Dimensions of Externalizing Symptoms in Children and Adolescents

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List of Abbreviations

AD	affective dysregulation
-II	irritability/emotional impulsivity
ADHD	attention-deficit/hyperactivity disorder
-HI	hyperactivity/impulsivity
-HY	hyperactivity
-IN	inattention
-IM	impulsivity
ADHS	Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung
CD	conduct disorder
CFA	confirmatory factor analysis
CFI	comparative fit index
DMDD	disruptive mood dysregulation disorder
DSM	Diagnostic and Statistical Manual of Mental Disorders
ECV	explained common variance
EFA	exploratory factor analysis
g-factor	general factor
HiTOP	Hierarchical Taxonomy of Psychopathology
ICD	International Statistical Classification of Diseases and Related Health Problems
ODD	oppositional defiant disorder
OPP	oppositionelles Trotzverhalten
p-factor	general factor of psychopathology
RDoC	Research Domain Criteria

RMSEA	root mean square error of approximation
s-factor	specific-factor
SMD	severe mood dysregulation
SRMR	standardized root mean square residuals
TLI	Tucker-Lewis index

Introduction

In this dissertation I will discuss advantages and disadvantages of the current categorical classification systems for mental disorders in children and adolescents and aim to suggest a different, more parsimonious classification approach. In order to do this, in my introduction I will first focus on the current categorical classification systems as applied in the fifth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association [APA], 2013) and the eleventh version of the International Statistical Classification of Diseases and Related Health Problems (ICD-11; World Health Organization [WHO], 2020) and will then present alternative, more dimensional approaches that have been suggested and developed more and more in recent years. Next, I will discuss the issue of frequently found comorbidities between disorders accompanying the categorical classification approach, as this is a major point of criticism. In this context I will look at attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD), two mental disorders frequently found to be comorbid during childhood and adolescence, in more detail. In a next step I will elaborate on potential suggested mechanisms, explaining this frequently found comorbidity and will develop a new hypothesis, suggesting affective dysregulation (AD) as the connecting element of ADHD and ODD. I will then discuss latent factor analysis as one potential method to help disentangle comorbidity, as it will be used in this dissertation to examine my newly developed hypothesis of AD being the common core of ADHD and ODD. As a last step of my introduction specific goals of my dissertation will be formulated. I will then provide a short summary of both my articles, including goals of the study, methods, key findings and limitations.

My first article (Junghänel et al., 2020) focuses on ADHD and ODD in a clinical sample and applies a newly developed kind of model, the bifactor S-1 model (Burns et al., 2020; Eid et al., 2018), which will be explained in detail later on. The aim here was to examine how this new model works when assessing comorbidity of two or more symptom complexes compared to traditional bifactor models, which will also be discussed at a later point in more detail. In addition, I took the two different conceptualizations of ADHD according to the DMS-5 (APA, 2013) and the ICD-10/ICD-11 (WHO, 1992, 2020) into accounting, by testing a two-factorial and a three-factorial structure of ADHD.

In my second article I then develop a hypothesis, presuming AD as the underlying core of ADHD and ODD (Junghänel et al., 2022). I use the previously examined new bifactor

S-1 model to test this hypothesis and further apply more bifactor specific indices, which help to make a decision for or against a specific model in a more nuanced way. Additionally, I address several limitations of my first article in this second article: In article one I have not assessed construct validity, a short-coming that I addressed in article two. I have also looked at a large range of other latent factor models and compared the information I got from these different models, in order to make a more informed decision on which model provides us with the biggest gain of information in order to answer my hypothesis. In article two, I also took into account the frequently reported two-dimensional structure of ODD (ODD-irritability and ODD-defiant/oppositional; Aebi et al., 2016; Burke et al., 2014, 2021). Last, a different, more representative sample compared to the purely clinical sample in article one, was used in this second article.

In the end, I will summarize the results from both my articles together and will discuss what my results mean for the classification of mental disorders in children and adolescents. I will suggest an alternative, more parsimonious classification approach, which future research will have to further examine. I will lay out limitations of the current studies and will make suggestions for future research in this area which should address the limitations of my studies.

