pauw & Mervielde, 2010). Prior analyses of the TMCQ have also supported convergent validity of the three higher-order traits (Nystrom & Bengtsson, 2017), with the factor structure of scales also being validated in ADHD samples (Nigg et al., 2020). Surgency was not included in analyses. As noted in the introduction, it has opposite relations with ADHD (positively associated)

and internalizing disorders (negatively associated), and thus seemed less promising as a common cause (i.e., ADHD with high positive affect should be a protective factor against internalizing disorders). Effortful control scores were reversed so that for both effortful control and negative affect, higher scores indicated greater maladaptivity. The text below refers to "low effortful control" as the risk factor to retain clarity.

2.3 Analytic Plan

2.3.1 Network Visualization and Interpretation

Gaussian Graphical Models (GGM) were constructed using the R packages *bootnet* and *qgraph* to depict relations between the traits and symptoms of ADHD and internalizing disorders (Epskamp et al., 2018; Epskamp & Fried, 2018; Epskamp et al., 2012). Four networks were created for primary analyses: (1) Year 1 ADHD-depression, (2) Year 6 ADHD-depression, (3) Year 1 ADHD-anxiety, and (4) Year 6 ADHD-anxiety. Networks were estimated using the graphical least absolute shrinkage operator (GLASSO; Friedman et al., 2008) in combination with extended Bayesian Information Criterion (EBIC) model selection (Foygel & Drton, 2010), resulting in sparse networks containing only the strongest regularized partial Spearman correlations.[3] A gamma (γ) hyperparameter of 0.2 for ADHD-anxiety networks, and 0.1 for ADHD-depression networks, was selected for the EBIC to balance network stability with regularization's

[3] Partial correlations range from -1 to 1 and correspond with the remaining association between two variables within a network after controlling for all other variables. This contrasts with bivariate correlations which do not account for other variables. However, spurious relations (i.e., false positives) are still possible in networks given the high number of parameters that are estimated. Hence, regularization techniques apply a "penalty" to the strength of all relations within a network, decreasing their strength and removing weaker relations. Together, these two techniques are thought to increase the likelihood of creating a network structure that minimizes the number of spurious relations while highlighting the strongest ones (see Epskamp & Fried, 2018).

specificity and sensitivity. Visualization of networks was based on Multidimensional Scaling (MDS) techniques, which have recently been proposed to facilitate more accurate visual interpretation than more commonly used Fruchterman-Reingold networks (Fruchterman & Reingold, 1991; Jones et al., 2018). MDS networks were created using the R package *networktools* (Jones, 2018).

2.3.2 Identifying Bridge Symptoms in Networks

Bridge Expected Influence (i.e., the sum of partial correlations attached to a symptom from variables measuring another construct, like symptoms of another disorder or temperament traits, and vice versa; Jones et al., 2019), was used to identify bridge elements in networks. Prior work has suggested BEI may be preferable to other types of centrality when networks include both positive and negative relations among elements (McNally, 2016; Robinaugh et al., 2016). In the current study, BEI was used to examine two different types of bridge elements: (1) those pertaining to ADHD and internalizing disorder symptoms' relations with temperament traits, and (2) those pertaining to relations between symptoms of ADHD and internalizing disorders. To determine bridge symptoms, bootstrapped tests statistically comparing the BEI of symptoms and traits were conducted using 2000 samples with replacement and the *bootnet* R-package. These tests involved creating a difference score between the bootstrapped BEI values of symptoms and traits, along with a confidence interval around this difference score. Bridge elements were identified as those that exhibited a significantly ($p < .05$) higher BEI than other elements based on these tests.

2.3.3 Estimating SEM Models

All models were estimated in Mplus version 7.4 (Muthén & Muthén, 1998-2012)

using the using the weighted least square estimator to account for ordinal data (WLSMV; Flora & Curran, 2004). Analyses were conducted separately for ADHD-depression and ADHD-anxiety in Years 1 and 6. Two common cause models were tested: the first specified all ADHD and depression/anxiety symptoms as loading on to a general factor. Additionally, given the strong internal consistency particularly among ADHD symptoms, along with findings of recent studies supporting multi-level conceptualizations of psychopathology (Forbes et al., 2021), a second model was tested where ADHD and depression/anxiety symptoms were specified as loading onto corresponding inattention, hyperactivity/impulsivity, and depression/anxiety factors, with these factors then specified as loading onto a general factor. In both models, the general factor was correlated with effortful control and negative affect (also specified as correlating) to explore these traits as common causes of ADHD-internalizing disorder overlap. Good model fit was determined using the following criteria: Root Mean Square Error of Approximation (RMSEA) < .06 and Comparative Fit Index (CFI) > .95, and the Tucker-Lewis Index (TLI) > .90 (Weston & Gore Jr, 2006).

The direct causation model was also assessed using Mplus. Consistent with recent studies and using the maximum likelihood estimator (Fenesy & Lee, 2019; Riglin et al., 2020; Stern et al., 2020; Ward et al., 2019), inattention and hyperactivity/impulsivity latent factors encompassing respective ADHD symptoms were specified as predictors of a depression (and separately, anxiety) latent factor encompassing all CDI-S (MASC-ADI) symptoms. Inattention and hyperactivity/impulsivity factors were specified as correlating in both models. Latent factors were used instead of composite scores to reduce measurement error.

2.3.4 Assessing Age and Gender-Based Differences in Network Structure

Age and gender-based differences in the structure and overall strength of relations among elements in ADHD-depression and ADHD-anxiety networks were assessed using the R-package *NetworkComparisonTest* (NCT; Van Borkulo et al., 2017). Each comparison only included participants who had complete data, as required by the dependent comparison test within NCT (Van Borkulo et al., 2017). Three primary tests of invariance were conducted by permuting the data to reflect the null hypothesis 1000 times: the first compared global expected influence estimates (GEI), or the sum of all partial correlations in each network pair (i.e., Year 1 versus Year 6, boys versus girls). The second assessed whether network pairs contained generally consistent relations among symptoms and traits by assessing the maximum difference in respective relations (i.e., network structure; M). The third test statistically compared the BEI of respective symptoms and traits in network pairs.

Table 2.1 Demographic Information

	Year 1		Year 6	
	ADHD n = 476	Non-ADHD n = 323	ADHD n = 123	Non-ADHD n = 254
Gender [n (%) Girls] [ab]	143 (30.0)	167 (51.7)	33 (26.8)	102 (40.2)
Age [*M* (SD)]	9.75 (1.51)	9.58 (1.61)	14.24 (1.40)	14.44 (1.42)
Race [n (%) White] [a]	395 (83.0)	285 (88.2)	93 (75.6)	207 (81.5)
Yearly Family Income [n (%)] [ac]				
0 – 50,000	130 (27.3)	58 (18.0)	-	-
50,001 - 100,000	193 (40.5)	142 (44.0)	-	-
100,001 – 150,000	87 (18.3)	68 (21.1)	-	-
> 150,001	24 (5.0)	27 (8.4)	-	-
Estimated FSIQ [*M* (SD)] [ab]	108.50 (13.83)	114.88 (12.93)	108.21 (15.16)	114.48 (12.85)
Inattentive Sum Score [*M* (SD)] [ab]	17.09 (5.63)	3.23 (4.06)	17.47 (4.97)	6.55 (5.91)
Hyperactive/Impulsive Sum Score [*M* (SD)] [ab]	13.18 (6.49)	2.57 (3.40)	10.59 (5.79)	3.75 (4.81)
CDI-S Depression Sum Score [*M* (SD)] [ab]	2.70 (2.67)	1.58 (2.01)	2.90 (3.45)	2.15 (2.77)
MASC-ADI Anxiety Sum Score [*M* (SD)] [a]	12.91 (4.67)	12.21 (4.15)	12.80 (4.29)	12.21 (4.49)
Negative Affect [*M* (SD)] [ab]	2.71 (0.58)	2.32 (0.50)	2.79 (0.63)	2.30 (0.57)
Effortful Control [*M* (SD)] [abd]	3.04 (0.35)	2.30 (0.38)	3.48 (0.46)	2.52 (0.66)

Notes.

[a] Significant difference between those with and without ADHD in Year 1 ($p < .05$).

[d] Significant difference between those with and without ADHD in Year 6 ($p < .05$).

[c] Income data was only obtained at Year 1, and 55 participants did not have income data available.

[b] Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

CHAPTER 3. RESULTS

3.1 Missing Data[4]

The larger study had originally included 849 participants, but those with any missing data had the rest of their data listwise deleted for each analysis (i.e., Year 1 ADHD-depression, Year 1 ADHD-anxiety, Year 6 ADHD-depression, Year 6 ADHD-anxiety) due to the requirements of network analysis. In Year 1, this resulted in data for 57 participants being deleted for ADHD-depression analyses (final n = 792), and data for 52 participants being deleted for ADHD-anxiety analyses (final n = 797). Comparison of participants with and without missing data suggested those with missing data did not differ from those with complete data in terms of gender (*ps* ≥ .17), race/ethnicity (*ps* ≥ .26), negative affect (*ps* ≥ .08), Year 1 CDI-S sum score (*p* = .14), and Year 1 MASC-ADI sum score (*p* = .25), although those with missing data were significantly younger (*ps* ≤ .04, *partial* η^2*s* = .01) and had significantly lower FSIQ scores (*ps* ≤ .03, *partial* η^2*s* = .01). For ADHD-depression analyses, those with missing data had higher effortful control and Year 1 ADHD-RS sum scores (*p*s = .04, *partial* η^2*s* = .01), although the effect sizes of these differences were small and not likely meaningful. These differences were not present in ADHD-anxiety analyses (*p*s ≥ .10).

[4] Past studies based on simulations have suggested three participants per estimated parameter as a rule of thumb to achieve adequate statistical power for network analysis (Fried & Cramer, 2017). Hence, it is likely that network analyses were somewhat underpowered, particularly in Year 6. However, the use of regularization techniques, stability of relations and BEI in networks, and a focusing on the most robust relations mitigated these power concerns. Past studies examining SEM have failed to establish a consensus concerning appropriate sample sizes for SEM, with one generally accepted rule of thumb suggesting 10 observations per indicator variable (Nunnally, 1967). The current study met this criterion, although further work is needed to confirm results.

In Year 6, data was available for 372 participants for ADHD-depression analyses, and 375 for ADHD-anxiety, due to planned missingness and participant attrition. Comparison of participants with and without missing data suggested those with complete versus missing data did not differ in terms of gender (*ps* ≥ .17), race/ethnicity (*ps* ≥ .26), Year 1 CDI-S sum score (*p* = .79*)*, Year 1 MASC-ADI sum score (*p* = .11), Year 1 negative affect (*ps* ≥ .41), and Year 1 effortful control (*ps* ≥ .15). Those with missing data were significantly older (*ps* < *.001*, *partial* η^2*s* = .05), had significantly lower FSIQ scores (*ps* =.001, *partial* η^2*s* = .01), and significantly higher ADHD-RS sum scores (*p* = .001, *partial* η^2 = .01) than those without missing data, although the effect sizes of these differences were small and not likely meaningful.

3.2 Direct Causation and Common Cause Effects in Networks[5]

Preliminary analyses assessing stability in networks, as pertaining to relations

[5] Tautological overlap among elements in networks was examined using the Goldbricker function in the R package *networktools* (Jones, 2018). This package sought to identify potential pairs of variables correlated both with each other (r > .50) and in highly similar patterns with other elements (less than 25% of overlapping correlations with other variables being significantly different [*p* < .05]). In ADHD-depression analyses, redundancy was identified in Year 1 between the inattentive symptoms *has difficulty organizing tasks/activities* and *is forgetful*, as well as between impulsive symptoms *blurts out* and *interrupts/intrudes.* In Year 6, redundancy was identified between the inattentive symptoms *has difficulty organizing tasks/activities* and *is forgetful*, as well as the depressive symptoms *I feel like crying every day* and *I look ugly.* To address this redundancy, new variables were created for each overlapping variable pair based on the first principal component of the two variables within a principal component analysis. Results of revised network analyses including combined variables were generally consistent with those presented in the main text. In ADHD-anxiety network analyses, redundancy was identified in Year 1 between the inattentive symptoms *has difficulty organizing tasks/activities* and *is forgetful.* In Year 6, redundancy was identified between *has difficulty organizing tasks/activities* and *is forgetful,* between f*idgets* and *has difficulty engaging in leisure activities quietly,* and between *has difficulty awaiting turn* and *interrupts/intrudes.* After combining variable pairs, results did not meaningfully change. Depictions of networks combining redundant variables are available in the Appendices, with detailed results available upon request.

As an additional analysis, ADHD-depression and ADHD-anxiety networks across years were replicated after removing the Attention scale from the calculation of effortful control, and the Fear and Sadness scales and Depressive Mood scales from the calculation of negative affect on the TMCQ and EATQ-R, respectively. Network comparison tests suggested no differences in structure between these revised networks and those presented in the main body (correlations between respective relations: *r*s ≥ .95; tests for differences in network structure: *p*s > .98). Detailed results of these analyses are available upon request.

among variables in networks as well as variables' BEI, indicated networks were stable unless noted otherwise below (see Appendices for detailed information). It should also be noted that given use of regularization techniques, it is likely that all relations presented below, even if seemingly negligible in strength, had values meaningfully different than zero (Epskamp & Fried, 2018). Visualization of ADHD-depression and ADHD-anxiety networks across years, as well as detailed results of BEI analyses in networks, are depicted in Figures 3.1-3.4.

3.2.1 ADHD-Depression

Visual interpretation of networks across years suggested inattentive and hyperactive/impulsive symptoms as being related to depressive symptoms across years, with impulsive symptoms, as a set, appearing to be somewhat more strongly related to depression in Year 6 than 1. Effortful control appeared noticeably more related to ADHD symptoms, particularly those in the inattentive symptom domain, across years, while negative affect appeared to demonstrate more consistent relations with symptoms of both disorders.

Across years, lower levels of effortful control emerged as a primary bridge risk marker via positive relations with inattentive (Year 1: *has difficulty sustaining attention, does not follow through, reluctant to engage in tasks requiring sustained mental effort; difficulty awaiting turn*; Year 6: all inattentive symptoms) and depressive (Year 1: *nothing will ever work out for me; I do everything wrong; I do not have any friends; nobody really loves me;* Year 6: *nobody really loves me*) symptoms. Although it had a lower BEI than effortful control across years, negative affect was found to be positively related to several hyperactive/impulsive (Year 1: *blurts out; has difficulty awaiting turn;*

loses things; Year 6: *does not listen; has difficulty awaiting turn; intrudes*) and depressive symptoms (Year 1: *I feel like crying every day; things bother me all the time; I am sad all the time; Year 6: things bother me all the time; I feel alone all the time; I hate myself*). Bridge symptoms were primarily from the ADHD inattentive symptom domain via relations with low effortful control, although *difficulty awaiting turn* (hyperactive/impulsive symptom domain) also emerged as a bridge symptom via positive relations with negative affect across years.

Exploration of ADHD-depression relations suggested no differences in ADHD symptoms' BEI in Year 1. Conversely, two bridge symptoms of depression were identified (correlated ADHD symptoms are listed in parentheses): *I do everything wrong* (*fails to give close attention; does not follow through; fidgets; leaves seat; shifts around excessively; blurts out*) and *I do not have any friends* (*does not listen; has difficulty organizing; is forgetful; has difficulty awaiting turn*). In Year 6, three ADHD bridge symptoms, all from the inattentive symptom domain, were identified (correlated depressive symptoms are listed in parentheses): *loses things* (*I look ugly; I do not have any friends; nobody really loves me*), *does not follow through* (*I do everything wrong; nobody really loves me*), and *has difficulty sustaining attention* (*nothing will ever work out for me; I do everything wrong; things bother me all the time*). No depressive symptoms emerged as bridge symptoms.

3.2.2 ADHD-Anxiety

Visual interpretation suggested results consistent with those in ADHD-depression analyses: both inattentive and hyperactive/impulsive symptoms appeared related to anxiety symptoms across years, with impulsive symptoms perhaps being more related to

anxiety than hyperactive symptoms in Year 6. Additionally, effortful control appeared to be more closely related to ADHD symptoms, particularly those in the inattentive symptom domain, while negative affect appeared to demonstrate relations with symptoms of both disorders across years.

Regarding trait-disorder relations, results in Year 1 suggested effortful control as having a significantly higher BEI than negative affect. However, follow-up investigation suggested that effortful control's BEI was highly driven by robust positive relations with almost all ADHD inattentive symptoms across years (i.e., lower levels of effortful control associated with greater inattention). Conversely, lower levels of effortful control were related to increased severity of only one anxiety symptom in Year 1 (*I get dizzy or faint feelings*) and 6 (*I am afraid other people will think I'm stupid*). In contrast, negative affect demonstrated positive relations with anxiety (*I feel tense or uptight; I have trouble catching my breath; I am afraid that other kids will make fun of me; I get dizzy or faint feelings*), inattentive (*does not listen; reluctant to engage in tasks requiring sustained mental effort; loses things*) and hyperactive/impulsive (*shifts around excessively; has difficulty engaging in leisure activities quietly; talks excessively; blurts out; has difficulty awaiting turn; leaves seat; interrupts or intrudes*) symptoms across years. Bridge symptoms of were primarily from the ADHD inattentive symptom domain via relations with low effortful control, although *difficulty awaiting turn* also emerged as a bridge symptom via positive relations with negative affect across years.

Assessment of BEI suggested no bridge symptoms in Year 1. In Year 6, the inattentive symptom *loses things* (correlated anxiety symptoms: *I feel tense or uptight; I get dizzy or faint feelings; I am afraid other people will think I'm stupid; I get scared*

riding in the car or the bus) emerged as an ADHD bridge symptom, whereas *I am afraid that other people will think I'm stupid* (correlated ADHD symptoms: *has difficulty sustaining attention; loses things; talks excessively*) emerged as the only anxiety bridge symptom.

3.3 Summary of Network Results

3.3.1 ADHD-Depression Network Summary

Pertaining to trait-disorder relations, low effortful control emerged as a transdiagnostic risk marker in Year 1 and 6, although it was related primarily to increased severity of inattentive ADHD bridge symptoms and secondarily to increased severity of depressive bridge symptoms associated with decreased perceived self-competency, low self-worth, and social problems. Negative affect also emerged as a transdiagnostic risk marker via positive relations with hyperactive/impulsive symptoms (namely the ADHD bridge symptom *difficulty awaiting turn*) and depressive symptoms associated with negative mood and distress.

Regarding ADHD-depression relations in Year 1, two depression bridge symptoms were identified: *I do everything wrong* (via unique relations with ADHD symptoms associated with difficulties sustaining attention, following through, restlessness, and a tendency to blurt out), and *I do not have any friends* (difficulties listening, staying organized, waiting one's turn, and forgetfulness). In Year 6, three ADHD bridge symptoms were identified: *loses things* (via unique relations with depressive symptoms associated with low self-worth and social problems), *does not follow through* (decreased perceived self-competency and low self-worth), and *difficulties*

sustaining attention (hopelessness, decreased perceived self-competency, distress).

3.3.2 ADHD-Anxiety Network Summary

Investigation of trait-disorder relations suggested effortful control as demonstrating relations primarily with inattentive bridge symptoms. Conversely, negative affect appeared better conceptualized as a transdiagnostic risk marker via positive relations with both inattentive and hyperactive/impulsive symptoms (primarily the hyperactive/impulsive bridge symptom *difficulty awaiting turn*), as well as anxiety symptoms associated with somatic problems and peer-related fears. When focused on ADHD-anxiety relations, results did not suggest any bridge symptoms in Year 1. In Year 6, results suggested one ADHD bridge symptom, *loses things* (via relations with anxiety symptoms associated with somatic problems, fear of negative evaluation from peers, and fear of riding in vehicles), and one anxiety bridge symptom, *I am afraid that other people will think I'm stupid* (correlated ADHD symptoms were associated with difficulty sustaining attention, losing things, and talking excessively).

3.4 Common Cause and Direct Causation Effects via SEM

3.4.1 ADHD-Depression: Common Cause Model

Detailed results for this model are provided in Figure 3.5. In Years 1 and 6, the first model (inattentive, hyperactive/impulsive, and depressive symptoms loading on to a general factor) produced significant chi-square values (χ^2s[404] $\geq$ 763.98, *ps* < .001). Fit indices indicated the model exhibited poor fit across years particularly based on RMSEA (Year 1: RMSEA = .10, CFI = .95, TLI = .94; Year 6: RMSEA = .16, CFI = .83, TLI = .81). Across years, the second model (i.e., inattentive, hyperactive/impulsive, and

depressive symptoms loading onto corresponding inattention, hyperactivity/impulsivity, and depression factors, with these factors, in turn, loading on to the general factor) produced significant chi-square values (χ^2s[401] ≥ 3768.90, *ps* < .001). Fit indices indicated good fit across years (Year 1: RMSEA = .04, CFI = .99, TLI = .99; Year 6: RMSEA = .05, CFI = .98, TLI = .98).

Standardized results of the second model suggested all inattentive, hyperactive/impulsive, and depressive symptoms loaded on to respective latent factors (λs ≥ .44, *p*s < .001), with inattention and hyperactivity/impulsivity latent factors' loadings on the general factor also being significantly greater than zero (λs ≥ .84, *p*s < .001). The strength of the depression factor's loading on the general factor was also significantly greater than zero (λs ≥ .17, *p*s ≤ .01), although it was much weaker than that of ADHD latent factors. The general factor was found to be significantly and positively correlated with both effortful control and negative affect (Year 1: *rs* ≥ .51; *ps* < .001, Year 6: *rs* ≥ .56; *ps* < .001). In sum, conceptualization of the common cause model through SEM suggested inattentive, hyperactive/impulsive, and depressive symptoms were most accurately reflected via three corresponding latent factors, with these factors, in turn, encompassed by a general factor that was positively related to low effortful control and negative affect.

3.4.2 ADHD-Depression: Direct Causation Model

Detailed results for this model are provided in Figure 3.6. Across years, the direct causation model (i.e., inattention and hyperactivity/impulsivity latent factors as indicators of a depression latent factor) produced significant chi-square values (χ^2s[347] ≥ 618.24, *ps* < .001). Fit indices indicated this model fit provided good fit to the data across years

(Year 1: RMSEA = .04, CFI = .99, TLI = .99; Year 6: RMSEA = .05, CFI = .99, TLI = .99). Standardized model results suggested all inattentive, hyperactive/impulsive, and depressive symptoms loaded significantly onto their respective factors (λs $\geq$ .44, *p*s < .001). The inattention latent factor emerged as an indicator of the depression latent factor across years, although this effect was marginally significant in Year 1 (Year 1: β = .17, *p* = .07; Year 6: β =.58, *p* < .001). The hyperactivity/impulsivity latent factor did not demonstrate a significant relationship with the depression latent factor in Year 1 (β = .15; *p* = .11) and demonstrated a *negative* relation with depression in Year 6 (β = -.45, *p* = .001). In sum, inattention appeared to demonstrate a significant positive relationship with depression across years, while hyperactivity/impulsivity did not appear to be a significant indicator in Year 1 and demonstrated a negative relation with depression in Year 6.