Categorical versus Dimensional Classification of Mental Disorders

Currently, mental disorders are classified in a categorical way according to the DSM-5 (APA, 2013) and the ICD-10/ICD-11 (WHO, 1992, 2020), meaning that a disorder is either considered absent or present, depending on the number and combination of existing symptoms. This approach is useful for a number of reasons, as it facilitates communication among clinicians, researchers and patients, improves reliable diagnostic assessment, allows for comparison of research results and likely most importantly guides the decision process on which therapeutic interventions might help best in this particular case (Carragher et al., 2015; Caspi et al., 2014). However, at the same time, it comes with a number of non-negligible disadvantages or at least open questions. When using categories and applying cut-offs to diagnose a mental disorder it implies, that having a mental disorder is *qualitatively* different from not having a mental disorder (similar to having a broken leg, versus not having a broken leg). However, research has suggested differences in cognition and behavior to be *quantitatively* different and to exist on a continuum ranging from “normal” to “highly pathological” (Kotov et al., 2017), which is more equivalent to having low or high blood pressure, with established cut-offs indicating at what point treatment might be necessary. This does not mean, however, that categories or cut-offs are useless. On that note, Krueger et al.

(2005) emphasizes that even if psychopathology does prove to be continuous in nature, the use of thresholds can still be necessary in clinical practice, as the presence of symptoms or symptom complexes only becomes problematic for an individual starting at a certain level (i.e. fear up to a certain point is absolutely necessary to survive, it can however severely impair the quality of life when it becomes too excessive). Empirical research, taking symptom levels as well as the likelihood of adverse outcomes into account might help to decide where cut-offs should be placed. Yet the step of understanding the nature of psychopathology (i.e. categorical vs. dimensional, meaning quantitative or qualitative differences in symptomatology), should not be bypassed by deciding on cut-offs for diagnoses right away. We might also lose valuable information when solely relying on diagnostic thresholds. This holds true in particular when we turn to the next point of criticism: the heterogeneity within diagnostic categories. This heterogeneity is caused by the polythetic-categorical approach, applied by current classification systems, meaning that disorders are currently defined by multiple symptoms, which are not all required to receive a certain diagnosis (Carragher et al., 2015). Though this is frequently addressed through the specification of disorder subtypes, these subtypes are typically defined rather rationally instead of being based on research results (Kotov et al., 2017). This is related to another problem, which is the fact that a large number of patients is frequently classified under the category “not otherwise-specified”, which is often used in situations, in which not all necessary symptom criteria for a specific disorder are fulfilled (Carragher et al., 2015).

Another major disadvantage which strongly challenges the categorical approach is the extensive comorbidity that is frequently found. Caspi et al. (2020) found that in a sample of participants of the big Dunedin Study, about 85% of study participants with an initial disorder had acquired at least one additional disorder by the age of 45 years. Similarly, Newman et al. (1998) describes the rule of 50, stating that about 50% of individuals meeting the symptom criteria for one disorder also meet the symptom criteria for a second disorder, of which 50% could be diagnosed with a third disorder and so forth. If mental disorders were truly distinct, separate categories, how can we explain this number of comorbidities? And how could alternative classification systems, accounting for this finding, look like? Dimensional approaches might be a solution for the problem of heterogeneity within one disorder and frequently observed comorbidities between disorders. Krueger and Markon (2006) have suggested a liability-spectrum model of psychopathology, stating that a specific disorder might result from a more general overarching liability, therefore understanding