3.4.3 ADHD-Anxiety: Common Cause Model

Detailed results for this model are provided in Figure 3.7. In Years 1 and 6, the first model (inattentive, hyperactive/impulsive, and anxiety symptoms loading on to a general factor) produced significant chi-square values (χ^2s[404] $\geq$ 2566.42, *ps* < .001). Fit indices indicated this model did not fit the data across years (Year 1: RMSEA = .11, CFI = .94, TLI = .94; Year 6: RMSEA = .12, CFI = .90, TLI = .89). Across years, the second model (i.e., inattentive, hyperactive/impulsive, and anxiety symptoms loading onto corresponding inattention, hyperactivity/impulsivity, and anxiety factors, with these factors, in turn, loading on to the general factor) produced significant chi-square values (χ^2s[401] $\geq$ 777.37, *ps* < .001). Fit indices indicated this model provided good fit to the data across years (Year 1: RMSEA = .04, CFI = .99, TLI = .99; Year 6: RMSEA = .05, CFI = .98, TLI = .98).

Standardized results of the second model suggested the loadings of all inattentive and hyperactive/impulsive symptoms on their respective factors were significantly greater than zero (λs $\geq$ .79, *p*s < .001). Loadings of all anxiety symptoms on the anxiety latent factor were significantly greater than zero across years (λs $\geq$ .16, *p*s $\leq$ .003), except for the item *I avoid watching scary movies and TV shows* in Year 1 (λ = .10, *p* = .09). The loadings of inattention and hyperactivity/impulsivity latent factors on the general factor were significantly greater than zero (λs $\geq$ .86, *p*s < .001). The anxiety factor also exhibited a loading significantly greater than zero across years (λs $\geq$ .14, *p*s $\leq$ .02), although much lower than inattention and hyperactivity/impulsivity factors. The general factor was found to be significantly and positively correlated with both low effortful control and negative affect (Year 1: *rs* $\geq$ .51; *ps* < .001, Year 6: *rs* $\geq$ .56; *ps* < .001). In sum, conceptualization of the common cause model through SEM across years suggested inattentive, hyperactive/impulsive, and most anxiety symptoms could be accurately encompassed through three corresponding factors, with these factors, in turn, encompassed by a general factor that was positively related to low effortful control and negative affect.

3.4.4 ADHD-Anxiety: Direct Causation Model

Detailed results for this model are provided in Figure 3.8. Across years, the direct causation model (i.e., inattention and hyperactivity/impulsivity latent factors as indicators of an anxiety latent factor) produced significant chi-square values (χ^2s[347] $\geq$ 670.85, *ps* < .001). Fit indices indicated this model fit provided good fit to the data across years (Year 1: RMSEA = .04, CFI = .99, TLI = .99; Year 6: RMSEA = .05, CFI = .99, TLI = .98). Standardized model results suggested all inattentive, hyperactive/impulsive, and

almost all anxiety symptoms loaded significantly onto their respective factors ($\lambda s \geq .18$, $ps < .001$). As in common cause analyses, the anxiety symptom *I avoid watching scary movies and TV shows* exhibited a marginally significant loading on the anxiety latent factor in Year 1 ($\lambda = .10$, $p = .06$), although its loading was significantly greater than zero in Year 6 ($\lambda = .17$, $p = .01$). In Year 1, neither inattention nor hyperactivity/impulsivity latent factors emerged as significant indicators of the anxiety latent factor ($ps \geq .26$). In Year 6, the inattention factor demonstrated a significant positive relation with the anxiety latent factor ($\beta = .38$, $p = .001$), while the hyperactivity/impulsivity factor demonstrated a significant negative relation ($\beta = -.27$, $p = .03$). In sum, inattention and hyperactivity/impulsivity did not appear to be significant indicators of the anxiety latent factor in Year 1. However, in Year 6, inattention appeared to be positively related, and hyperactivity/impulsivity negatively related, to the anxiety factor.

3.5 Summary of SEM Results

3.5.1 ADHD-Depression SEM Summary

Results of SEM conceptualizations of ADHD-depression co-occurrence provided support for both common cause and direct causation effects. Across years, examination of a common cause conceptualization suggested inattentive, hyperactive/impulsive, and depressive symptoms as adequately reflected through three corresponding latent factors, with these factors (particularly inattention and hyperactivity/impulsivity, less so depression), in turn, encompassed by a general factor. Higher scores on this general factor were associated with lower levels of effortful control and higher levels of negative affect. Separately, results provided support for a direct causation conceptualization, with

the inattention latent factor being positively associated with a depression latent factor across years. Conversely, the hyperactivity/impulsivity factor was not a significant indicator in Year 1 and was negatively related to the depression factor in Year 6.

3.5.2 ADHD-Anxiety SEM Summary

As with those of ADHD-depression, results of an SEM approach to ADHD-anxiety co-occurrence provided some support for common cause and direct causation conceptualizations. Regarding common cause conceptualizations, results suggested symptoms could be adequately reflected by three latent factors (inattention, hyperactivity/impulsivity, anxiety), with these three factors (particularly inattention and hyperactivity/impulsivity, less so anxiety) encompassed by a general factor. Higher scores on this general factor were associated with lower levels of effortful control and higher levels of negative affect. Pertaining to direct causation effects, results suggested the inattention factor was positively related, and the hyperactivity/impulsivity factor negatively related, to the anxiety factor in Year 6. Neither ADHD factor was related to the anxiety factor in Year 1.

3.6 Robustness of Network Structure in Year 1 Versus 6

3.6.1 ADHD-Depression Year 1 Versus 6

Preliminary examination of correlations between relations among variables within networks across years suggested respective relations among variables, overall, were robustly correlated (r = .63). Assessment of variables' BEI with respect to trait-disorder relations suggested one ADHD symptom, *leaves seat in classrooms/situations where remaining seating is expected*, that had a significantly higher BEI in Year 1 versus 6 (p =

.03). Focusing specifically on ADHD-depression relations, assessment of symptoms' BEI suggested no differences between Years 1 versus 6 (*ps* > .05). Examination of individual edges within networks suggested no differences across years (*p*s > .05).

3.6.2 ADHD-Anxiety Year 1 Versus 6

Preliminary examination of correlations between relations among variables within networks across years suggested respective relations among variables, overall, were robustly correlated (r = .66). Assessment of variables' BEI with respect to trait-disorder relations suggested one ADHD symptom, *leaves seat in classrooms/situations where remaining seating is expected*, that had a significantly higher BEI in Year 1 versus 6 (p = .03). Focusing specifically on ADHD-anxiety symptom-symptom relations, assessment of symptoms' BEI suggested no differences between Years 1 versus 6 (*ps* > .05). Examination of individual edges within networks suggested no differences across years (*p*s > .05).

3.7 Gender Differences in Bridge Symptoms

3.7.1 Year 1 ADHD-Depression Boys Versus Girls

Network visualizations are depicted in Figure 3.9. Preliminary examination of correlations between relations among variables within networks across years suggested respective relations among variables, overall, were robustly correlated (r = .65). However, it should be noted that preliminary stability analyses suggested BEI, as pertaining to ADHD-depression relations, was not stable (CSs ≤ .13). Hence, results were interpreted with caution. Assessment of variables' BEI with respect to trait-disorder relations suggested no differences in ADHD and depressive symptoms' relations with

traits between boys and girls (*p*s ≥ .59). Similarly, no differences were identified in symptoms BEI focusing specifically on ADHD-anxiety relations (*ps* ≥ .64). Examination of individual edges within networks suggested no differences across years (*p*s > .05).

3.7.2 Year 6 ADHD-Depression Boys Versus Girls

Network visualizations are depicted in Figure 3.10. Preliminary examination of correlations between relations among variables within networks across years suggested respective relations among variables, overall, were robustly correlated (*r* = .49). However, preliminary stability analyses suggested BEI, as pertaining to ADHD-depression relations, was not stable (CSs ≤ .21), so results were interpreted with caution. Assessment of variables' BEI with respect to trait-disorder relations suggested no differences in ADHD and depressive symptoms' relations with traits between boys and girls (*p*s ≥ .66). Similarly, no differences were identified in symptoms BEI focusing specifically on ADHD-depression relations (*ps* ≥ .53). Examination of individual edges within networks suggested no differences across years (*p*s > .05).

3.7.3 Year 1 ADHD-Anxiety Boys Versus Girls

Network visualizations are depicted in Figure 3.11. Preliminary examination of correlations between relations among variables within networks across years suggested respective relations among variables, overall, were robustly correlated (*r* = .63). BEI as pertaining to ADHD-anxiety relations was not stable (CSs = .13), so results were interpreted with caution. Assessment of variables' BEI with respect to trait-disorder relations suggested no differences in ADHD and depressive symptoms' relations with traits between boys and girls (*p*s ≥ .21). Similarly, no differences were identified in symptoms BEI focusing specifically on ADHD-anxiety relations (*ps* ≥ .39). Examination

of individual edges within networks suggested no differences across years (*p*s > .05).

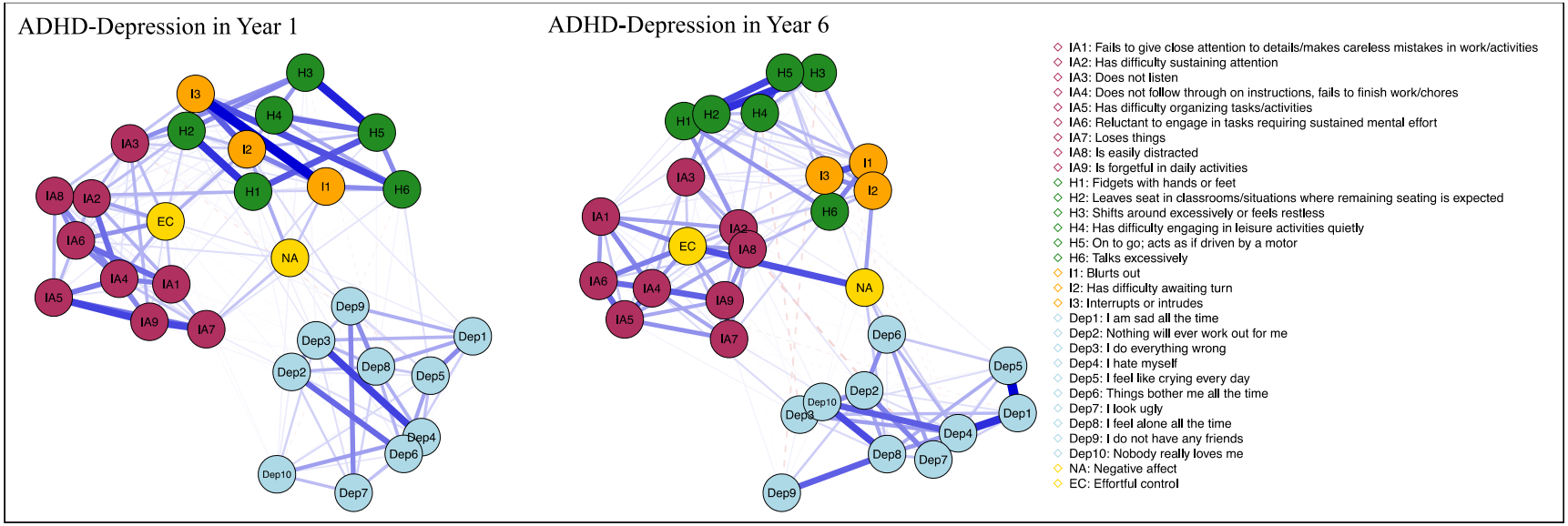
3.7.4 Year 6 ADHD-Anxiety Boys Versus Girls

Network visualizations are depicted in Figure 3.12. Preliminary examination of correlations between relations among variables within networks across years suggested respective relations among variables, overall, were robustly correlated (r = .57). BEI pertaining to ADHD-anxiety relations was not stable (CSs = 0), so results were interpreted with caution. Assessment of variables' BEI with respect to trait-disorder relations suggested no differences in ADHD and depressive symptoms' relations with traits between boys and girls (*p*s ≥ .58). Similarly, no differences were identified in symptoms BEI focusing specifically on ADHD-anxiety relations (*ps* ≥ .78). Examination of individual edges within networks suggested no differences across years (*p*s > .05).

3.8 Summary of Network Comparison Tests

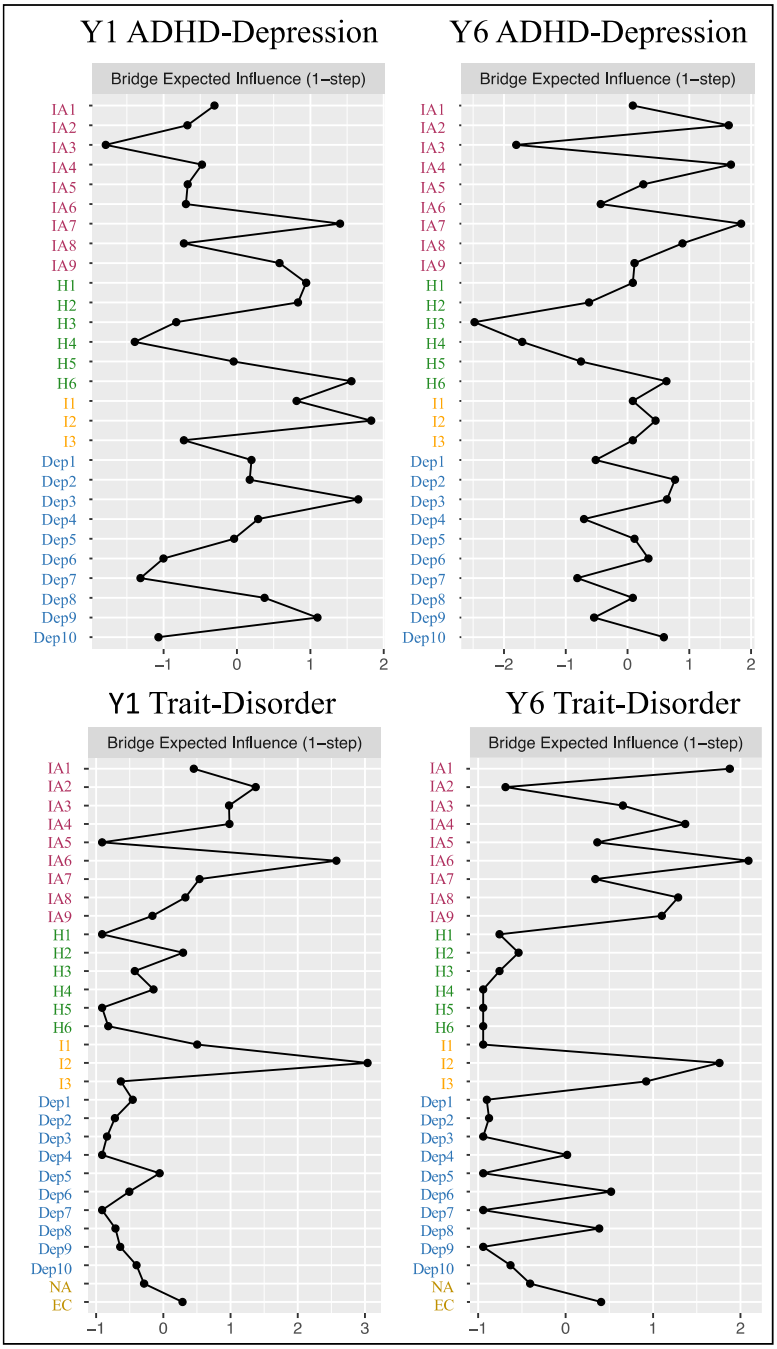
Overall, results of network comparison tests suggested no significant differences in the structure of ADHD-depression and ADHD-anxiety networks across measurement point and gender. Further, it was only when examining ADHD-depression and ADHD-anxiety networks in Year 1 versus Year 6 that any significant differences in BEI were identified: across ADHD-depression and ADHD-anxiety networks, *leaves seat in classrooms/situations where remaining seating is expected* was found to be more strongly related to traits in Year 1 versus Year 6. However, sample sizes were relatively low for network comparison tests and likely contributed to a lack of stability in BEI-related results. Thus, any results pertaining to differences in variables' BEI should be interpreted with caution.

Figure 3.1 ADHD-Depression Network Across Years



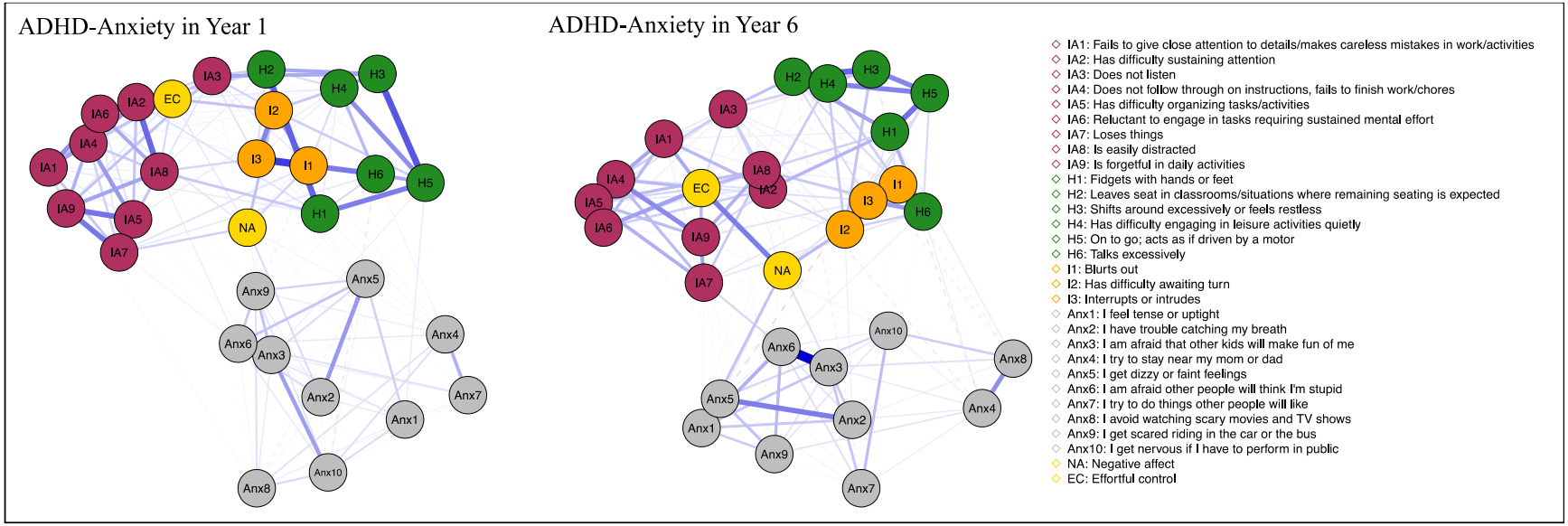
Note. Visualization was created using Multidimensional Scaling. This allows for broad visual interpretations of network structure based on the spacing between variables. Variables are depicted as nodes, with edges connecting these nodes representing regularized partial Spearman correlations. Edge thickness represents the strength of the relation. Solid edges indicate positive relations and dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.2 Bridge Expected Influence Values for ADHD-Depression Networks



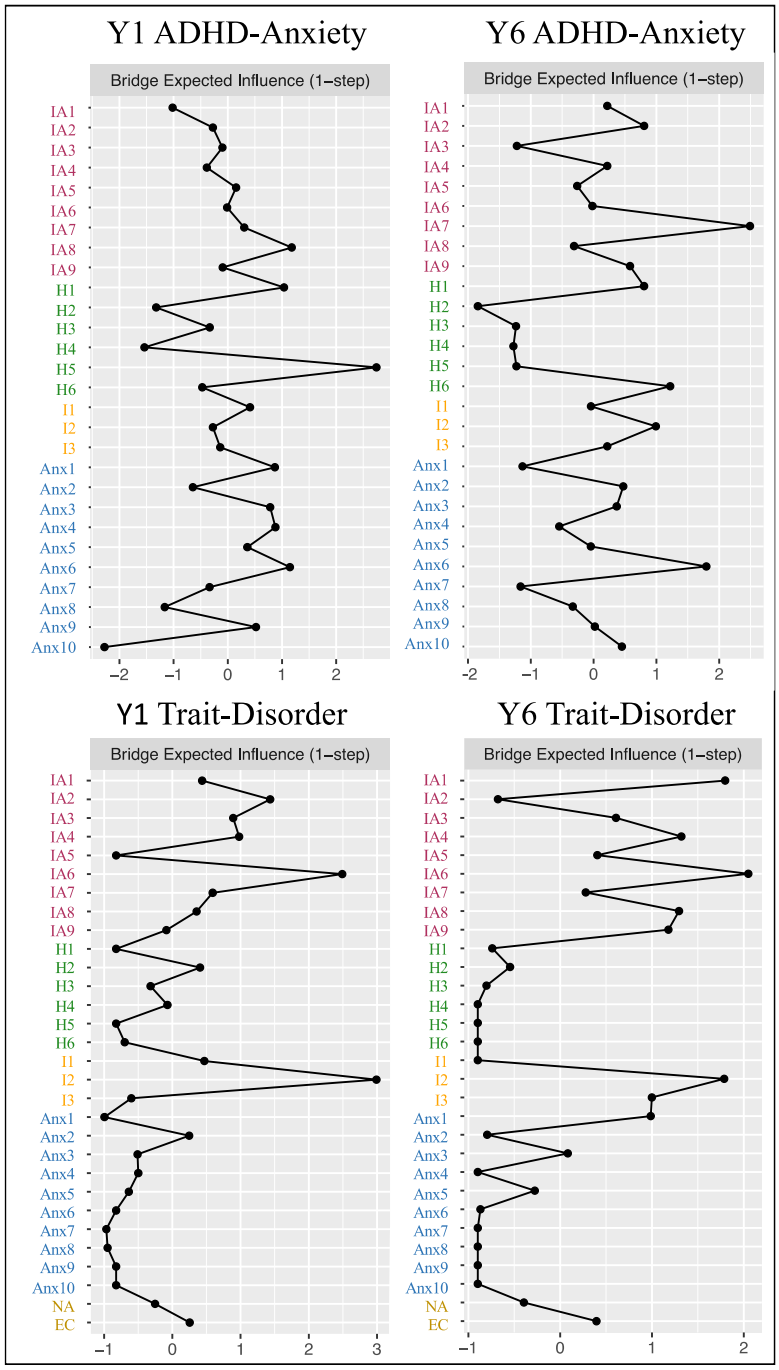
Note. Variables are listed on the y-axes, with BEI z-scores depicted on the x-axis. Values farther to the right indicate that the respective variable demonstrated more robust relations with those of the other community (i.e., ADHD-depression; traits-disorders). The top two figures depict BEI with respect to ADHD-depression relations, and the bottom two figures depict BEI with respect to trait-disorder relations.

Figure 3.3 ADHD-Anxiety Network Across Years



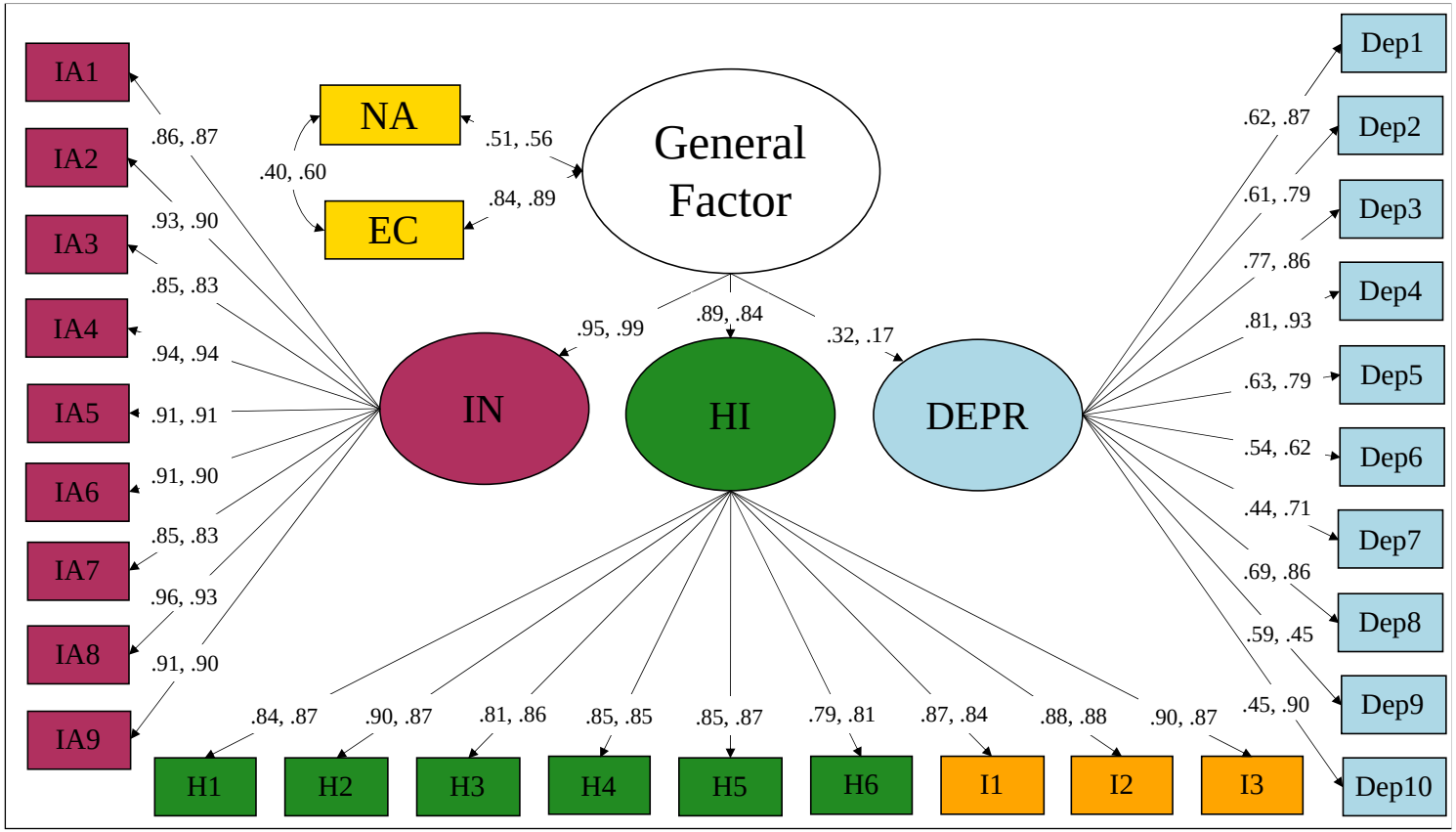
Note. Visualization was created using Multidimensional Scaling. This allows for broad visual interpretations of network structure based on the spacing between variables. Variables are depicted as nodes, with edges connecting these nodes representing regularized partial Spearman correlations. Edge thickness represents the strength of the relation. Solid edges indicate positive relations and dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.4 Bridge Expected Influence Values for ADHD-Anxiety Networks



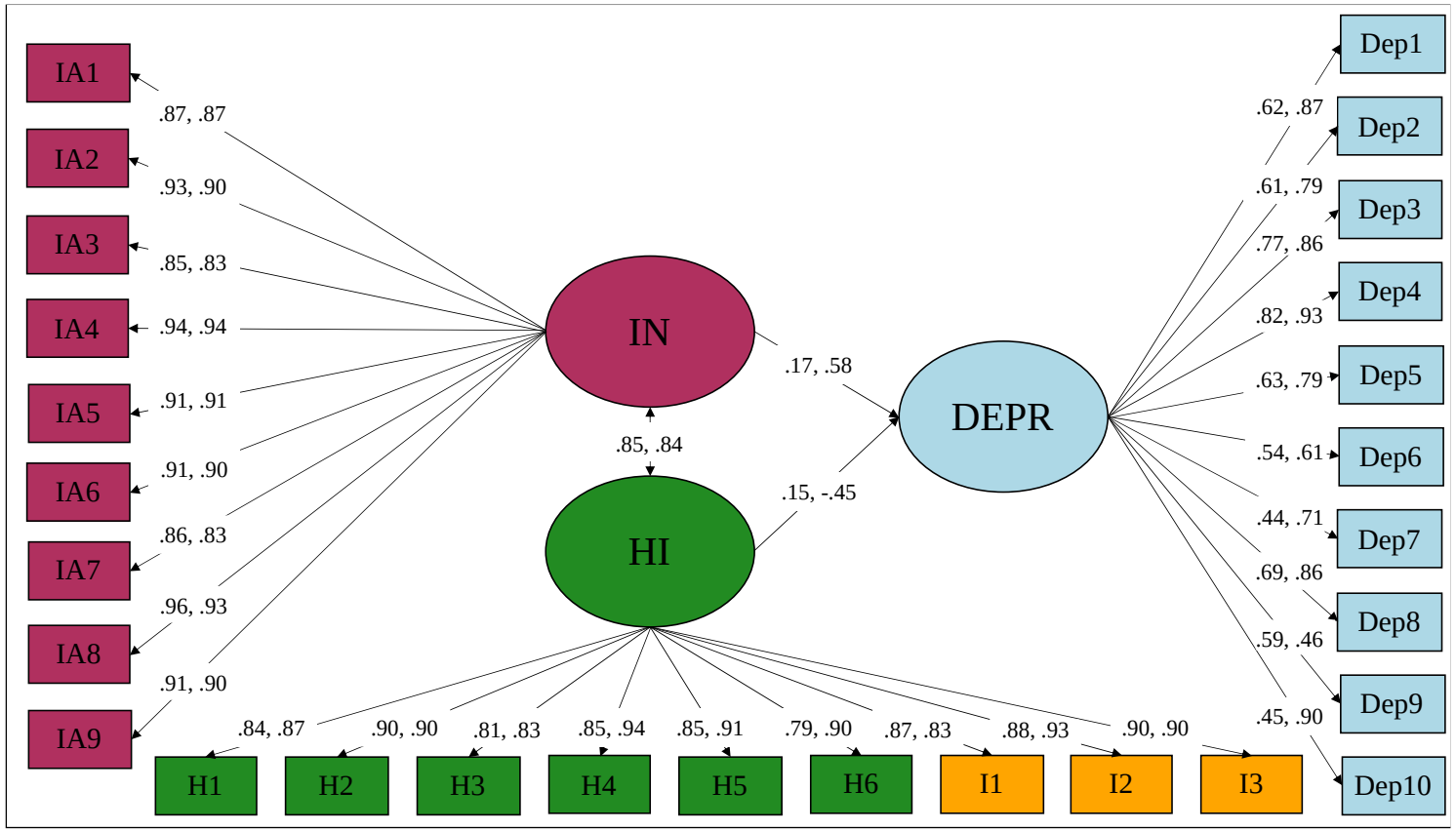
Note. Variables are listed on the y-axes, with BEI z-scores depicted on the x-axis. Values farther to the right indicate that the respective variable demonstrated more robust relations with those of the other community (i.e., ADHD-anxiety; traits-disorders). The top two figures depict BEI with respect to ADHD-anxiety relations, and the bottom two figures depict BEI with respect to trait-disorder relations.

Figure 3.5 Common Cause Model, via SEM, of ADHD-Depression Co-Occurrence



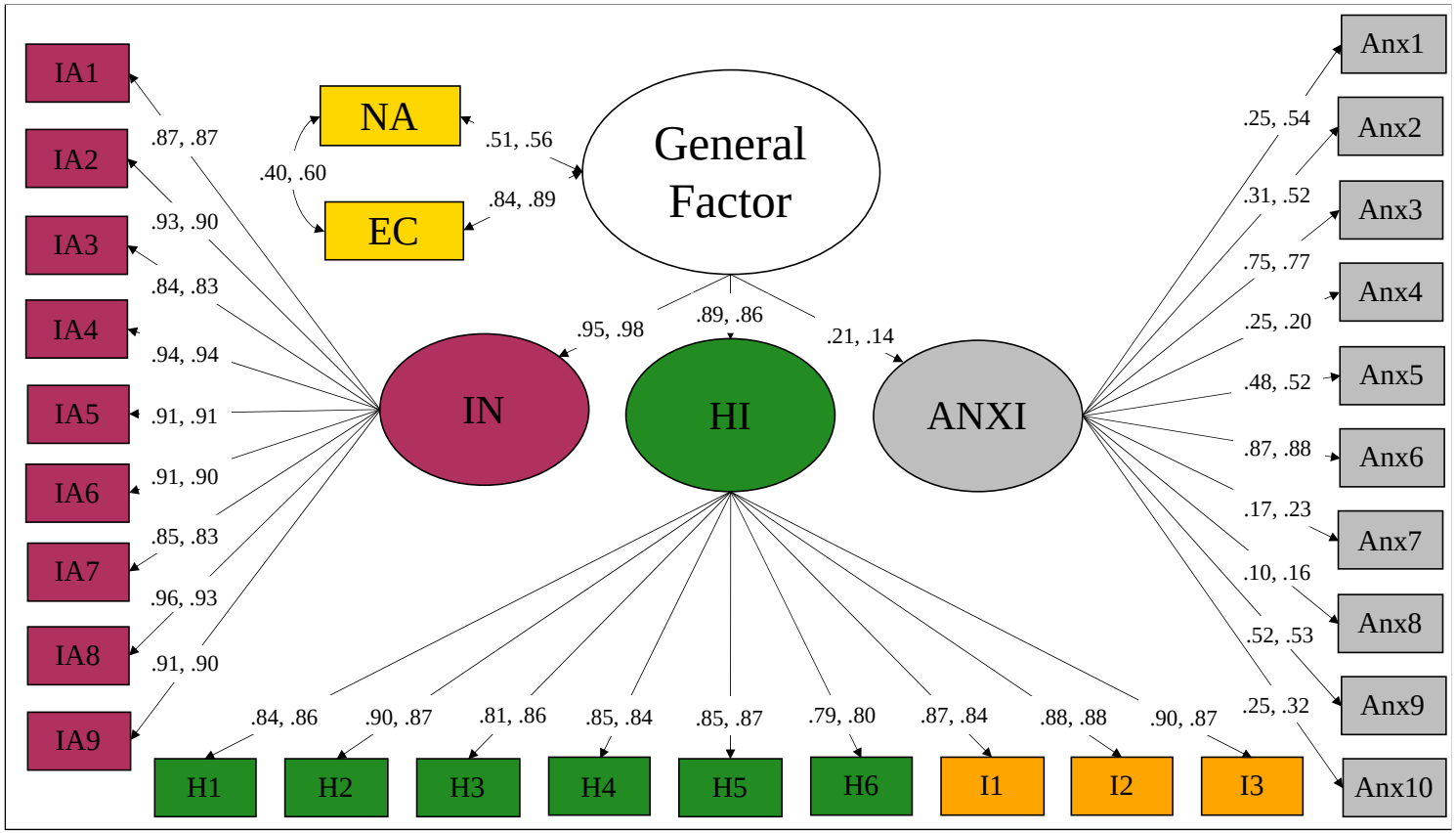
Note. IN = inattention factor; HI = hyperactivity/impulsivity factor; DEPR = depression factor; Dep = depressive symptoms; IA = inattentive symptoms; H = hyperactive symptoms; I = impulsive symptoms; NA = negative affect; EC = effortful control. Loadings and correlations are standardized. All presented statistics were significantly different than zero across years (ps < .05). Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.6 Direct Causation Model, via SEM, of ADHD-Depression Co-Occurrence



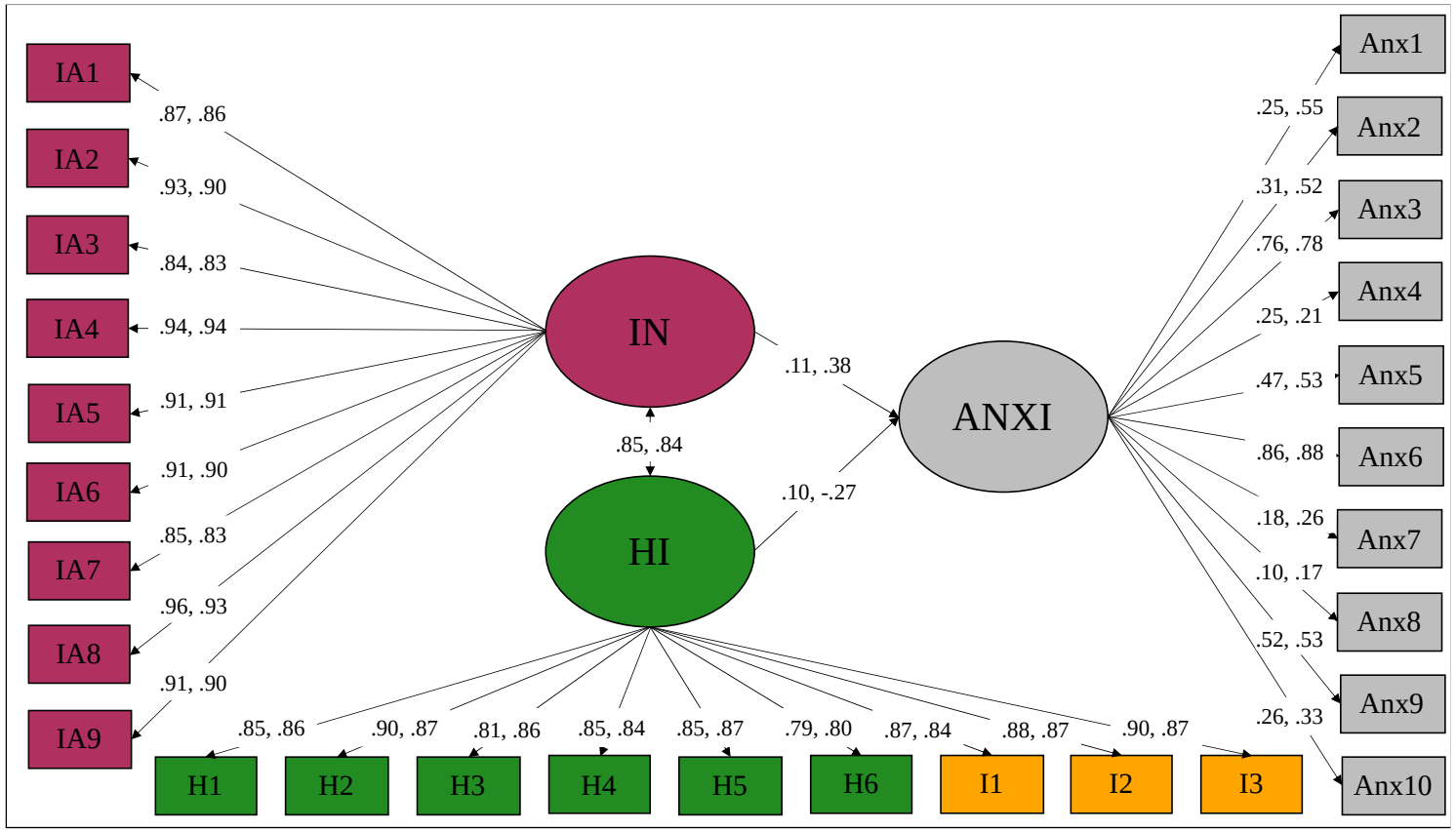
Note. IN = inattention factor; HI = hyperactivity/impulsivity factor; DEPR = depression factor; Dep = depressive symptoms; IA = inattentive symptoms; H = hyperactive symptoms; I = impulsive symptoms; NA = negative affect; EC = effortful control. Loadings and correlations are standardized. Presented statistics were significant across years (*p*s < .05) except for HI's relation with depression in Year 1 (*p* = .11). Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.7 Common Cause Model, via SEM, of ADHD-Anxiety Co-Occurrence



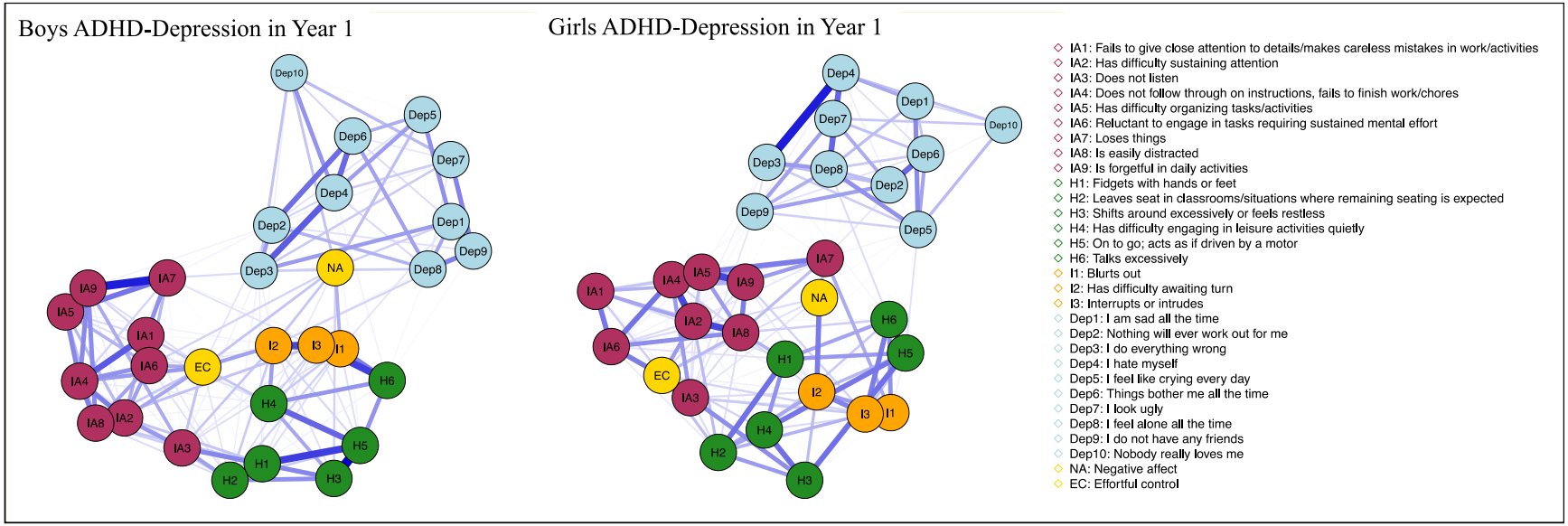
Note. IN = inattention factor; HI = hyperactivity/impulsivity factor; ANXI = anxiety factor; Anx = anxiety symptoms; IA = inattentive symptoms; H = hyperactive symptoms; I = impulsive symptoms; NA = negative affect; EC = effortful control. Loadings and correlations are standardized. All presented statistics were significantly different than zero across years (ps < .05), except "I avoid watching scary movies and TV shows" (Anx8) on the ANXI factor in Year 1 (p = .09). Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.8 Direct Causation Model, via SEM, of ADHD-Anxiety Co-Occurrence



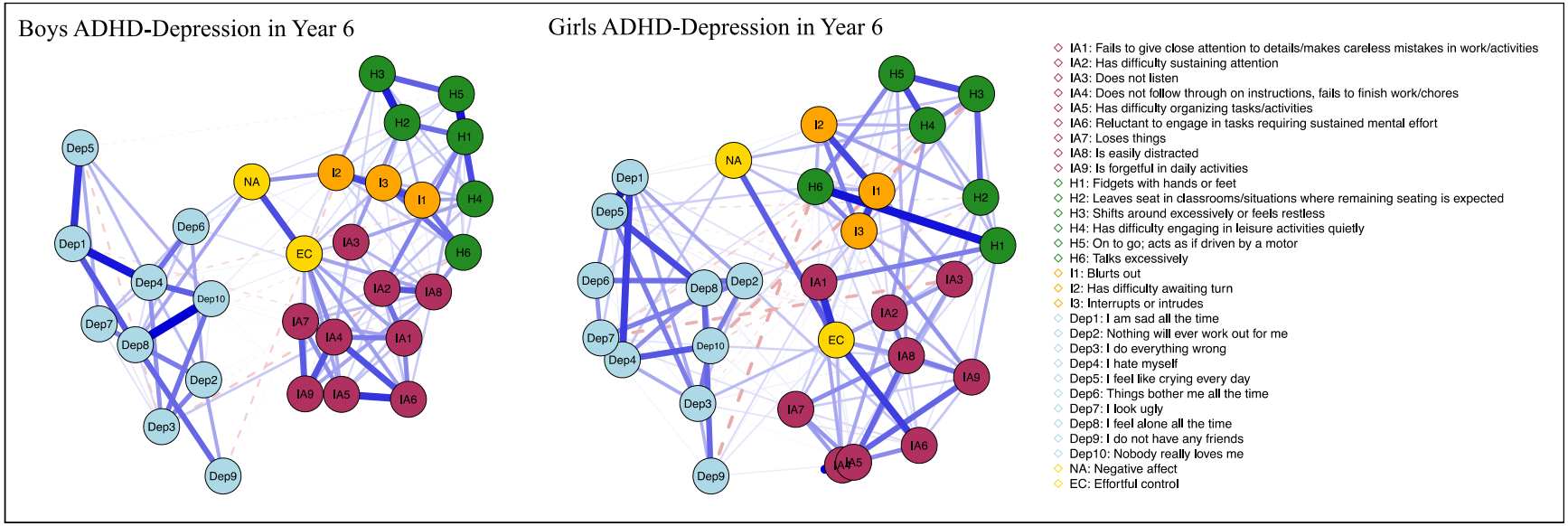
Note. IN = inattention factor; HI = hyperactivity/impulsivity factor; ANXI = anxiety factor; Anx = anxiety symptoms; IA = inattentive symptoms; H = hyperactive symptoms; I = impulsive symptoms; NA = negative affect; EC = effortful control. Loadings and correlations are standardized. Presented statistics were significant across years (*p*s < .05), except "I avoid watching scary movies and TV shows" (Anx8) on the ANXI factor in Year 1 (*p* = .09), as well as IA and HI's relations with ANXI in Year 1 (*p*s ≥ .26). Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.9 ADHD-Depression Networks in Year 1 Separated by Gender