psychopathology in a hierarchical way. A hierarchical model of psychopathology has also been supported by Lahey et al. (2021), who suggest genetics and environmental factors to non-specifically influence the risk for multiple disorders/dimensions of psychopathology, whereas person-specific experiences appear to influence the development of distinct disorders/dimensions more strongly. Research of childhood psychopathologies has done some pioneer work examining potential dimensions of psychopathology with the study of Achenbach and Edelbrock (1981), as they suggested an internalizing (including for example anxiety/depression) and an externalizing dimension (including for example ADHD, ODD) underlying a range of childhood mental disorders. This is in line with Krueger and Markon (2006), who suggest in their review that the externalizing spectrum is reflected better by a graded continuum than by discrete categories, as continuous models of liability have shown better fit to multivariate patterns of comorbidity than discrete models have. The externalizing and internalizing dimensions have been repeatedly observed in children, adolescents as well as adults (Kotov et al., 2017; Krueger et al., 2007) and have also been recognized by the DSM-5 (APA, 2013), which in their newest version places internalizing and externalizing disorders in adjoining chapters to indicate their contentual proximity. The idea of a few major dimensions underlying a number of mental disorders has led to several major research efforts in recent years, the two most influential ones being the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) and the Research Domain Criteria (RDoC; (Cuthbert & Insel, 2013; Insel et al., 2010). Both approaches are still work in progress and are constantly expanded upon, yet they have offered promising insights into the underlying structure of psychopathology and it can be expected that both approaches, potentially even in combination (Michelini et al., 2021), will help shed some light on the high number of comorbidities. While they use entirely different approaches, they both aim at reorganizing psychological symptom complexes and leaving current diagnostic categories behind, by either applying a bottom-up approach and focusing on etiological mechanisms underlying a range of symptom complexes (RDoC; Cuthbert & Insel, 2013; Insel et al., 2010) or by exploring observed covariation patterns among psychopathological symptoms when applying a top-down approach (HiTOP; Kotov et al., 2017). Whereas the RDoC focus mainly on brain circuits, applies neuroscientific tools and lacks (at least up to this point) a large array of clinically relevant symptoms, the HiTOP, not considering biological mechanisms, has integrated research on clinically relevant symptoms and symptom complexes in order to produce a system reflecting the currently available knowledge on associations between the

former (Kotov et al., 2017). The HiTOP includes six hierarchical layers, ranging from signs and symptoms on the lowest level via symptom components and traits, syndromes and disorders, subfactors, spectra to a super spectrum on the highest level, which allows for a flexible description of a patient, depending on how much specificity is required in the specific context (Kotov et al., 2017). The spectra incorporate and expand upon the previously described internalizing and externalizing dimensions, whereas the super spectrum corresponds to a higher order dimension, similar to a general psychopathology factor. This general psychopathology factor, first described in detail and termed the p-factor by Caspi et al. (2014), in accordance to the g-factor of general intelligence, has been associated with increased life impairment and greater familiality (Caspi et al., 2014). The idea behind the p-factor (similar to the g-factor of general intelligence) is that such a measure, based on a range of problems (or in case of the g-factor of general intelligence *abilities*), might be a better prognosticator than a measure relying on a more narrow spectrum of problems (Achenbach, 2021). Or as Carragher et al. (2015, p. 340) put it, "the implicit assumption underlying this quantitative, organizational meta-structure of psychopathology is that certain disorders are reflections of a few core psychopathological dimensions". It is tempting to fall under the spell of the idea of a p-factor as it would solve our problems with observed heterogeneity within disorders and comorbidities of a range of disorders. However, it has been proven difficult to identify exactly what this p-factor stands for, despite the obvious positive correlation between all included symptoms. Is it merely a sum of existing problems and therefore stands for symptom severity (Fried et al., 2021)? Or does it truly represent liability (Krueger & Markon, 2006; Lahey et al., 2021)? Which implications would these different interpretations have and how would it improve clinical practice? And importantly, what exactly does a high p-value translate to on a behavioral and/or cognitive level? As Eid et al. (2018) pointed out: "If one cannot give a clear answer to this question [What an individual factor score tells us about the psychological state of an individual], the assumption of a general factor might not be reasonable" (p. 556). Similar to the g-factor of general intelligence, where we mainly know what it stands for and is associated with, we need to further examine the exact nature of *p* and how it could help us eventually improve treatment, as this needs to remain the major goal of psychopathological classification. In order to achieve this, we need to formulate and test specific hypothesis about *p* (Fried et al., 2021).

ADHD and ODD and their Comorbidity in Children and Adolescents

At this point, with our current knowledge, identifying a p-factor underlying *all* mental disorders appears to be a challenge too great to conquer and it might therefore be more feasible to first focus on a more narrow spectrum of disorders that frequently co-occur and formulate and test hypotheses about the reasons for their frequent co-occurrence. Two mental disorders, very prevalent in childhood and adolescents are ADHD and ODD, which are both part of the externalizing spectrum (Achenbach & Edelbrock, 1981; Kotov et al., 2017). ADHD is defined by a number of inattention, hyperactivity and impulsivity symptoms. In the DSM-5 (APA, 2013), it consists of the two dimensions inattention (ADHD-IN) and hyperactivity/impulsivity (ADHD-HI), whereas in the ICD-10/ICD-11 (WHO, 1992, 2020) a very similar symptomatology is called hyperkinetic disorder and consists of the three dimensions IN, hyperactivity (HY) and impulsivity (IM). In the DSM-5 (APA, 2013) as opposed to the ICD-10/ICD-11 (WHO, 1992, 2020), subtypes (primarily inattentive, primarily hyperactive/impulsive and combined presentation) can be assessed and diagnosed. For ADHD, Polanczyk et al. (2015) reported a worldwide pooled prevalence of just above 5% for ADHD in children and adolescents under the age of 18 in their systematic review. ODD is a disorder defined in the DSM-5 (APA, 2013) and the ICD-10/ICD-11 (WHO, 1992, 2020) through oppositional and defiant behavior, generally without prominent dissocial behaviors (such as it is observable in conduct disorder [CD]). ODD frequently concerns the non-compliance to rules and it often seen in familiar contexts at home or with peers. The pooled worldwide prevalence for ODD has been reported at just above 3% (Canino et al., 2010). In his review regarding comorbidities within the externalizing spectrum, Willcutt et al. (2012) found about half of children with a diagnosis of ADHD to also meet criteria for the diagnosis of ODD. Reason for this might be genetic factors underlying a general liability for externalizing disorders (Krueger et al., 2005, 2007). Beauchaine et al. (2010) have suggested that comorbidity of ADHD and ODD might result from the combination of inherited impulsivity and oppositionality, aspects highly relevant in ADHD *and* ODD, and growing up in High-Risk Environments, therefore lacking skills to regulate one's own emotions. The ICD-10 (WHO, 1992), accounts for these frequently found comorbidities and allows for a combined diagnosis of ADHD and ODD symptomatology with the diagnosis "hyperkinetic conduct disorder" (F90.1). The close proximity is further supported when looking at treatment approaches of ADHD and ODD as they show a great deal of overlap. For both disorders, parent trainings including increasing positive interactions between parent and child, talking

about emotions and practicing and establishing consistency in parenting behavior are of great importance (Costin & Chambers, 2007; Lee et al., 2012; Petermann & Lehmkuhl, 2012). Another important therapeutic invention in ODD and also in ADHD are social competency trainings, including conflict resolution trainings, role plays and training of social problem-solving skills (Antshel & Remer, 2003; Görtz-Dorten et al., 2019; Petermann & Lehmkuhl, 2012; Willis et al., 2019). Programs addressing conduct as well as hyperkinetic problem behaviors such as the Incredible Years program (Webster-Stratton, 2001) have shown that similar treatment components ameliorate ADHD as well as ODD symptomatology (Hobbel & Drugli, 2013; Webster-Stratton et al., 2011). On a similar note, Hechtman et al. (2005) found multimodal treatment (medication *and* behavioral treatment) of ADHD to reduce ODD symptomatology as well.

On a behavioral level, two dimensions frequently associated with both disorders stand out and might at least be partly responsible for the frequent comorbidity as well as the similar treatment approaches. The first dimension is irritability, which has often been identified as one of the (at least) two key dimensions of ODD – the other one frequently found being the oppositional/defiant dimension of ODD (Burke et al., 2014; Rowe et al., 2010). The two dimensions have been found to be distinct (Burke et al., 2021) and in particular the irritable dimension has been associated with self-reported attention problem (Aebi et al., 2013). This is highly interesting in this context as ADHD has also been strongly associated with irritability (Eyre et al., 2017). Eyre et al. (2017) found at least one irritable symptom in 91% of children diagnosed with ADHD. Interestingly, comorbid ODD was also particularly common in their sample, especially in cases of ADHD *and* irritability combined. The second dimension relevant to both disorders is impulsivity. In ADHD impulsivity is even considered one of the core components of the disorder. As pointed out before, the number of core components of ADHD differs between the two main classification systems (two dimensions in the DSM-5 and three dimensions in the ICD-10/ICD-11), which is interesting, as the combination of hyperactivity and impulsivity in the DSM-5 hints at impulsivity being an “umbrella term” (Berg et al., 2015) for a range of different impulsive behaviors (e.g. physical/emotional impulsivity) and the combination of hyperactivity and impulsivity as one core dimension of ADHD suggests that ADHD might mainly capture physical impulsivity. In ODD, impulsivity is no spelled-out dimension, however strong associations with impulsivity have been reported (Avila et al., 2004). Martel et al. (2017) even considered impulsivity the core of the externalizing spectrum. In the following, affective dysregulation, a concept incorporating