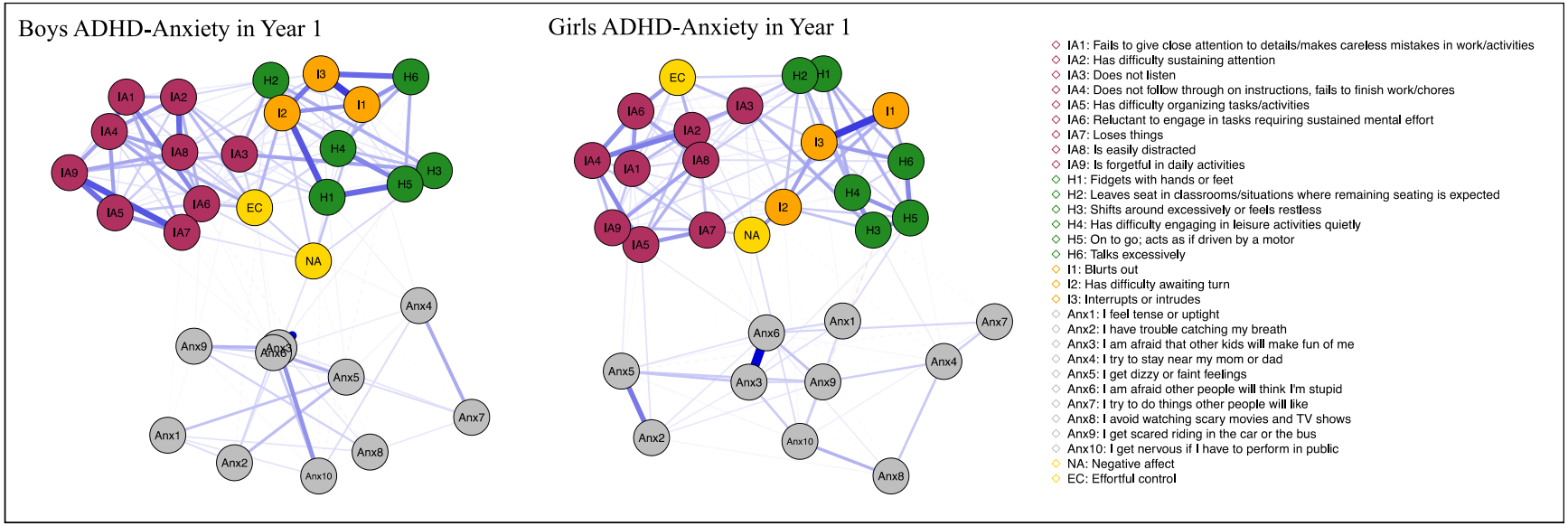
Note. Visualization was created using Multidimensional Scaling. This allows for broad visual interpretations of network structure based on the spacing between variables. Variables are depicted as nodes, with edges connecting these nodes representing regularized partial Spearman correlations. Edge thickness represents the strength of the relation. Solid edges indicate positive relations and dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.10 ADHD-Depression Networks in Year 6 Separated by Gender



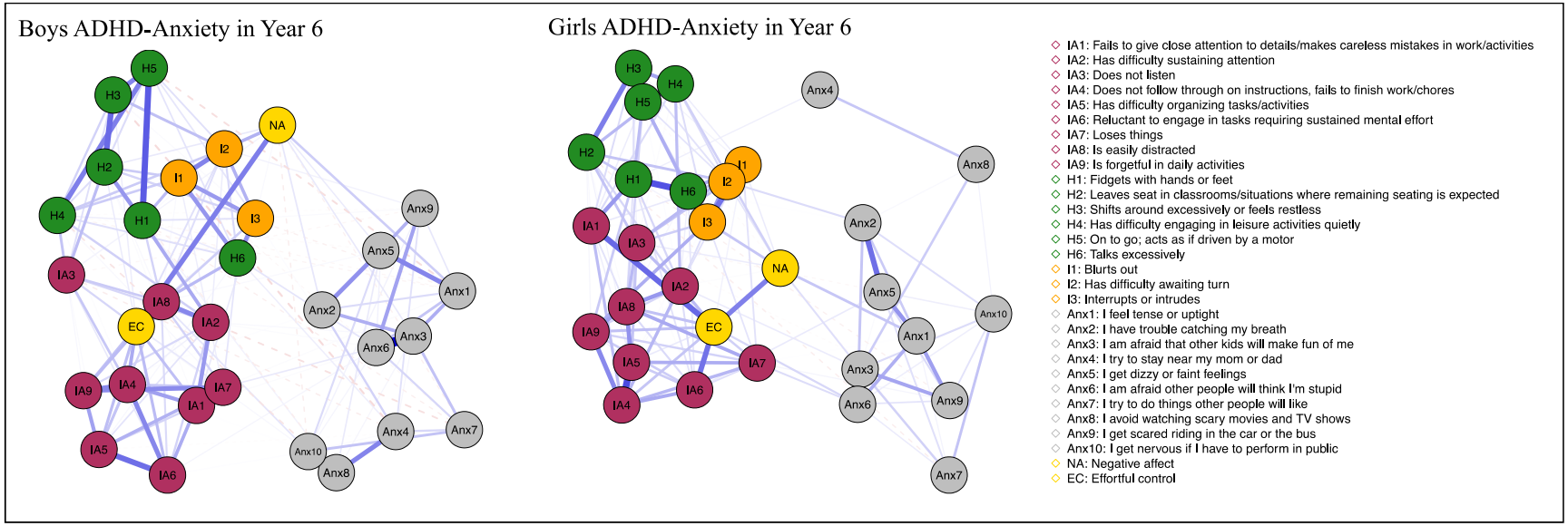
Note. Visualization was created using Multidimensional Scaling. This allows for broad visual interpretations of network structure based on the spacing between variables. Variables are depicted as nodes, with edges connecting these nodes representing regularized partial Spearman correlations. Edge thickness represents the strength of the relation. Solid edges indicate positive relations and dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.11 ADHD-Anxiety Networks in Year 1 Separated by Gender



Note. Visualization was created using Multidimensional Scaling. This allows for broad visual interpretations of network structure based on the spacing between variables. Variables are depicted as nodes, with edges connecting these nodes representing regularized partial Spearman correlations. Edge thickness represents the strength of the relation. Solid edges indicate positive relations and dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

Figure 3.12 ADHD-Anxiety Networks in Year 6 Separated by Gender



Note. Visualization was created using Multidimensional Scaling. This allows for broad visual interpretations of network structure based on the spacing between variables. Variables are depicted as nodes, with edges connecting these nodes representing regularized partial Spearman correlations. Edge thickness represents the strength of the relation. Solid edges indicate positive relations and dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

CHAPTER 4. DISCUSSION

The current study represented the first investigation of a network approach's utility for conceptualizing ADHD-internalizing disorder co-occurrence, specifically with respect to integrating and parsing heterogeneity within temperament-based common cause and direct causation effects. Results added to the existing literature by suggesting both effects as making unique contributions to the characterization of ADHD-internalizing disorder co-occurrence. Regarding common cause effects, low effortful control, while emerging as a transdiagnostic risk marker, appeared to be primarily related to increases in ADHD inattentive bridge symptoms. On the other hand, higher levels of negative affect appeared to demonstrate relations with increased severity of symptoms across disorders (i.e., hyperactive/impulsive symptoms, particularly the bridge symptom *difficulty awaiting turn*, depressive symptoms associated with negative mood and distress, and anxiety symptoms associated with somatic problems and peer-related fears). Simultaneously, unique cross-disorder relations were identified in networks as also explaining the nature of ADHD-internalizing disorder co-occurrence, with follow-up analyses suggesting the ADHD symptoms *loses things, does not follow through, and difficulties sustaining attention*, the depressive symptoms *I do everything wrong* and *I do not have any friends*, and the anxiety symptom *I am afraid that other people will think I'm stupid* as bridge symptoms that played key roles in these relations.

Network-related findings appeared to be generally consistent with and complementary to those of SEM conceptualizations investigating such effects separately. Relations within the network approach were generally robust across measurement point and gender. Overall, results provided support for the utility of the network approach for

parsing heterogeneity in unique trait-symptom and symptom-symptom relations that has generally been inaccessible via SEM conceptualizations. Continued clarification of these relations could ultimately contribute to a better understanding of the multiple mechanisms through which ADHD's co-occurrence with internalizing disorders may occur, as well as inform the creation of screening tools and interventions targeted first at core symptoms within these mechanisms to improve the effectiveness, efficiency, and specificity of current clinical practices.

4.1 Novel Symptom-Level Insights Facilitated by the Network Approach

4.1.1 Common Cause Effects Explaining ADHD-Depression Co-Occurrence

Across measurement points, and in line with hypotheses, both network and SEM conceptualizations suggested low effortful control and high negative affect as transdiagnostic risk markers positively related both to each other and to ADHD and depression. Findings corroborated prior work suggesting these traits as interacting transdiagnostic risk markers of multiple types of psychopathology (Meinzer et al., 2014; Muris et al., 2007; Nigg, 2017; Nigg et al., 2020; Rutter & Arnett, 2020). Additionally, results were consistent with recent work suggesting ADHD and depression as both stemming from a shared liability characterized by disinhibited negative affect (Carver et al., 2017; Forbes et al., 2021), with early assessment of this liability potentially providing early insight into risk for co-occurring depression in youth with ADHD.

Additionally, network analysis appeared to facilitate a novel investigation of heterogeneity in trait-symptom relations, thus providing more specific insights pertaining to how low effortful control and high negative affect may increase risk for co-occurrence.

That is, while low effortful control did demonstrate relations with depressive symptoms associated with decreases in perceived self-competence and social problems (i.e., *nothing will ever work out for me; I do everything wrong; I do not have any friends; nobody really loves me*), such relations were clearly secondary in networks compared to particularly strong relations between low effortful control and increased severity of several inattentive bridge symptoms across years (i.e., *has difficulty sustaining attention; does not follow through; reluctant to engage in tasks requiring sustained mental effort; difficulty awaiting turn*). Such results corroborated recent work suggesting effortful control as a specific indicator of externalizing problems/ADHD after removing overlap with internalizing problems (Shields et al., 2019). Though further longitudinal testing is needed, results were also consistent with the idea that, in the context of ADHD-depression co-occurrence, low effortful control may develop in association with high levels of negative affect and serve primarily as a liability for self-regulation deficits commonly attributed to the ADHD inattentive symptom domain (Gagne & Goldsmith, 2011; Miller et al., 2019; Nigg, 2017; Nigg et al., 2020).

Importantly, such a distinction of effortful control's relations with ADHD and depression was only possible through the network approach, as SEM models suggested effortful control and negative affect as demonstrating relatively robust relations with the general factor. Yet, such a distinction may be critical for specifying the role of low effortful control in ADHD-depression co-occurrence (e.g., contributing mostly to ADHD inattentive bridge symptoms), and ultimately contribute to an improved understanding of when interventions aimed at low effortful control may be effective in clinical practices (e.g., when a child exhibits hyperactive/impulsive symptoms, a focus on effortful control

may not be as useful as when a child exhibits inattentive symptoms). It should be noted that while tests of tautological overlap (i.e., similar wording) between variables included in networks suggested some overlap among symptoms of ADHD, this overlap did not appear to meaningfully impact ADHD trait-symptom relations (see footnote 5). However, given that most bridge symptoms with respect to trait-disorder relations fell within the inattentive symptom domain, continued examination of the effects of shared method variance (i.e., both traits and ADHD symptoms utilized parent-report) and tautological overlap in measures is needed to further explore low effortful control's utility as a liability and potential intervention target primarily for ADHD inattentive bridge symptoms and secondarily for depression via decreases in perceived self-competence and difficulties making friends.

4.1.2 Direct Causation Effects Explaining ADHD-Depression Co-Occurrence

SEM and network approaches both provided support for the existence of direct causation effects, as increases in ADHD severity (particularly inattentive) appeared to be associated with higher levels of depression in general. Results were consistent with hypotheses and provided support for the idea that ADHD-related difficulties in various functional domains, particularly social, may directly contribute to increased risk for the development of subsequent depression (Meinzer et al., 2014; Riglin et al., 2020). Additionally, the network approach appeared to facilitate novel and specific insights into symptoms' unique roles in contributing to such effects, with a few symptoms emerging as particularly noteworthy.

That is, two depressive bridge symptoms emerged in Year 1 (i.e., *I do everything wrong; I do not have any friends*), and three ADHD bridge symptoms were identified in

Year 6 (i.e., *loses things; does not follow through; has difficulty sustaining attention*). Findings pertaining to ADHD symptoms in Year 6 extended upon prior work implicating the inattentive symptom domain in contributing to risk for depression by suggesting that, in adolescence, such effects may best be captured via a focus on ADHD-related difficulties sustaining attention, problems following through, and a tendency to lose things (Meinzer et al., 2014; Riglin et al., 2020). Moreover, follow-up examination suggested that these symptoms' importance derived from relations with specific depressive symptoms, including those associated with low self-worth, social problems, hopelessness, and distress. Results highlighted the most robust means through which ADHD may relate to depression and suggested that interventions aimed at ADHD bridge symptoms, in addition to reducing the severity of ADHD itself, may provide downstream benefits to depressive symptoms, especially those most strongly associated with ADHD during adolescence and, thus, potentially the most important to address first when addressing impairments stemming from the effects of both disorders.

During childhood, ADHD symptoms appeared to demonstrate relatively consistent relations with symptoms of depression. Conversely, depression's relation with ADHD during this period appeared to involve symptoms associated with decreases in perceived self-competency and difficulties making friends. Such findings again highlighted the importance of accounting for symptom-level heterogeneity when examining disorders' relations with etiological factors, risk markers, and external correlates (Fried, 2017; Goh, Martel, et al., 2020; Martel et al., 2020). Results suggested that when children are diagnosed with ADHD, it may also be worthwhile to assess for reductions in self-competency and problems making friends. Such assessment may

provide a relatively straightforward investigation of risk for concurrent depression, with these depressive symptoms also potentially serving as the most prominent intervention targets to reduce the severity of depressive phenotypes that characterize this period of development.

4.2 Common Cause Effects Explaining ADHD-Anxiety Co-Occurrence

In line with hypotheses, results of both network analysis and SEM approaches suggested lower levels of effortful control, and higher levels of negative affect, as associated with increases in inattentive, hyperactive/impulsive, and anxiety symptoms' severity across years. Findings provided support for past work proposing these traits as interacting transdiagnostic risk markers in the context of ADHD-anxiety co-occurrence (De Pauw & Mervielde, 2010; Forbes et al., 2017; Nigg, 2006, 2017; Nigg et al., 2020). Further, exploration of ADHD-anxiety networks appeared to facilitate novel insights into traits' unique relations with specific symptoms of ADHD and anxiety, with results being somewhat consistent with ADHD-depression networks: effortful control exhibited a significantly higher BEI than negative affect which was driven by strong relations with increased severity in almost all ADHD inattentive bridge symptoms but only two anxiety symptoms (*I get dizzy or faint feelings* in Year 1; *I am afraid other people will think I'm stupid* in Year 6). Conversely, increases in negative affect were uniquely associated with increased severity of hyperactive/impulsive symptoms associated with restlessness and impulsivity (including the bridge symptom *difficulty awaiting turn*), anxiety symptoms associated with somatic problems and fear of negative evaluation from peers, and inattentive symptoms associated with difficulties sustaining attention and staying organized. Such findings corroborated recent work exploring the overlap between

disorders, which identified disinhibition as a key risk factor primarily for ADHD and negative affect as a shared liability for ADHD, particularly hyperactivity/impulsivity, and anxiety (Forbes et al., 2021; Martel, 2009).

Hence, as with ADHD-depression co-occurrence, effortful control appeared best characterized as a liability primarily for ADHD inattentive symptoms, with any relations with hyperactive/impulsive and anxiety symptoms being more secondary in nature. On the other hand, high levels of negative affect, while also potentially contributing to problems in the consolidation of effortful control, may serve as a shared liability for ADHD symptoms, particularly the bridge symptom *difficulty awaiting turn*, as well as anxiety symptoms associated with somatic problems and fear of negative evaluation from peers. Results were consistent with the idea that in the context of ADHD-anxiety co-occurrence, interventions aimed at reducing the effects of high levels of negative affect may be a key tool in clinical settings to reduce psychopathology more generally, while those aimed at effortful control may provide benefits mostly to inattentive bridge symptoms of ADHD. Such findings were distinct from those obtained through SEM modeling which suggested more consistent trait-disorder relations, yet such a distinction of risk markers' roles in the etiology of ADHD-anxiety co-occurrence could be crucial in clinical settings for planning treatment to focus on key symptoms that contribute to the characterization of both disorders first.

4.2.1 Direct Causation Effects Explaining ADHD-Anxiety Co-Occurrence

Interestingly, results pertaining to direct causation effects within network and SEM approaches appeared to diverge in Year 1. Specifically, examination networks revealed no significant differences in symptoms' BEI, suggesting that symptoms of

ADHD demonstrated relatively consistent relations with those of anxiety, and vice versa. Such findings fell somewhat in line with prior work suggesting generalized relations between ADHD and anxiety (Baldwin & Dadds, 2008; Becker et al., 2012; Jarrett, 2016), although it should be noted that other research has implicated the inattentive symptom domain as primarily responsible for this relationship (Michelini et al., 2015; Yüce et al., 2013).

Conversely, SEM results in Year 1 suggested neither inattention nor hyperactivity/impulsivity latent factors as significant indicators of the anxiety latent factor. This result contrasted with prior work in children and adolescents suggesting robust relations between ADHD and anxiety (Bowen et al., 2008; Jarrett, 2016; Tai et al., 2013). It may be that the conceptualization of inattention and hyperactivity/impulsivity as separate but highly correlated factors (r = .85) impacted results, with the inclusion of these factors simultaneously in a regression analyses reducing the strength of their unique relations with the anxiety factor due to multicollinearity. Additionally, loadings of some somatic symptoms of anxiety (e.g., *I feel tense or uptight* [Anx1]; *I have trouble catching my breath* [Anx2]), although statistically significant, did not appear to load strongly onto the anxiety factor in Year 1, even though prior work has identified somatic problems as key to the characterization of anxiety during childhood (Crawley et al., 2014). The lack of inclusion of these somatic symptoms in the resulting SEM model may have contributed to weakened associations between ADHD and anxiety latent factors. On the other hand, ADHD-anxiety relations in networks were relatively weak, with further study needed examining the practical relevance of such relations (average regularized partial correlation = .001). Further work is needed to explore the best methodology for

conceptualizing ADHD's relation with anxiety, as well as propose conditions where this relation potentially may not be clinically relevant (i.e., perhaps different demographic groups exhibit differential relations between ADHD and anxiety latent factors).

In Year 6, results of network and SEM approaches supported similar conclusions. That is, in contrast to Year 1 results, SEM findings in Year 6 suggested a significant relationship between higher levels of inattention and anxiety latent factors, consistent with recent work suggesting a strong relationship between these two domains of psychopathology (Michelini et al., 2015; Yüce et al., 2013). This Year 6 result contrasting with that of Year 1 (where no relationships were identified) may have resulted, in part, because the two somatic symptoms of anxiety noted above (e.g., *I feel tense or uptight*; *I have trouble catching my breath*) loaded notably more strongly on the anxiety factor in Year 6. It is possible that these somatic symptoms may thus play a key role in the conceptualization of ADHD's relationship with anxiety, although further work is needed exploring this idea. Pertaining to results of network analysis, the inattentive symptom *loses things* and the anxiety symptom *I am afraid that other people will think I'm stupid* emerged as bridge symptoms. Findings corroborated prior work highlighting relations between the inattentive symptom domain and anxiety-related intrusive worries (Jarrett & Ollendick, 2008), with results of the network approach suggesting an ADHD-related tendency to lose things and anxiety-related fears regarding negative peer self-evaluation as notable contributors in explaining these relations. Pertaining to differences between Year 1 and 6, it should be noted that MASC scores did not notably differ between measurement points (Year 1: M = 12.61; Year 6: M = 12.44), although there appeared to be somewhat higher rates of anxiety diagnoses in Year 1 (~15%) versus Year

6 (~10%) in the larger study. Further work is needed to explore the effects of these differences, including in samples with higher rates of anxiety diagnoses.

4.3 Complementary Findings Across Network and SEM Conceptualizations

In addition to differences pertaining to Year 1 ADHD-anxiety direct causation effects described above, network and SEM approaches also suggested some other differences in findings, thus highlighting the utility of both models for facilitating complementary insights into ADHD-internalizing disorder co-occurrence. For instance, in ADHD-depression networks, results suggested low effortful control as uniquely related to increases in (primarily) bridge inattentive and (secondarily) depressive symptoms, and high levels of negative affect as related to increased severity of hyperactive/impulsive and depressive symptoms (particularly the bridge symptom *difficulty awaiting turn*). Conversely, SEM results suggested inattentive, hyperactive/impulsive, and (less so) depression latent factors as loading onto the general factor, with this general factor being significantly correlated with both low effortful control and negative affect. Given SEM's focus on common variance between disorder factors, it is possible that effortful control and negative affect may demonstrate *some* relation with almost all symptoms of both ADHD and depression. However, the network approach focused on identifying the strongest unique relations between traits and ADHD, and it was only once commonalities were "partialed out" that key distinct relations between low effortful control and inattentive bridge symptoms, as well as between negative affect and hyperactive/impulsive and depressive symptoms, could be revealed. Further work is needed to determine the nature of these relations, particularly considering the use of partial correlations in networks. It may be that such relations reflect the strongest

associations involving specific symptoms of ADHD and depression that persist after accounting for all other symptoms (e.g., the relationship between effortful control and *difficulty sustaining attention* persisting even after accounting for all other symptoms). Conversely, such relations may reflect particularly unique (but not necessarily strong) associations between certain symptoms and traits that do not overlap with those involving other symptoms (negative affect demonstrating conceptually unique relations with *I get dizzy or faint feelings* and *has difficulty awaiting turn*). Such conclusions, despite both being valuable, have different implications regarding the nature of co-occurrence, so additional study is needed to distinguish between them and determine their validity in networks.

Another instance of complementary findings pertains to direct causation effects across ADHD-depression and ADHD-anxiety analyses. That is, SEM results revealed a negative relation between the hyperactive/impulsive symptom domain and internalizing disorders in Year 6. Although also present in networks, such a finding was not necessarily a focus given initial network-related hypotheses centered on identifying positive cross-disorder relations. Yet, such negative relations were consistent with prior studies suggesting a subset of children with ADHD, particularly those exhibiting behavior problems and aggression related to the hyperactive/impulsive symptom domain, who demonstrate positive illusory self-perceptions particularly with respect to competencies in various functional domains (Bourchtein et al., 2017). These biases, although somewhat protective against internalizing disorders in the short term, have been found to contribute in the long term to poorer interpersonal skills, higher rates of aggression, and increased risky behaviors (Martin et al., 2019). Hence, additional work is

needed to determine whether such biases may serve as a useful target of intervention against impairment in the long term.

4.4 Network Robustness Across Measurement Point and Gender

In contrast to results described above suggesting different bridge symptoms in Year 1 versus 6, as well as hypotheses, results of the network comparison test suggested virtually no significant differences in symptoms' BEI values across measurement point and gender. However, these BEI-focused comparison analyses were likely underpowered and unstable, given that comparison of Year 1 with Year 6 networks could only be conducted in participants with complete data due to the requirements of the Network Comparison Test. Similarly, comparison of networks in boys versus girls necessitated that the sample be split almost in half. Hence, additional work is needed in larger samples to explore whether symptoms' relevance in networks change based on age, gender, and other relevant factors.