irritability and impulsivity, will be discussed in detail as a potential underlying core of ADHD as well as ODD symptomatology.

Affective Dysregulation

AD is a term that is ambiguously defined, therefore the prevalence rates vary depending on its exact definition (Brotman et al., 2006; Copeland et al., 2013; Holtmann et al., 2008). Irrespective of the exact prevalence rate however, Brotman et al. (2017) concludes in their review that this symptom complex is among the most frequent reasons for children and adolescents to be referred to psychiatric care. AD is sometimes used interchangeably with emotion dysregulation or irritability (Evans et al., 2017; Leibenluft, 2011; Shaw et al., 2014). Whereas emotion dysregulation describes the coping process with emotional challenges, AD might be rather understood as a state resulting from dysfunctional emotion regulation (Döpfner et al., 2019; Leibenluft & Stoddard, 2013; Waltereit et al., 2019). Irritability sometimes refers to a more narrow construct than AD when it omits the impulsive component, though there are also conceptualizations of irritability entailing a tonic and phasic component, which then corresponds to the combination of prolonged anger and temper outbursts (Copeland et al., 2015). Brotman et al. (2017) define irritability as "a low threshold for experiencing anger in response to frustration" (p.319). When in the following we use the term AD, we refer to a construct being composed of anger/irritability (affective component) and impulsivity/aggression (behavioral component), which corresponds to Leibenluft & Stoddard (2013). At this point it should be noted that impulsivity has been suggested to be an "umbrella concept" (Berg et al., 2015) entailing various forms of impulsivity. This is important in this context in so-far, as AD- vs. ADHD-related impulsivity differ in that AD-related impulsivity is rather emotional in nature (Barkley & Fischer, 2010), whereas ADHD-related impulsivity appears to be rather physical in nature, correspondingly comprising one dimension together with hyperactivity in the definition of ADHD in the DSM-5 (APA, 2013).

The need for a construct capturing AD/irritability symptomatology arose from the observation of an increased diagnosis of bipolar disorders in children and adolescents (Blader & Carlson, 2007; Malhi & Bell, 2019). However, irritability in bipolar disorders is, by definition, episodic, and differs from the chronic irritability frequently observed (and likely misdiagnosed with bipolar disorder) in many children and adolescents (Leibenluft, 2011). In addition, it has been emphasized that irritability is a symptom or at least a feature associated with a number of mental disorders and therefore appears to be of transdiagnostic nature

(Evans et al., 2017). One early attempt to operationalize chronic irritability came from Leibenluft et al. (2003; 2011), who described a construct termed severe mood dysregulation (SMD) by combining chronic irritability with temper outbursts and symptoms of hyperarousal (Leibenluft, 2011; Leibenluft et al., 2003). The increasing research on AD symptomatology as well as the pressing need to establish a diagnosis that fits this symptomatology better than a bipolar diagnosis, led the DSM-5 (APA, 2013) to include a new disorder capturing AD symptomatology, called disruptive mood dysregulation disorder (DMDD). Interestingly, though evidence from SMD was used for the inclusion of DMDD as an additional mood disorder in the DSM-5 (APA, 2013), most SMD defining symptoms were excluded, leaving DMDD to be defined by the combination of chronic irritability/anger (strongly corresponding to symptoms of the ODD-irritability dimension) and temper outbursts (APA, 2013; Burke et al., 2021). To meet DMDD criteria (APA, 2013), intense, temper outbursts, which are age-inappropriate, must occur at least three times per week and severe irritable mood must be present for most of the days. These two core symptoms must be present over the course of one year and have to be observable in at least two out of three settings (home/school/peer group). DMDD cannot be diagnosed in children younger than 6, and the age of onset of the aforementioned symptoms must be before the age of 10. The close proximity of DMDD and ODD (Burke et al., 2021) led the DSM-5 (APA, 2013) to include a hierarchical rule stating that once the criteria of DMDD are fulfilled, ODD cannot be diagnosed as a comorbid disorder.

The addition of DMDD as an additional categorical mood disorder to the DSM-5 (APA, 2013), was welcomed on the one hand as it countered the increased rates of bipolar disorders in children and allowed for a diagnosis of children that were significantly impaired by their chronic irritability and frequent temper outbursts, a combination of symptoms that up to this point were only partially included in the diagnosis of other externalizing disorders, such as ODD, ADHD or CD (Malhi & Bell, 2019). On the other hand, it also led to a lot of criticism (Evans et al., 2017; Lochman et al., 2015; Runions et al., 2016), as DMDD showed significant overlap to a range of other disorders (Copeland et al., 2013; Evans et al., 2017), in particular ODD (Evans et al., 2017; Mayes et al., 2016) and ADHD (Eyre et al., 2017; Mulraney et al., 2016), therefore potentially lacking diagnostic distinction (Malhi & Bell, 2019). However, the additional assessment of DMDD symptomatology did not appear to be useless, as Eyre et al. (2017) found youth suffering from ADHD *and* DMDD to be more impaired than youth with *only* ADHD. Similarly, Shaw et al. (2014) found the combination of

ADHD and emotion dysregulation to be associated with more severe impairment in several contexts (peer group, family, academic performance), even after controlling for ODD as a comorbid diagnosis.

Yet it became apparent that children with a DMDD diagnosis had mostly already been identified (though not precisely described in their AD symptomatology) as they have previously been given at least one other diagnosis, most frequently ODD and/or ADHD (Evans et al., 2017). On top of that, no specific treatment instruction for children with DMDD exist, leading clinicians to select therapeutic interventions, originally developed to treat other disorders, such as ODD, ADHD and depression (Malhi & Bell, 2019). This point of criticism is of major importance as we have to keep in mind that the main point of a classification system is to guide the most ideal treatment for the individual patient. This criticism as well as suggestions by Evans et al. (2017) and Lochman et al. (2015) led the ICD-11 (WHO, 2020) to choose a different approach of including AD symptomatology in their classification system, as they decided to add the specifier "with chronic irritability/anger" to the diagnosis of ODD. Interestingly, a similar suggestion to add an irritability specifier to the diagnosis of ADHD has recently been made by Karalunas et al. (2019) as they found one group of patients with ADHD as well as irritability, which was not reduceable to the combined presentation of ADHD and ODD alone.

Though the specifier approach of the ICD-11 (WHO, 2020) now accounts for the close proximity of ODD and DMDD symptomatology and potentially facilitates diagnosis, while still capturing children with chronic irritability and temper outbursts more adequately than with a diagnosis of bipolar disorder, it is still categorical in its approach. It has however been suggested, that AD symptomatology is a continuously distributed trait (Brotman et al., 2017), therefore fitting well within dimensional approaches such as the RDoC (Cuthbert & Insel, 2013; Insel et al., 2010) or the HiTOP approach (Kotov et al., 2017). Adequate methodology should be applied to better understand the symptomatology of AD as well as its associations with other mental disorders, in particular ADHD and ODD. AD symptomatology is a promising candidate to explain the comorbidities frequently found between ADHD and ODD as in addition to irritability it also entails (emotional) impulsivity. This corresponds to Carver et al. (2017), who states that being highly reactive to emotions can lead to difficulties and psychopathologies of all sorts (i.e. liability), depending on the combination with other factors. They therefore hypothesize an overall tendency to impulsive reactions, which is closely associated with definitions of AD (e.g. Brotman et al. (2017, p.319), who define irritability as

"a low threshold for experiencing anger in response to frustration") to constitute the general liability factor of psychopathology (Carver et al., 2017).