Conversely, bootstrapping analyses suggested that the *relations* between variables in networks were stable, with results suggesting respective relations between traits and symptoms were robustly correlated and similar in strength. It should be noted that the NCT applied a relatively strict family detection rate correction when investigating individual relations, so it is possible that some meaningful differences in relations were missed. The NCT also has the option to apply no statistical correction, but this would likely have led to false positive results. Hence, though results should be interpreted with caution due to limited statistical power, they provided preliminary support for the idea that though ADHD and internalizing disorder phenotypes may differ based on age and gender (Franke et al., 2018; Kessler et al., 2012; Kwong et al., 2019; Leopold et al., 2016;

Lijster et al., 2017; Salk et al., 2017; Skogli et al., 2013; Willcutt et al., 2012; Zahn-Waxler et al., 2008), relations among temperament traits, symptoms of ADHD, and symptoms of internalizing disorders may be relatively consistent, with a few isolated edge weights not impacting the overall NCT but leading to some age-related differences in bridge symptoms (i.e., *leaves seat in classrooms/situations where remaining seating is expected* being more related to traits in childhood versus adolescence).

4.5 Implications of Integrating Network and SEM Approaches

Overall, results across network and SEM approaches suggested that common cause and direct causation effects may both be relevant and thus important to account for simultaneously when conceptualizing the nature of internalizing disorders in youth with ADHD. Additionally, network analyses provided support for the idea that common cause and direct causation effects may uniquely and additively contribute to co-occurrence phenotypes through specific symptom-symptom and trait-symptom relations, with a continued focus on these relations potentially highlighting avenues through which ADHD and internalizing disorders may be most efficiently assessed for and treated.

It is interesting that in SEM models, good fit was only achieved when a second-order model was tested (i.e., symptoms loading onto disorder latent factors, with these factors, in turn, loading onto a general factor). Conversely, the model where all symptoms of ADHD and internalizing disorders were specified as loading onto a general factor did not exhibit good fit. This finding fell in line with recent studies examining the overlap between different types of psychopathology using similar statistical methodology (Forbes et al., 2021), and suggested that there may not be enough overlapping information in ADHD and internalizing disorder symptoms to accurately coalesce within

a general overarching factor (as evidenced by the wide range of factor loadings of symptoms on anxiety and depression factors in SEM models). Instead, results suggested that the overlap between ADHD and internalizing disorders may be best conceptualized at the disorder level, with the coalescing of overlap in disorders' symptoms separately serving as an important first step before examining cross-disorder overlap. Given the idea that there may be some meaningful distinction between disorders at the symptom level, future studies should seek to explore other conceptualizations of co-occurrence (e.g., bifactor models) to potentially parse the nature of such overlap/distinction between symptoms of different disorders.

Clinically, an integrative model derived from the network approach may also contribute to the development of novel assessments and treatments that account for risk of co-occurring internalizing disorders in those with ADHD. That is, during assessments of ADHD in childhood, screening tools may be enhanced by placing an additional focus on reductions in perceived self-competence and difficulties making friends to assess for concurrent depression risk. Alternatively, during adolescence, inattentive ADHD bridge symptoms may serve as the most efficient indicators of risk for internalizing disorders, particularly as related to low self-worth, difficulties making friends, and reductions in perceived self-competency (depression), as well as negative evaluation from peers (anxiety). Further, as described above, a continued focus on the strongest relations between disorders and their shared mechanisms may provide an avenue to identify specific targets for personalized interventions that can be adapted based on ADHD- and temperament-based phenotypes (i.e., "goodness of fit"; McClowry et al., 2008).

Crucially, by providing a means to identify the strongest unique common cause and direct causation effects, the network approach has the potential to facilitate the development of more focused interventions that could be adjusted based on individual clients' symptom profiles. For instance, instead of intervening on a general and ambiguous relationship between ADHD and depression, clinicians could focus on a particularly robust relationship between the bridge symptoms *difficulties following through on instructions* (ADHD) and *low self-worth* (depression), thus providing personalized interventions based on a client's most impairing symptoms. Further, it could be interesting to investigate the utility of interventions focused on adjusting the structure of the co-occurrence network itself. Currently, interventions for psychological disorders are primarily aimed at reducing the severity of symptoms over time. However, clarifying relations among symptoms through network analysis opens a novel avenue for interventions to focus on weakening the strength of *relations between symptoms* as well. For instance, networks highlighted a relation between the ADHD symptom *talking excessively* and the anxiety symptom *I am afraid that other people will think I'm stupid.* Hence, while training on non-verbal cues may help youth with ADHD reduce the likelihood of *talking excessively,* network findings also suggested that cognitive strategies aimed at weakening the likelihood that excessive talking contributes to fear of negative peer evaluation may also provide novel and unique benefits. Notably, such an intervention focused on symptom-symptom relations would likely be easier to personalize, implement, and adjust compared to a broader and likely more cumbersome intervention aimed at a more general relationship between disorders.

4.6 Limitations and Future Directions

There were some limitations in the current study. Given the use of cross-sectional data, directional conclusions pertaining to trait-disorder and ADHD-internalizing disorder relations remain primarily based on theory and in need of further longitudinal and experimental testing (Goh & Martel, 2021). Sample sizes, although relatively large for a clinical study of ADHD, were somewhat small for the constructed networks, and likely contributed to limited power to test for network-related differences across gender and measurement point. Future studies should thus utilize larger samples to examine moderation effects of gender, age, and other relevant variables. As noted above, one recent concern with respect to network theory pertains to tautological overlap among items (Fried & Cramer, 2017). Although this overlap did not appear to notably affect results of the current study (see footnote 5), further research is needed to examine redundancy among symptoms and traits as a potential artifact requiring revision in future measures. Several depression and anxiety symptoms exhibited a positive skew in responses due to the larger study's focus on ADHD and various exclusion criteria (e.g., non-stimulant psychotropic medication), so future studies should seek to include samples overrecruited for internalizing disorders *and* ADHD to fully explore these disorders' co-occurrence. Some participants (Year 1: n = 187; Year 6: n = 73) fell outside the recommended age ranges for administration as pertaining to the TMCQ (7-10 years) and EATQ-R (9-15 years). These participants were still included to preserve statistical power, yet future studies should seek to validate results in samples falling in validated age ranges or using appropriate measures (as well as current DSM-5 ADHD symptoms). The inclusion of both self- and parent/caregiver-report measures is a strength of the current study, but additional research is needed using different measures to assess rater effects.

Samples were also relatively high functioning (average FSIQ Year 1: 112.56; Year 6: 112.01), underrepresented groups identifying as minorities, and had a higher reported median household income compared to the U.S. 2010 Census (http://www.census.gov). Future studies should seek to replicate results using larger nationally representative samples to ensure generalizability of results.

4.7 Conclusion

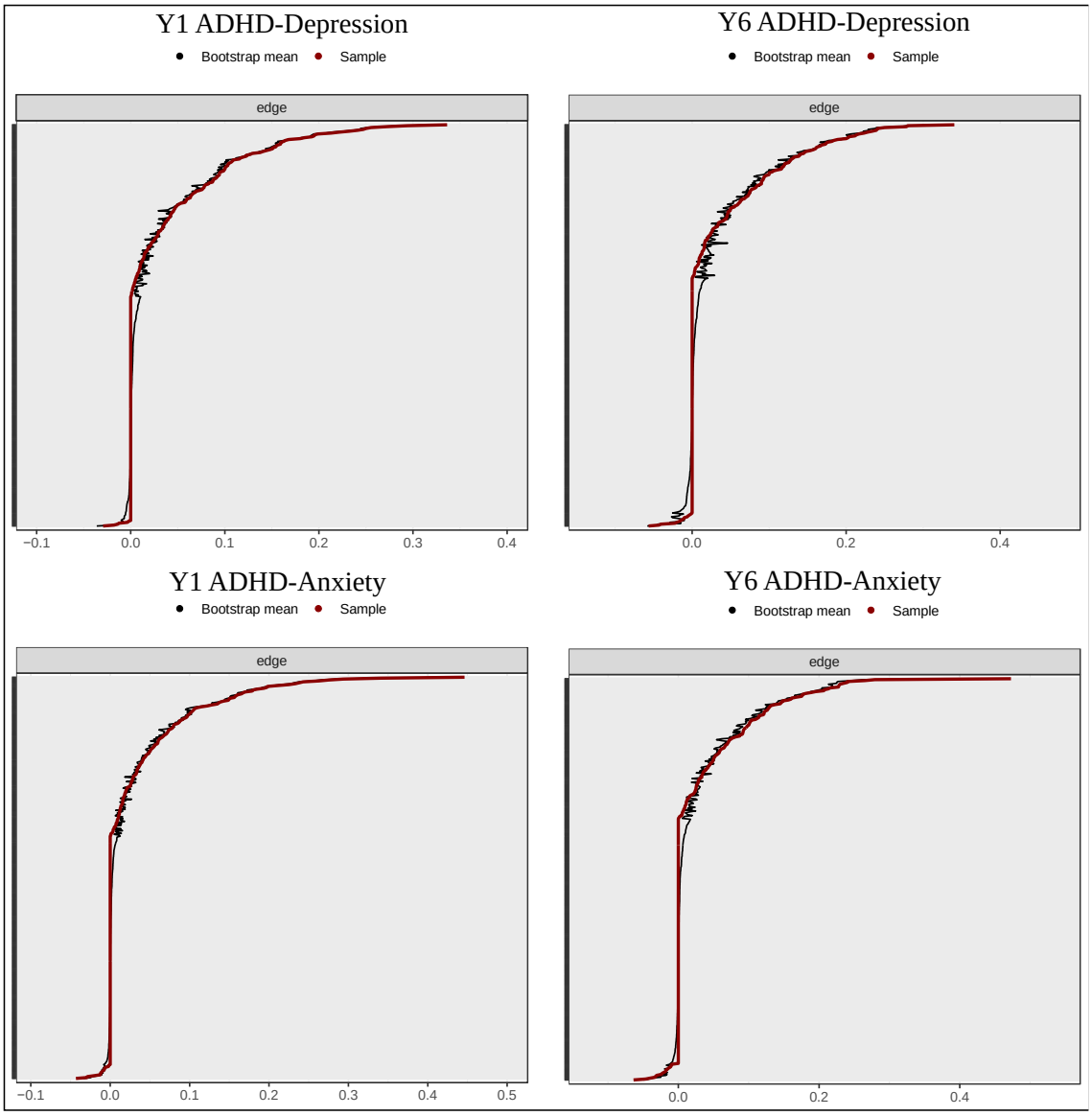
The current study represented the first attempt to integrate and parse trait-based common cause and direct causation effects underlying ADHD-internalizing disorder co-occurrence using a network approach. Results suggested this approach yielded insights generally consistent with and complementary to those obtained through prior SEM approaches, with the network approach facilitating a highlighting of key unique relations among disorders and traits that may be particularly relevant for co-occurrence's conceptualization, assessment, and treatment. Further research to confirm these relations and explore the role of other shared risk markers is needed, as this work could promote insights into the nature of co-occurrence while informing innovative assessment and intervention tools targeted at the most relevant mechanisms.

APPENDICES

APPENDIX 1. METHODS USED TO ASSESS ACCURACY OF EDGE WEIGHTS

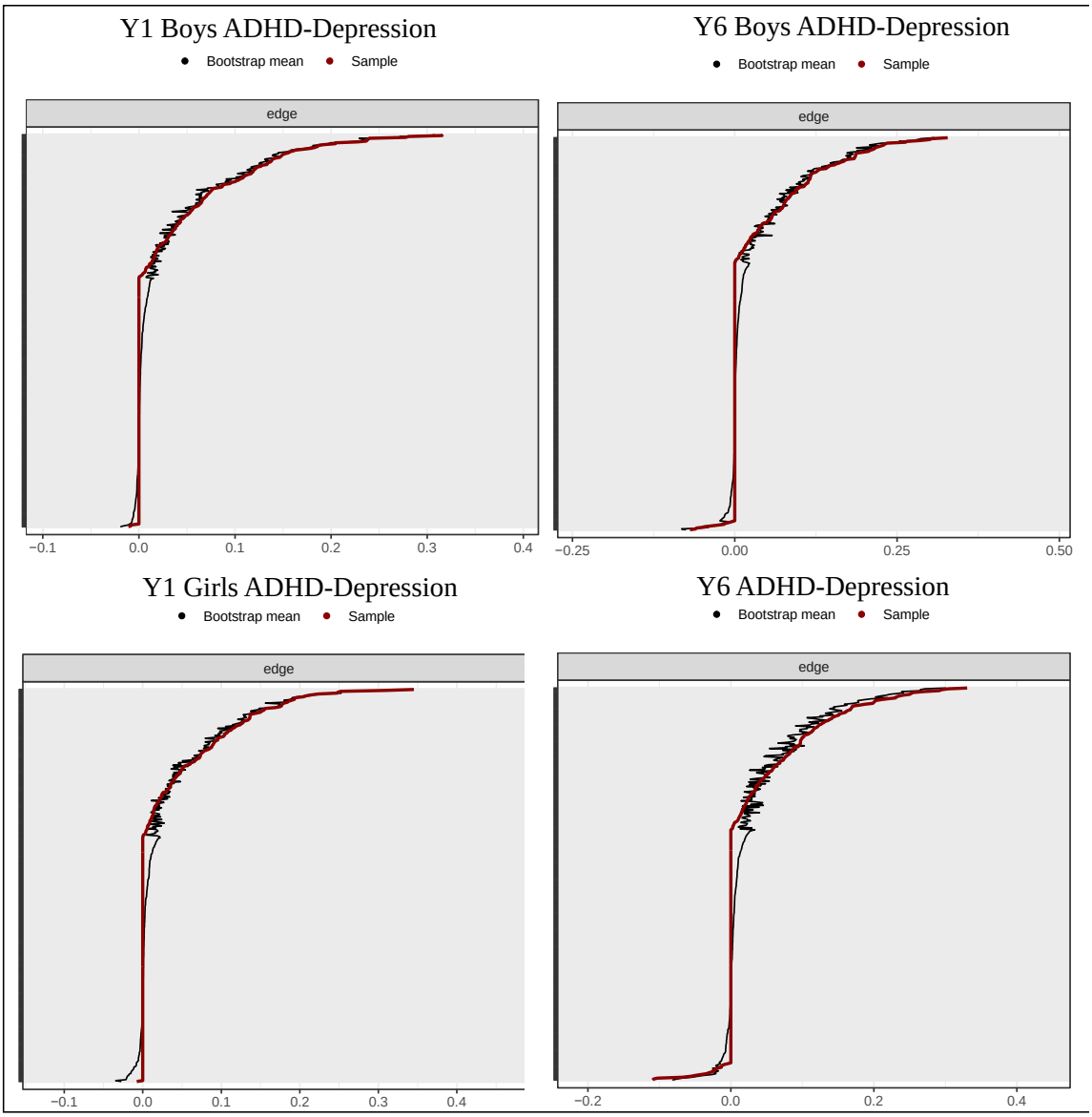
The accuracy of relations (i.e., regularized partial correlations) among variables was assessed using a non-parametric bootstrap approach. This approach involved the calculation of 95% confidence intervals (CIs) for all relations by resampling the data, with replacement, 2000 times, with the resulting CIs forming a distribution of the regularized partial correlation coefficients among variables. This distribution was then qualitatively compared with that of past network analysis studies to determine whether relations within a network was stable. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018), and repeated for all networks.

APPENDIX 2. EDGE STABILITY IN ADHD-DEPRESSION AND ADHD-ANXIETY NETWORKS IN YEARS 1 AND 6



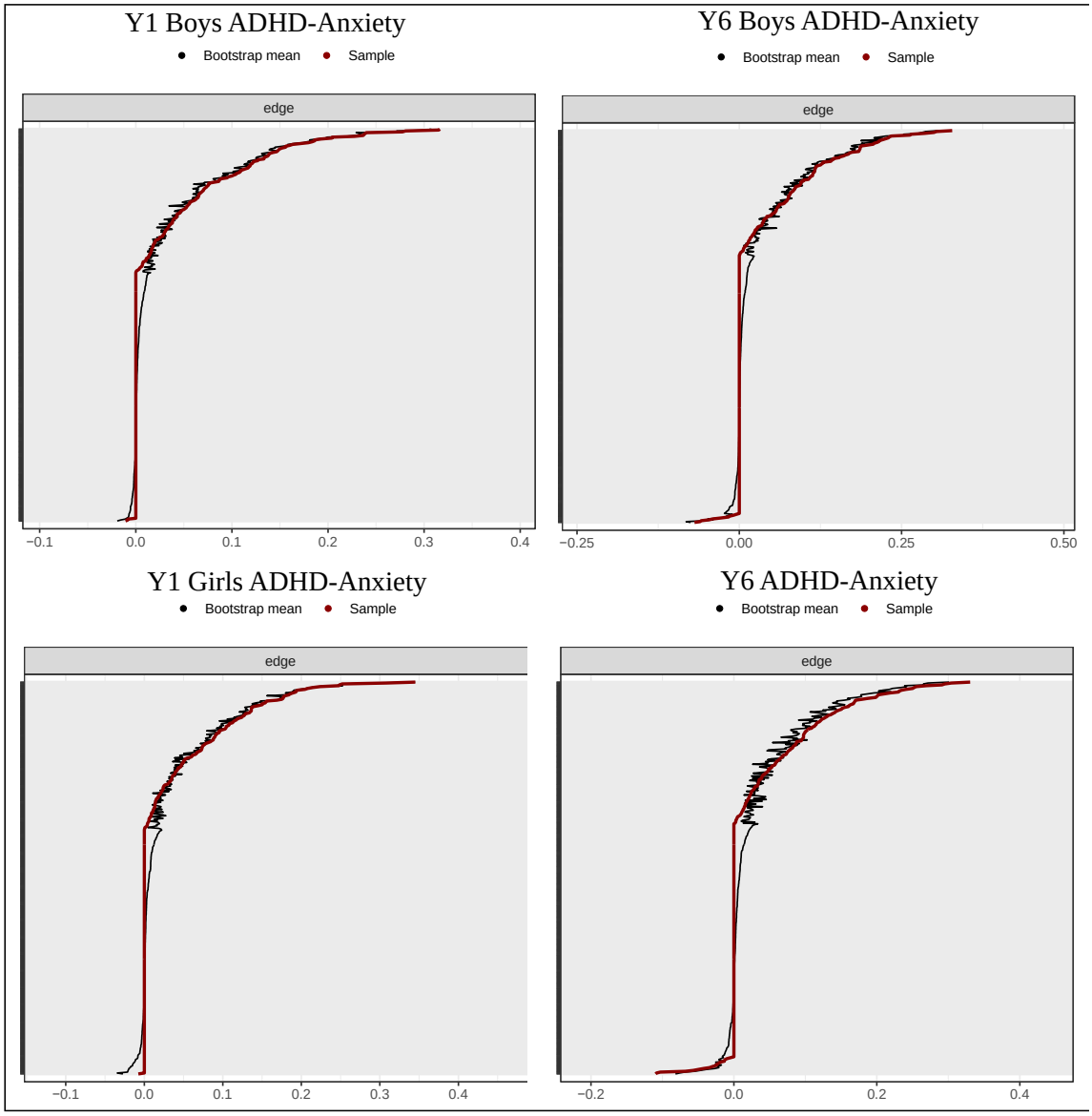
Note. The red line indicates the sample value, with the black dots indicating the bootstrapped means for each relation. The gray area indicated the 95% confidence intervals. Visually, results appeared similar to those from prior work applying network theory to other psychological constructs (e.g., Heeren, Jones, & McNally, 2018, Fried et al., 2018), and suggested that some relations within networks exhibited a strength that was significantly different from zero.

APPENDIX 3. EDGE STABILITY IN ADHD-DEPRESSION NETWORKS IN YEARS 1 AND 6 SEPARATED BY GENDER



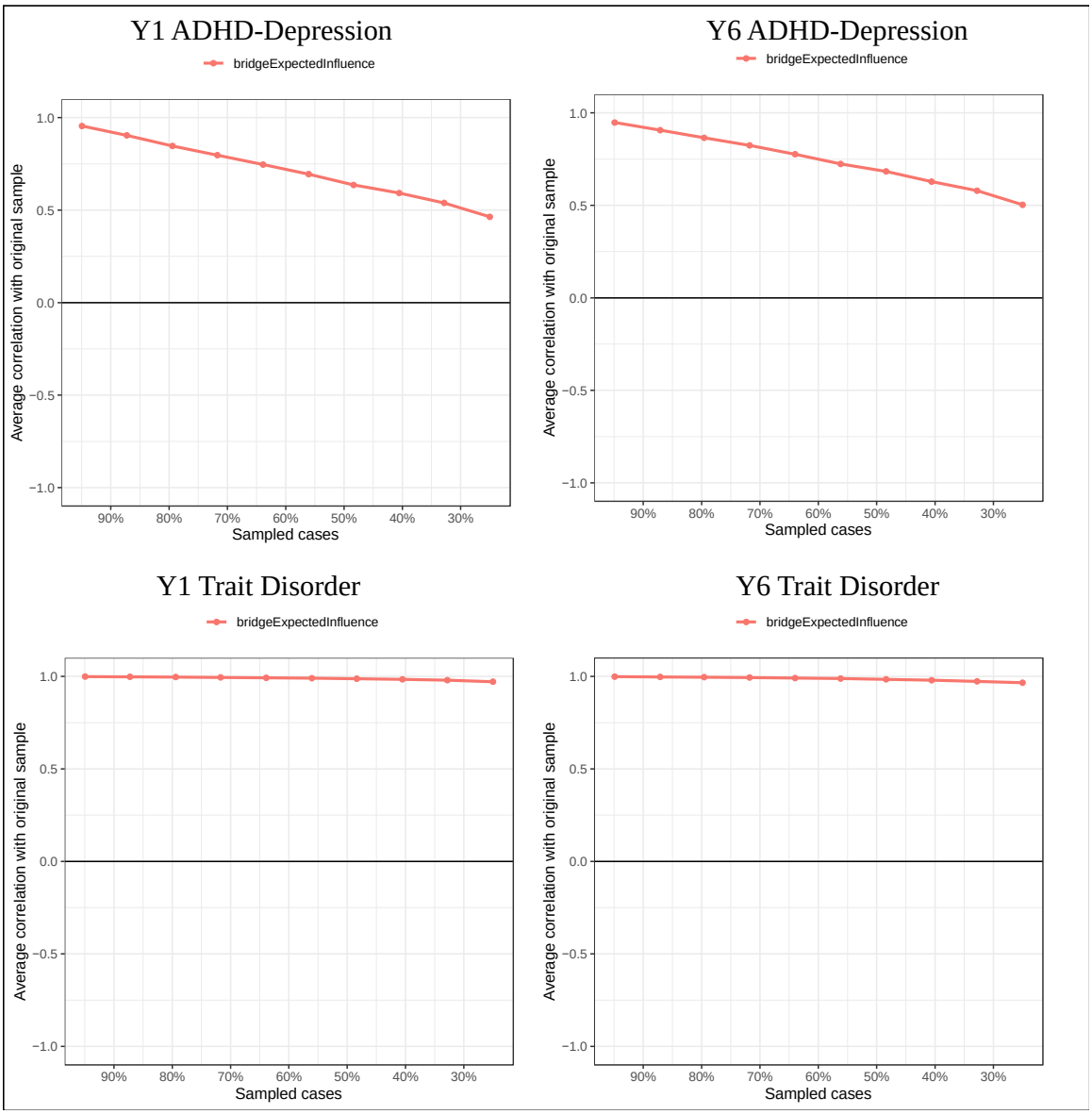
Note. The red line indicates the sample value, with the black dots indicating the bootstrapped means for each relation. The gray area indicated the 95% confidence intervals. Visually, results appeared similar to those from prior work applying network theory to other psychological constructs (e.g., Heeren, Jones, & McNally, 2018, Fried et al., 2018), and suggested that some relations within networks exhibited a strength that was significantly different from zero.

APPENDIX 4. EDGE STABILITY IN ADHD-ANXIETY NETWORKS IN YEARS 1 AND 6 SEPARATED BY GENDER



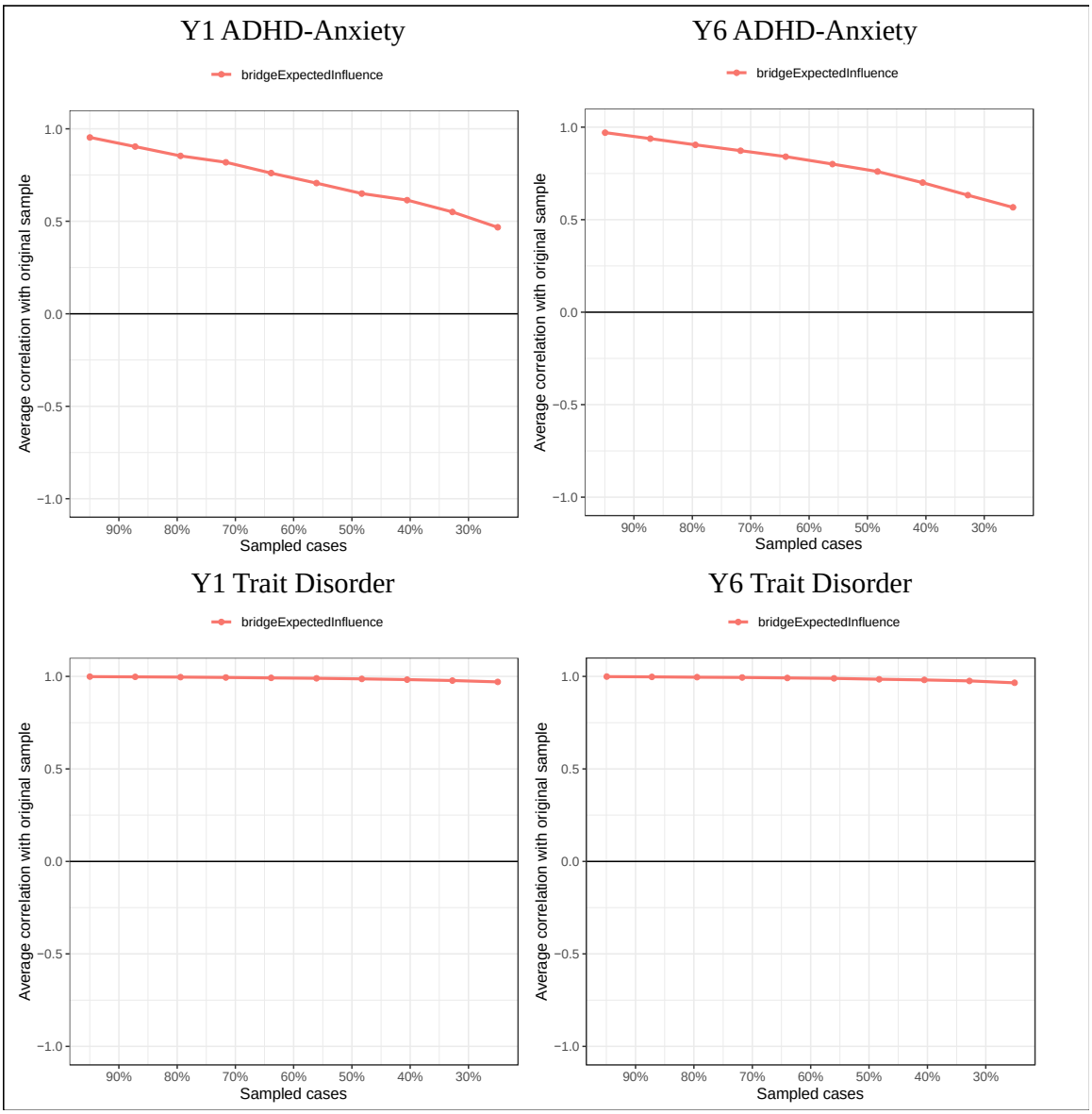
Note. The red line indicates the sample value, with the black dots indicating the bootstrapped means for each relation. The gray area indicated the 95% confidence intervals. Visually, results appeared similar to those from prior work applying network theory to other psychological constructs (e.g., Heeren, Jones, & McNally, 2018, Fried et al., 2018), and suggested that some relations within networks exhibited a strength that was significantly different from zero.

APPENDIX 5. RESULTS OF CASE-DROPPING BOOTSTRAP FOR ADHD-DEPRESSION NETWORKS IN THE OVERALL SAMPLE



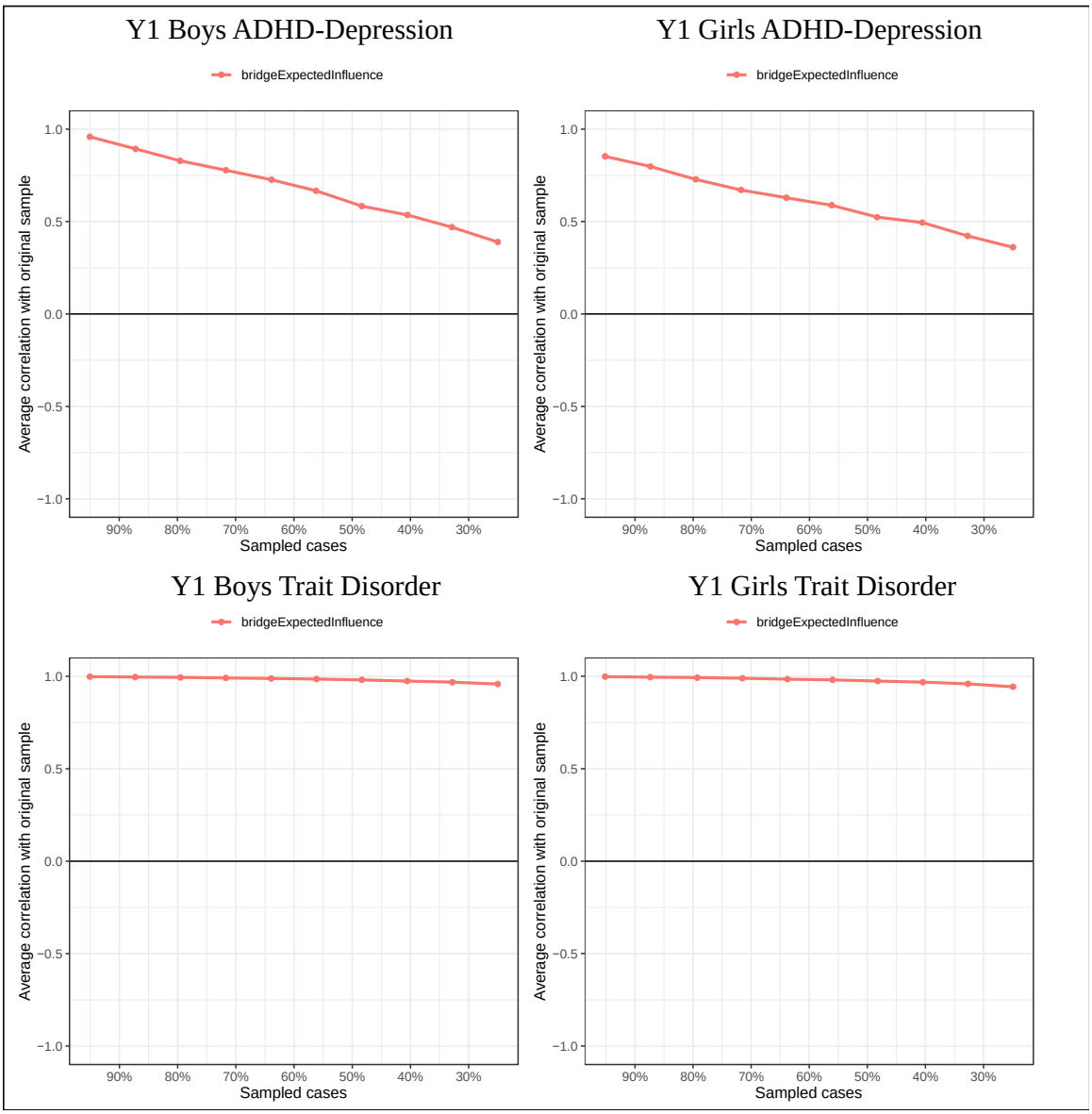
Note. Points represent the correlation between BEI indices in the new sample (after a percentage of cases have been dropped) and original sample. Assessment of the correlation-stability coefficient index (i.e., the proportion of cases that could be eliminated while retaining a correlation of at least 0.70 with the original sample's BEI estimates within a 95% confidence interval; Epskamp & Fried, 2018) suggested adequate stability (i.e., CS > .25; Epskamp & Fried, 2018) in BEI values (CSs ≥ .28). The top two figures depict BEI with respect to ADHD-depression relations, and the bottom two figures depict BEI with respect to trait-disorder relations within ADHD-depression networks.

APPENDIX 6. RESULTS OF CASE-DROPPING BOOTSTRAP FOR ADHD-ANXIETY NETWORKS IN THE OVERALL SAMPLE



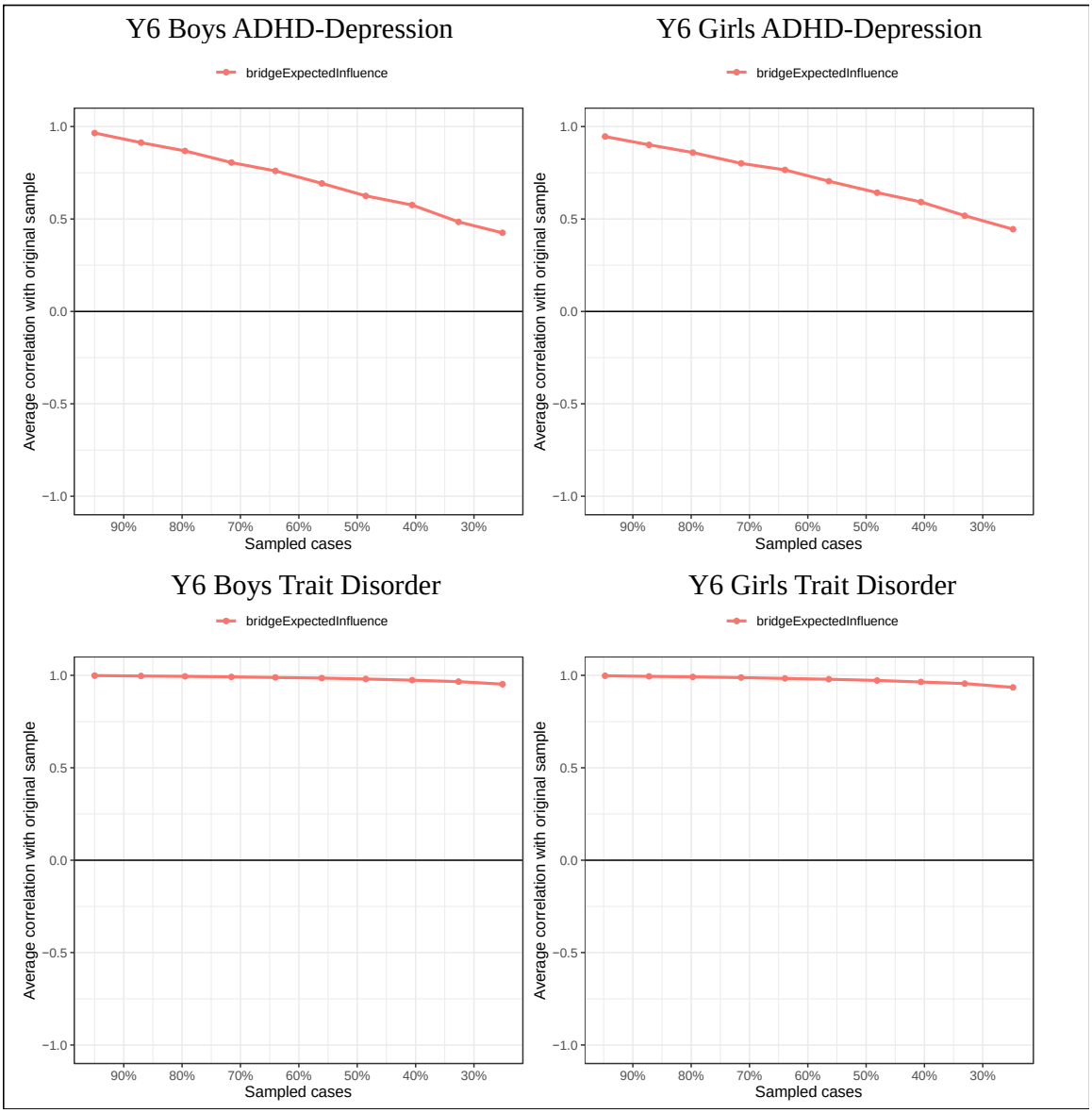
Note. Points represent the correlation between BEI indices in the new sample (after a percentage of cases have been dropped) and original sample. Assessment of the correlation-stability coefficient index (i.e., the proportion of cases that could be eliminated while retaining a correlation of at least 0.70 with the original sample's BEI estimates within a 95% confidence interval; Epskamp & Fried, 2018) suggested adequate stability (i.e., CS > .25; Epskamp & Fried, 2018) in BEI values (CSs ≥ .28). The top two figures depict BEI with respect to ADHD-anxiety relations, and the bottom two figures depict BEI with respect to trait-disorder relations within ADHD-anxiety networks.

APPENDIX 7. RESULTS OF CASE-DROPPING BOOTSTRAP FOR YEAR 1 ADHD-DEPRESSION NETWORKS SEPARATED BY GENDER



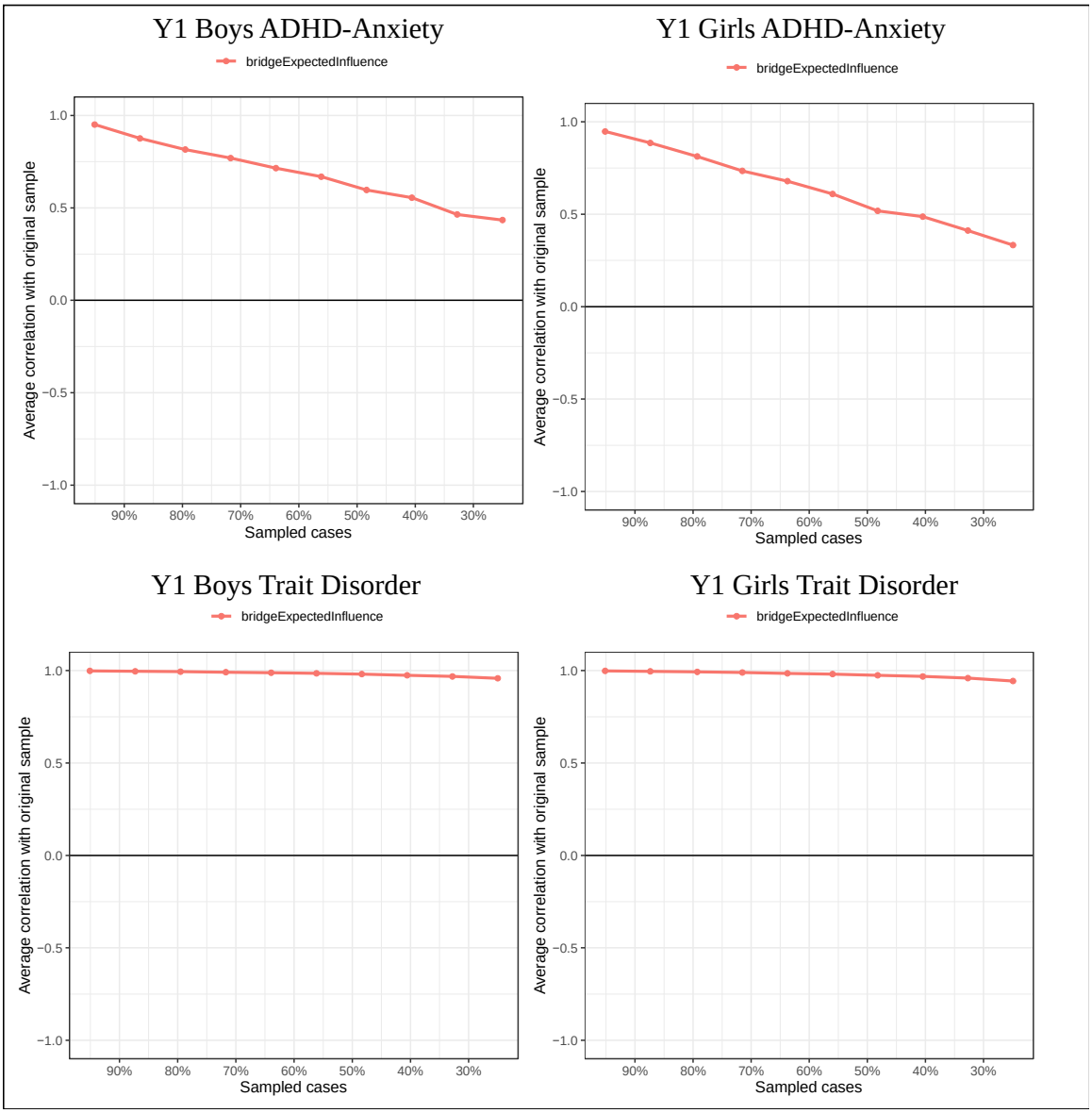
Note. Points represent the correlation between BEI indices in the new sample (after a percentage of cases have been dropped) and original sample. Assessment of the correlation-stability coefficient index (i.e., the proportion of cases that could be eliminated while retaining a correlation of at least 0.70 with the original sample's BEI estimates within a 95% confidence interval; Epskamp & Fried, 2018) suggested adequate stability (i.e., CS > .25; Epskamp & Fried, 2018) in BEI values with respect to trait-disorder relations (CSs ≥ .75), but not ADHD-depression relations (CSs ≤ .13), across years. The top two figures depict BEI with respect to ADHD-depression relations, and the bottom two figures depict BEI with respect to trait-disorder relations within ADHD-depression networks.

APPENDIX 8. RESULTS OF CASE-DROPPING BOOTSTRAP FOR YEAR 6 ADHD-DEPRESSION NETWORKS SEPARATED BY GENDER



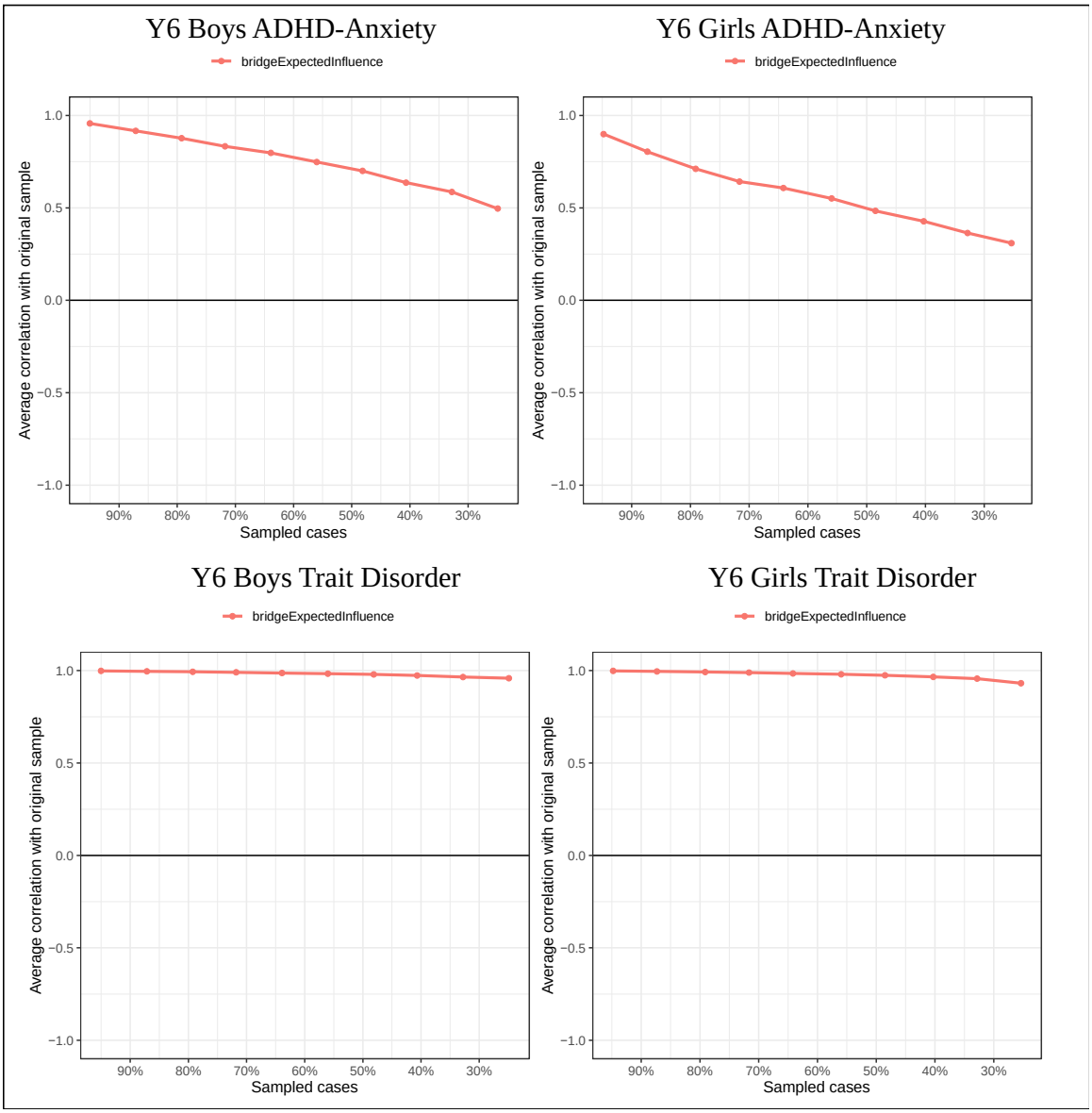
Note. Points represent the correlation between BEI indices in the new sample (after a percentage of cases have been dropped) and original sample. Assessment of the correlation-stability coefficient index (i.e., the proportion of cases that could be eliminated while retaining a correlation of at least 0.70 with the original sample's BEI estimates within a 95% confidence interval; Epskamp & Fried, 2018) suggested adequate stability (i.e., CS > .25; Epskamp & Fried, 2018) in BEI values with respect to trait-disorder relations (CSs ≥ .67), but not ADHD-depression relations (CSs ≤ .21), across years. The top two figures depict BEI with respect to ADHD-depression relations, and the bottom two figures depict BEI with respect to trait-disorder relations within ADHD-depression networks.

APPENDIX 9. RESULTS OF CASE-DROPPING BOOTSTRAP FOR YEAR 1 ADHD-ANXIETY NETWORKS SEPARATED BY GENDER



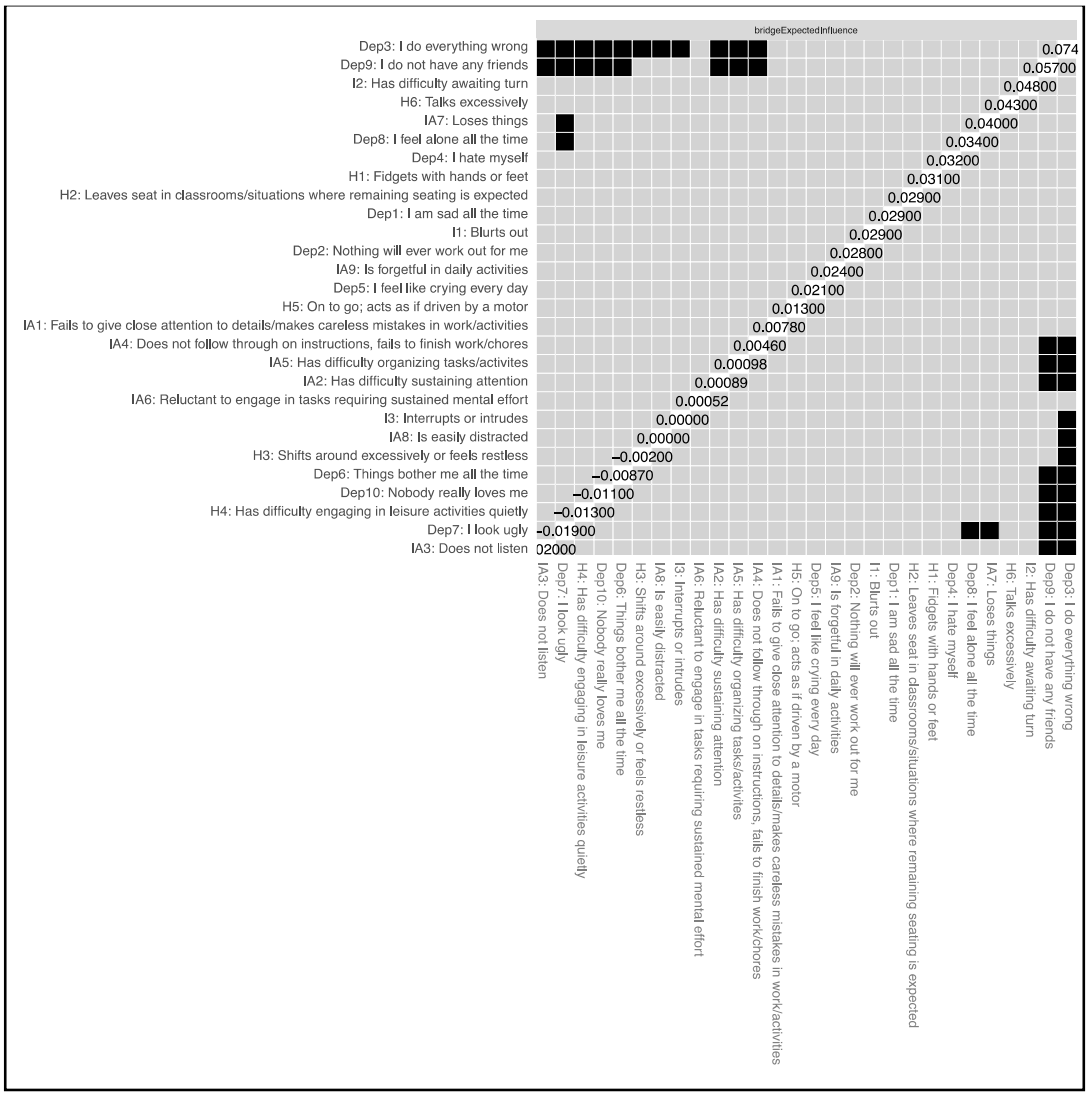
Note. Points represent the correlation between BEI indices in the new sample (after a percentage of cases have been dropped) and original sample. Assessment of the correlation-stability coefficient index (i.e., the proportion of cases that could be eliminated while retaining a correlation of at least 0.70 with the original sample's BEI estimates within a 95% confidence interval; Epskamp & Fried, 2018) suggested adequate stability (i.e., CS > .25; Epskamp & Fried, 2018) in BEI values with respect to trait-disorder relations (CSs ≥ .75), but not ADHD-anxiety relations (CSs ≤ .13), across years. The top two figures depict BEI with respect to ADHD-anxiety relations, and the bottom two figures depict BEI with respect to trait-disorder relations within ADHD-anxiety networks.

APPENDIX 10. RESULTS OF CASE-DROPPING BOOTSTRAP FOR YEAR 6 ADHD-ANXIETY NETWORKS SEPARATED BY GENDER



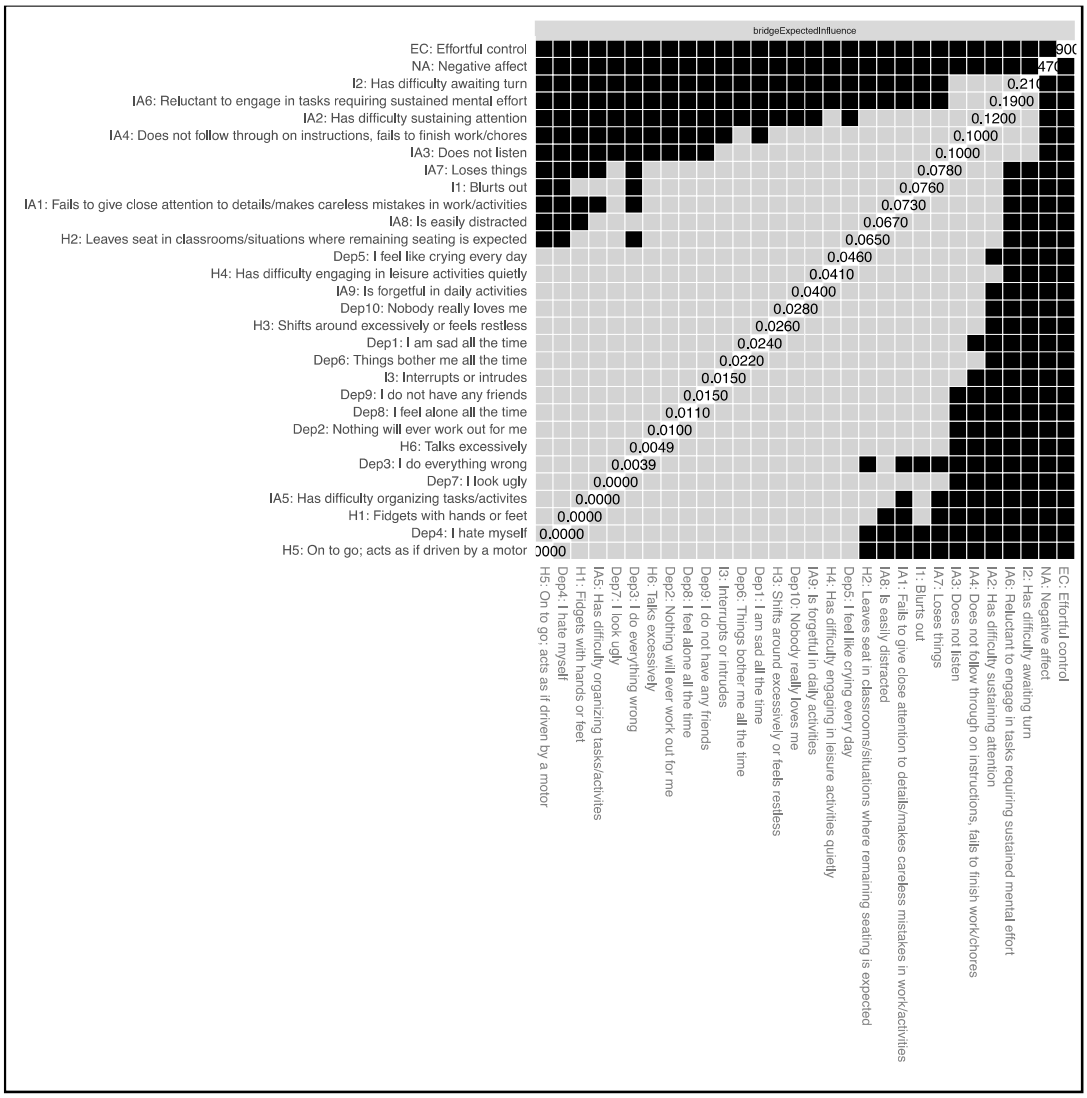
Note. Points represent the correlation between BEI indices in the new sample (after a percentage of cases have been dropped) and original sample. Assessment of the correlation-stability coefficient index (i.e., the proportion of cases that could be eliminated while retaining a correlation of at least 0.70 with the original sample's BEI estimates within a 95% confidence interval; Epskamp & Fried, 2018) suggested adequate stability (i.e., CS > .25; Epskamp & Fried, 2018) in BEI values with respect to trait-disorder relations (CSs ≥ .52), but not ADHD-anxiety relations (CSs = .0), across years. The top two figures depict BEI with respect to ADHD-anxiety relations, and the bottom two figures depict BEI with respect to trait-disorder relations within ADHD-anxiety networks.

APPENDIX 11. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO ADHD-DEPRESSION RELATIONS IN YEAR 1



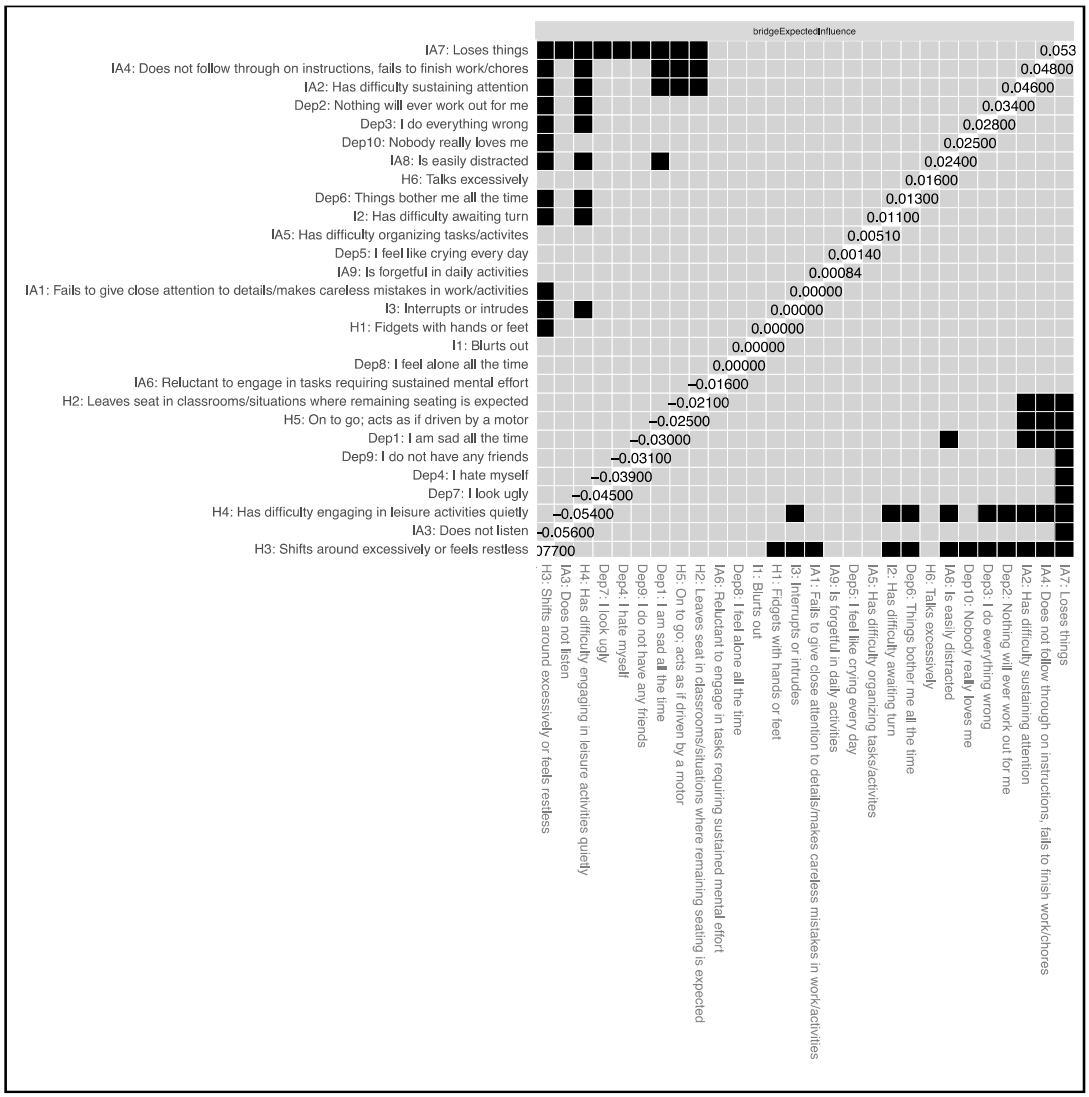
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 12. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO ADHD-DEPRESSION RELATIONS IN YEAR 6



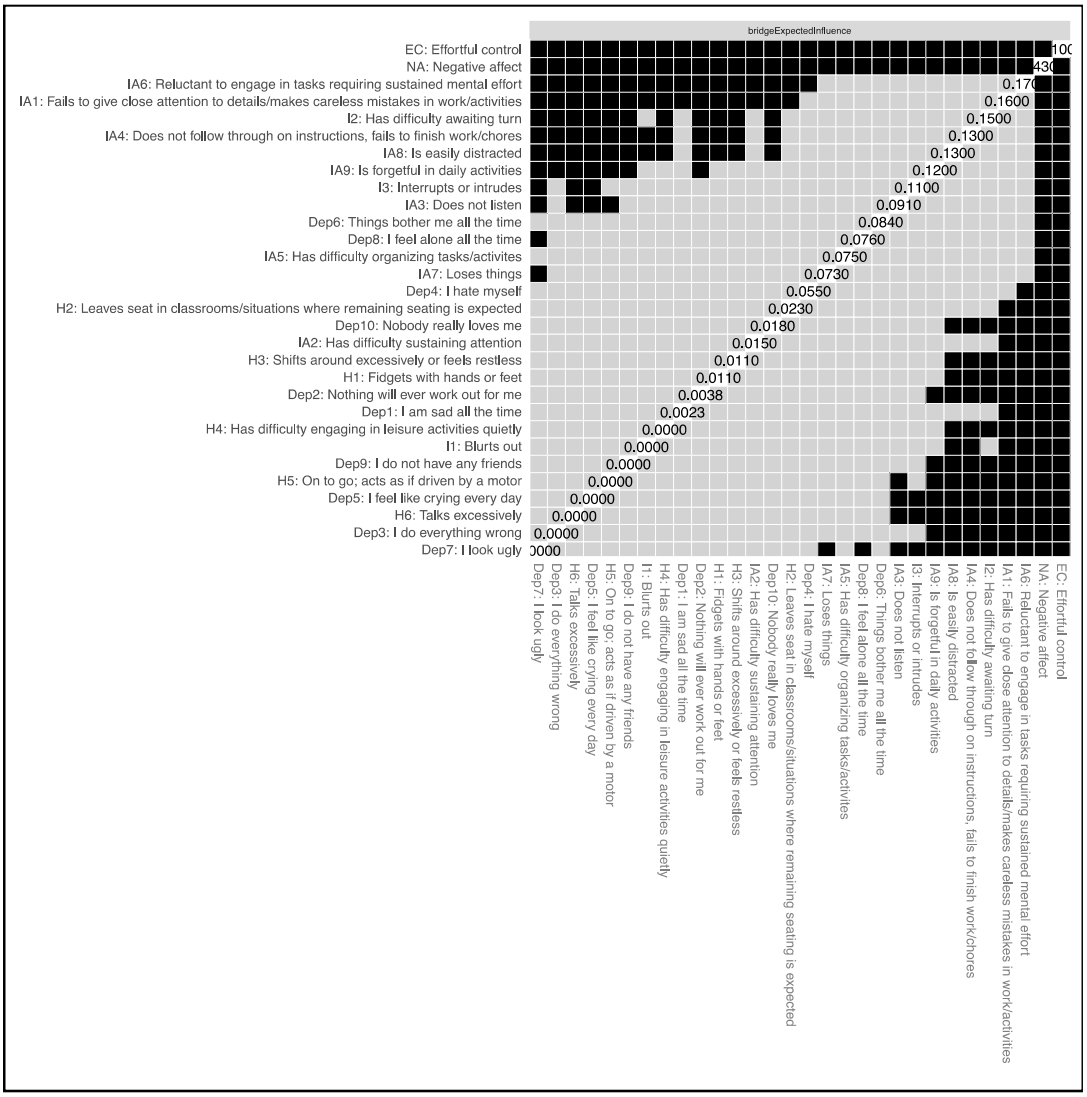
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 13. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO TRAIT-DISORDER RELATIONS IN YEAR 1 ADHD-DEPRESSION NETWORKS



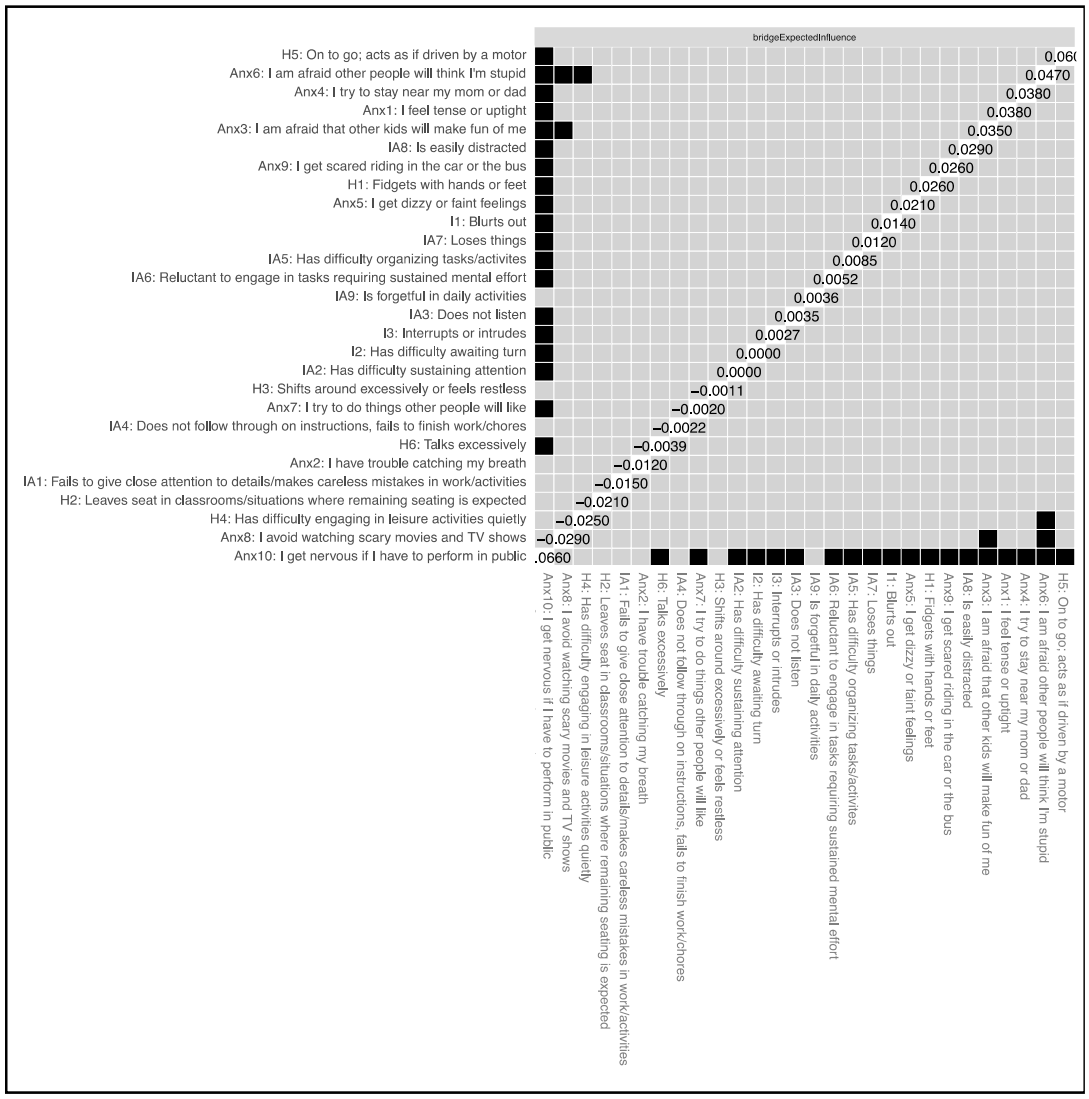
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 14. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO TRAIT-DISORDER RELATIONS IN YEAR 6 ADHD-DEPRESSION NETWORKS



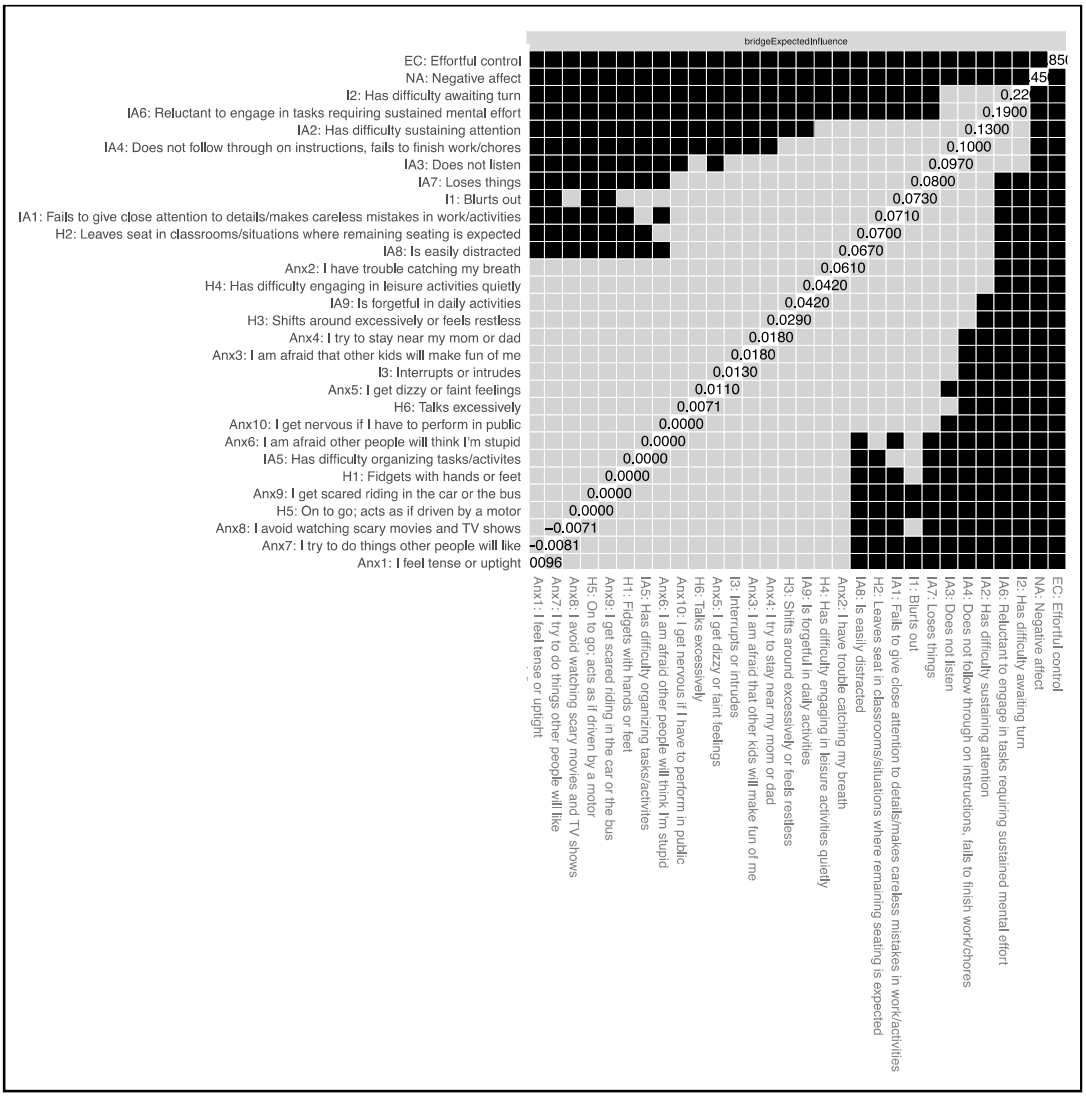
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 15. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO ADHD-ANXIETY RELATIONS IN YEAR 1



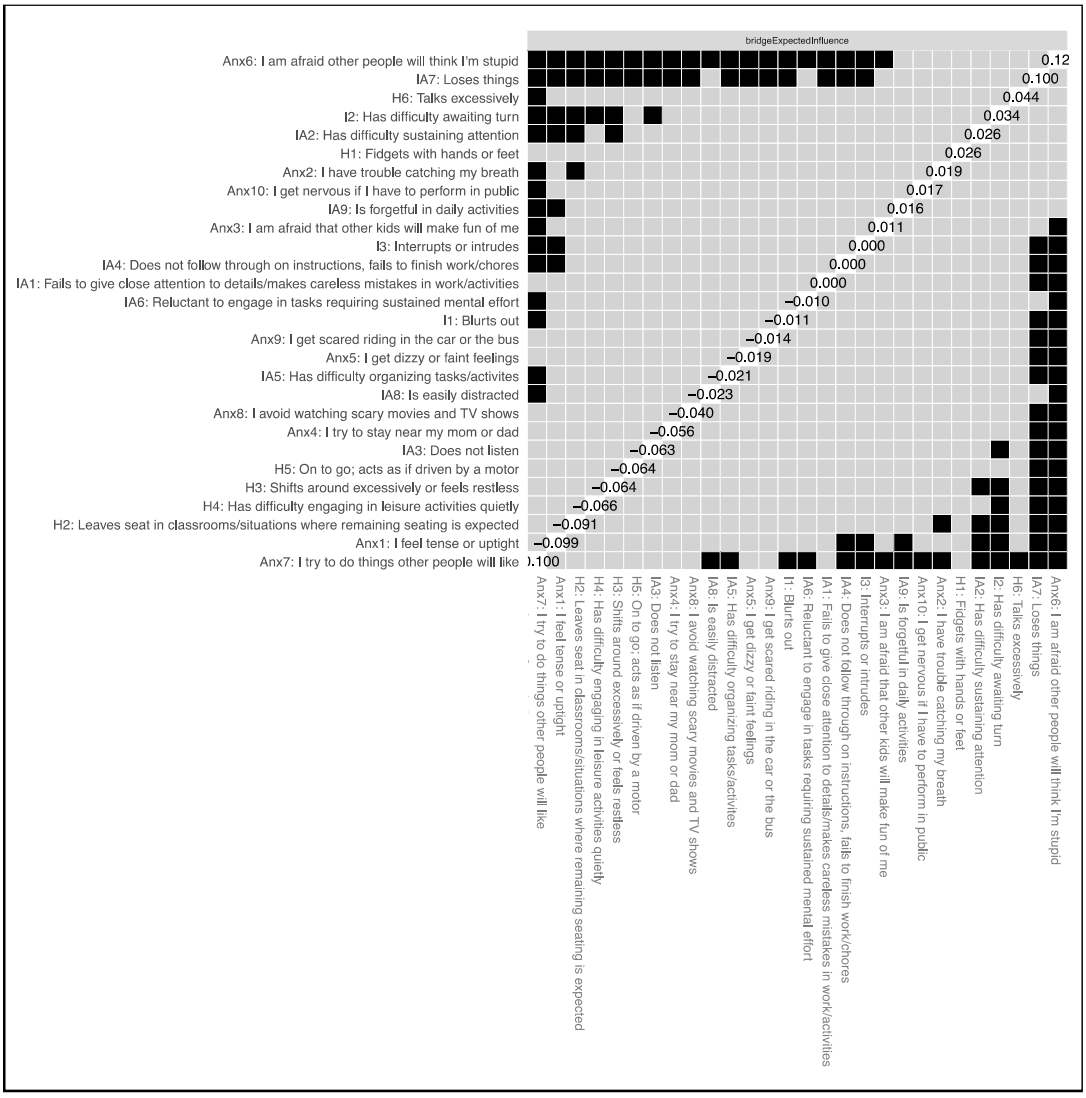
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 16. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO ADHD-ANXIETY RELATIONS IN YEAR 6



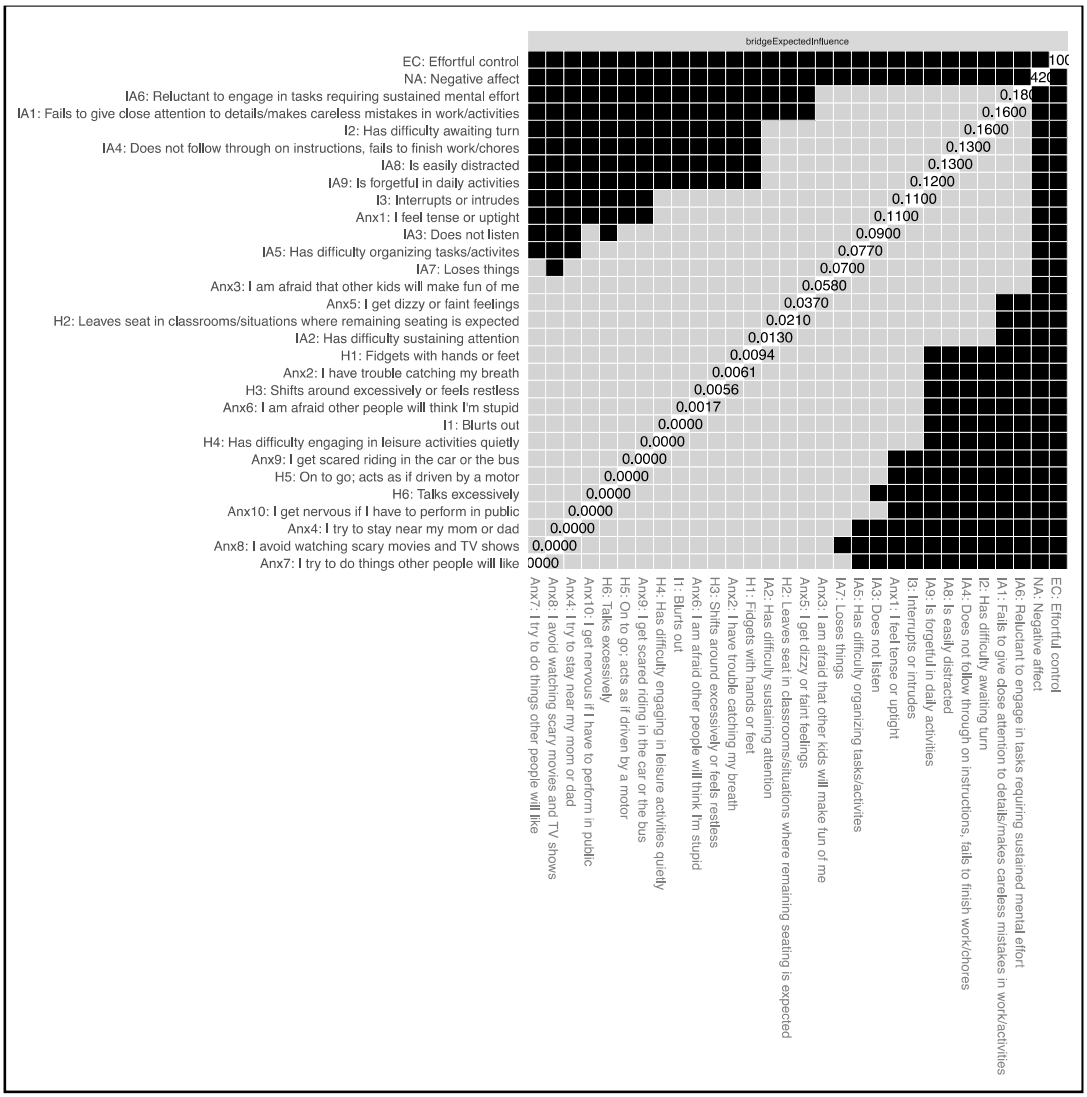
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 17. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO TRAIT-DISORDER RELATIONS IN YEAR 1 ADHD-ANXIETY NETWORKS



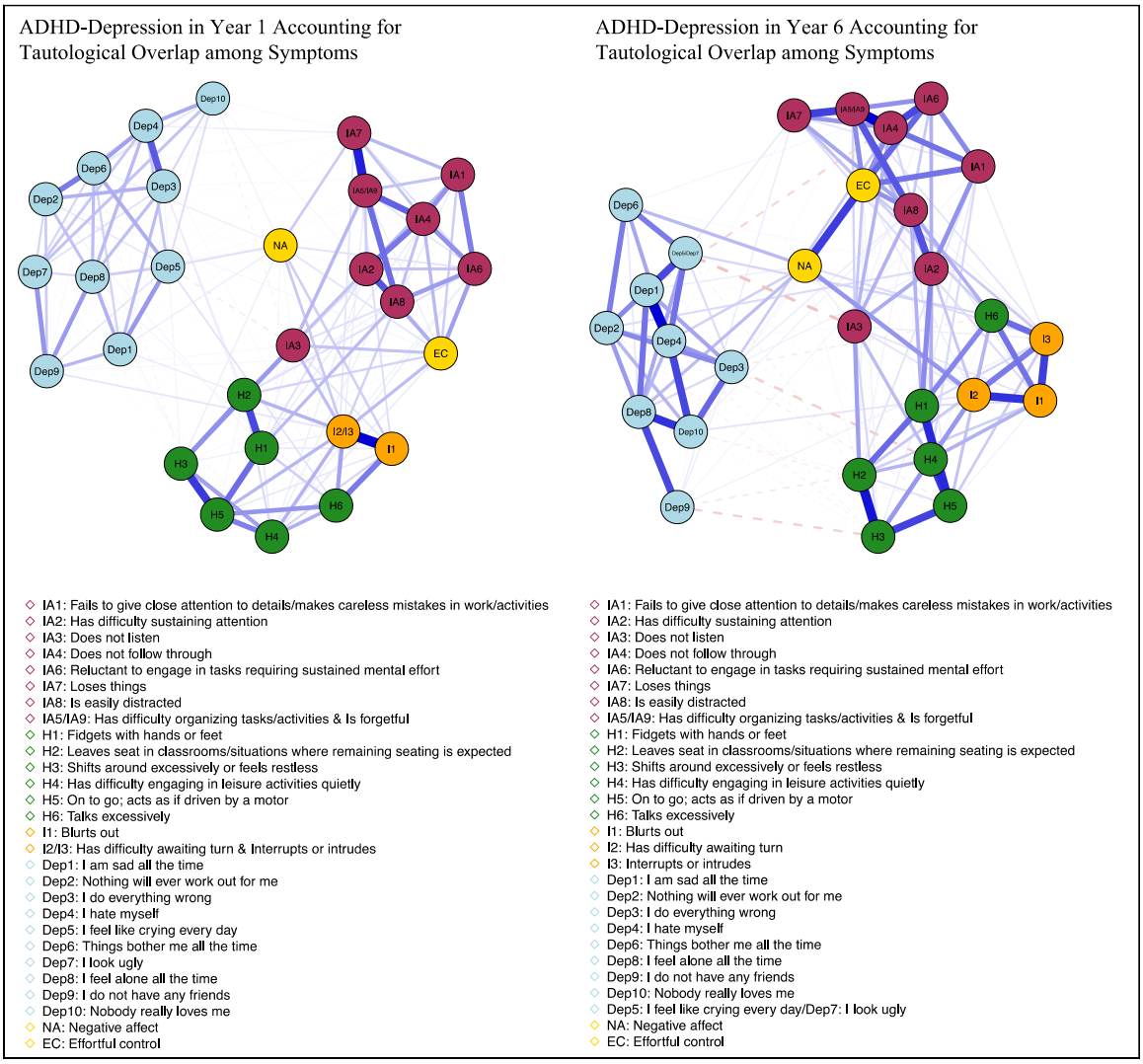
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 18. RESULTS OF BOOTSTRAPPED DIFFERENCE TESTS COMPARING BEI WITH RESPECT TO TRAIT-DISORDER RELATIONS IN YEAR 6 ADHD-ANXIETY NETWORKS



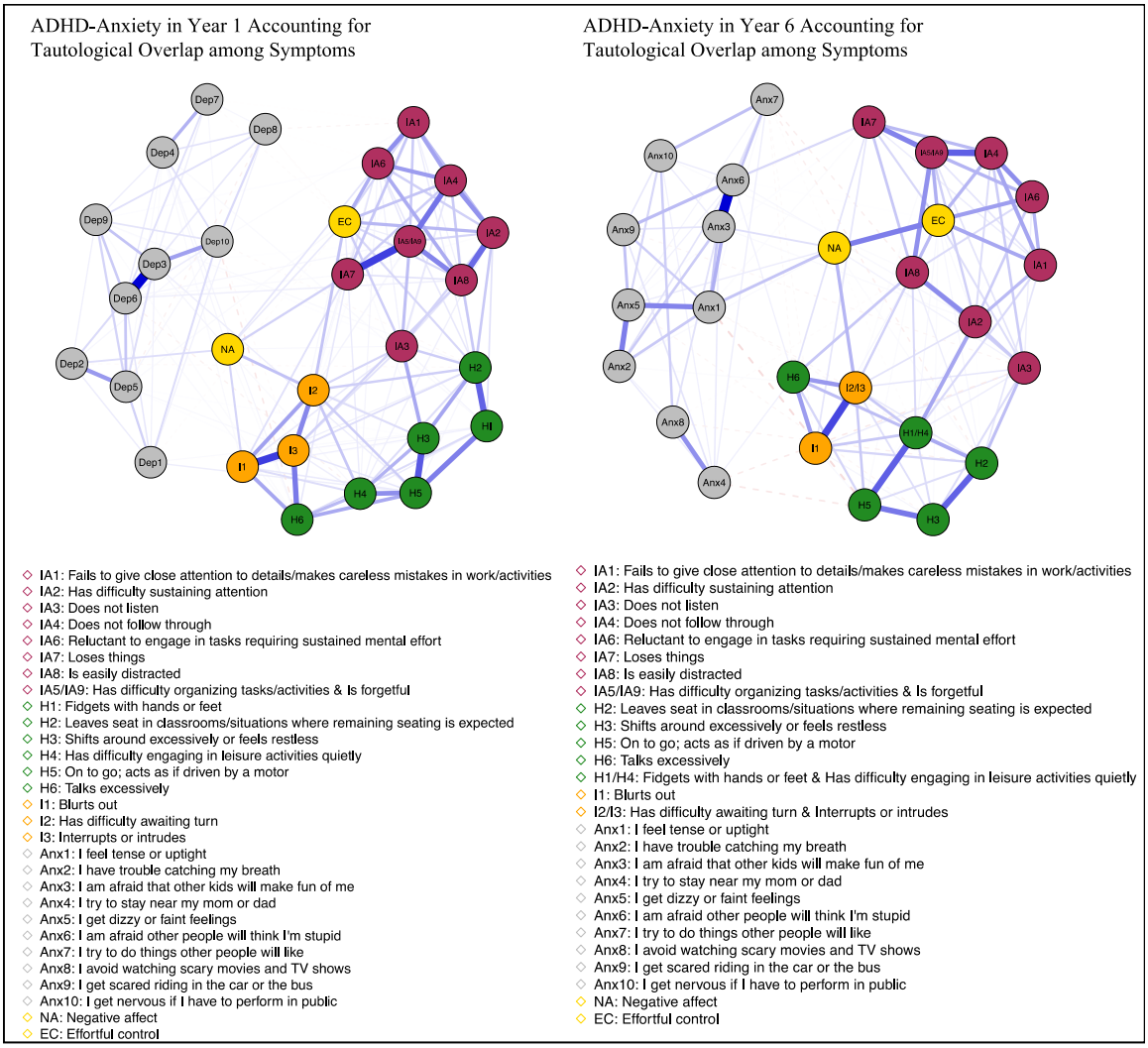
Note. The color of the boxes indicates whether there is a significant difference between variables (i.e., grey boxes reflect no significant differences and black boxes reflect significant differences). To conduct this test, bootstrapped values of BEI for all variables within networks were calculated using 2000 samples with replacement. Next, a difference score between the bootstrapped BEI values of two variables was estimated and a CI around this difference score was constructed. Lastly, a null-hypothesis test was performed on the range of the CI. In the situation that the range of the constructed CIs contained zero, the BEI of two different variables did not significantly differ from one another. This analysis was accomplished using the R package *bootnet* (Epskamp et al., 2018).

APPENDIX 19. ADHD-DEPRESSION NETWORK VISUALIZATIONS AFTER ACCOUNTING FOR REDUNDANCY AMONG VARIABLES



Note. Visualizations were based on the Fruchterman-Reingold algorithm. Variables are depicted as nodes, with edges connecting these nodes depicting regularized partial correlations. Edge thickness represents the strength of the relation. Blue solid edges indicate positive relations and red dashed edges indicate negative relations.

APPENDIX 20. ADHD-ANXIETY NETWORK VISUALIZATIONS AFTER ACCOUNTING FOR REDUNDANCY AMONG VARIABLES



Note. Visualizations were based on the Fruchterman-Reingold algorithm. Variables are depicted as nodes, with edges connecting these nodes depicting regularized partial correlations. Edge thickness represents the strength of the relation. Blue solid edges indicate positive relations and red dashed edges indicate negative relations. Effortful Control scores were reversed so that higher scores indicated greater dysfunction.

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VITA

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Edward Hines, Jr. VA Hospital; Hines, Illinois
Clinical Intern: General Psychology Track

2016 **M.A. in Psychology; Behavioral/Cognitive Neuroscience Track**
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Mentor: Sarah N. Mattson, Ph.D.
Master's Thesis: *Contributions of behavioral impairment to executive function deficits in children with prenatal alcohol exposure*

2011 **B.S. in Psychology**
University of California, San Diego, CA
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Senior Thesis: *Unconscious cognitive conflict in determining attraction of pure and blended faces*

PROFESSIONAL POSITIONS HELD

2016-2021 **Doctoral Research Assistant**
RISK Laboratory, University of Kentucky, Lexington, KY.

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2020	**Clinical Trials Therapist** Treatment Innovation for Psychological Services, Lexington, KY.
2019-2020	**Inpatient Psychiatric Hospital Psychology Trainee** Eastern State Psychiatric Inpatient Hospital, Lexington, KY.
2018-2019	**Health Psychology Practicum Student** Bluegrass Health Psychology, Lexington, KY.
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2016-2018	**Teaching Assistant** Department of Psychology, University of Kentucky, Lexington, KY.
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2013-2016	**Master's Level Research Assistant** Center for Behavioral Teratology, San Diego State University, San Diego, CA.
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SCHOLASTIC AND PROFESSIONAL HONORS

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2019 Elizabeth Munsterberg Koppitz Child Psychology Fellowship Finalist

2019 Rachel Steffens Memorial Research Award, University of Kentucky
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2016 Royster Fellowship, University of Kentucky

San Diego State University
2015 Travel Award; Fetal Alcohol Spectrum Disorders Study Group
2015 Student Merit Award; Research Society on Alcoholism
2015 IRA Travel Award
2015 Dean's Award for Oral Presentation; Student Research Symposium
2015 Enoch Gordis Award Finalist; Research Society on Alcoholism
2015 Student Travel Award; Center for Clinical and Cognitive Neuroscience

PROFESSIONAL PUBLICATIONS

Goh, P. K., Smith T. E., Lee, C. A., Bansal, P. S., Eng, A. G., & Martel, M. M. (in press). Etiological networks of ADHD: A simultaneous examination of temperament traits and executive function. *Journal of Clinical Child and Adolescent Psychology.*

Karalunas, S. L., Antovich, D., **Goh, P. K.**, Martel, M. M., Tipsord, J., Nousen, L., & Nigg, J. T. (in press). Network modeling of the co-development of temperament, executive functioning, and depression in youth with and without ADHD. *Development and Psychopathology*.

Bansal, P. S., **Goh, P. K.**, Eng, A. G., Elkins, A. R., Smith, T. E., Thaxton, M. & Martel, M. M. (in press). Identifying the inter-domain relations among CU traits, ODD, and CD in preschool children using network analysis. *Research on Child and Adolescent Psychopathology.*

Martel, M. M., Elkins, A. R., Eng, A. G., **Goh, P. K.**, Bansal, P. S., Smith, T. E., Mooney, M., A. Gustafsson, H. C., Karalunas, S. L., & Nigg, J. T. (in press). Multiple informant average integration of ADHD symptom ratings predictive of concurrent and longitudinal impairment. *Psychological Assessment.*

Martel, M. M., **Goh, P. K.**, Lee, C. A., Karalunas, S. L., & Nigg, J. T. (in press). Longitudinal ADHD symptom networks in childhood and adolescence: Key symptoms, stability, and predictive validity. *Journal of Abnormal Psychology.*

Goh, P. K., & Martel, M. M. (2020). Commentary: extending longitudinal network approaches – A reflection on Funkhouser et al. (2020). *Journal of Clinical Psychology and Psychiatry, 62*(2), 195-198. https://doi.org/10.1111/jcpp.13320

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Martel, M. M., **Goh, P. K.**, Smith, T. E., & Lee, C. A. (2018). Developmental pathways. In M. Martel (Ed.), *Developmental pathways to disruptive, impulse-control, and conduct disorders.* Cambridge, MA: Academic Press.

Goh, P. K., Doyle, L. R., Glass, L., Jones, K. L., Riley, E. P., Mattson, S. N., & the CIFASD. (2016). A clinically useful decision tree to identify children affected by prenatal alcohol exposure. *The Journal of Pediatrics, 177,* 121-127. https://doi.org/10.1016/j.jpeds.2016.06.047