Latent Factor Analysis as a Method to Disentangle Comorbidity

Latent factor analysis is one potential approach to reduce dimensionality and correspondingly transform nosology into a more parsimonious approach, consisting of fewer, investigable construct (Eaton, 2015). One way to do this is a data-based exploratory (or: bottom-up) approach called exploratory factor analysis (EFA), which looks at symptom covariation patterns and suggests a number of not directly observable dimensions/latent factors, that might be responsible for the correlation between symptoms on a data-level and the observed comorbidity between disorders on a contentual level (Eaton, 2015). Another way is to apply a top-down approach, called confirmatory factor analysis (CFA) to test specific hypotheses regarding which symptoms belongs to which dimension, and how these dimensions might be interrelated. A combination and alternation of bottom-up and top-down approaches is generally recommended as in a first step hypotheses have to be established based on symptom data to then be tested in other samples. This alternation allows for a refinement of hypotheses and might lead us, step by step, towards our goal of a more parsimonious classification approach of mental disorders. Latent factor analysis is therefore suitable to advance theory (Eaton, 2015). The general assumption of latent factor analysis is that the latent variable causes all directly observable symptoms that belong to it, therefore accounting for all correlations between these manifested symptoms (Brunner et al., 2012). Latent factor modeling cannot demonstrate causality directly. It can however, when applied to a large sample of psychiatric symptoms belonging to several mental disorders, suggest core dimensions of psychopathology potentially accounting for the observed comorbidity (Eaton, 2015), with that making theoretical conceptions of comorbidity explicit (Krueger & Markon, 2006). These core dimensions could in a next step then serve as a target for approaches, researching biological underpinnings of mental disorders (Eaton, 2015) such as the RDoC (Cuthbert & Insel, 2013; Insel et al., 2010). However, latent factor analysis is not quite as straight-forward as often a variety of models fit the data rather well and it is the researcher's task to make an informed decision based on theoretical and statistical considerations as to which model fits the data best (Brunner et al., 2012). In the following paragraphs, we will discuss a number of frequently applied latent factor models (first-order correlated factors model, higher-order correlated factors model, traditional bifactor model, bifactor S-1 model) which all come with advantages and disadvantages, depending on which research questions

they are meant to answer. We will focus on top-down approaches in this section, as bottom-up approaches such as EFA are an entire chapter for themselves. In this context for us, it is enough to mention that they are important in order to generate hypotheses, which can then be further examined applying top-down approaches, such as the ones that will be discussed in the following paragraphs.

First-Order Correlated Factors Model

The main assumption in a first-order correlated factors model is that a number of items are influenced by *one* latent factor (i.e. the latent factor ADHD-IN influences the ability to sit still in class, the ability to listen to orders etc.). These first-order latent factors are allowed to correlate (Brunner et al., 2012). This approach is very well suited for a first inspection of data, to see if the hypothesized latent factors are well defined in this sample and to assess how strongly associated the latent factors are with each other, which is important to potentially postulate and examine higher-order models of some sort. However, one major problem of first-order correlated factors models, at least in cases where this model is not simply used as a first inspection, but chosen as "the winning model" is, that correlations between first-order factors are not explained. These factors account for the correlation between manifested symptoms but it remains an open question what accounts for the (frequently quite high) correlations between the first-order factors. The problem of unexplained comorbidity is correspondingly just transferred to a higher level now. A first-order correlated factors model therefore also often contributes to the generation of hypotheses now regarding potential higher-order factors, which might influence the correlations between first-order factors (Brunner et al., 2012; Eid, 2020). In cases where high correlations between first-order factors are found, models explicitly assuming only one general factor (g-factor) accounting for these correlations (such as a unidimensional model, in which for example the factor "externalizing symptomatology" explains all symptoms of ADHD, ODD and CD) or a model assuming specific factors (s-factor) in addition to the g-factor (such as higher-order correlated factors models or bifactor models) can be postulated.

Higher-Order Correlated Factors Model

Higher-order correlated factors models are very similar to first-order correlated factors models, with the addition of a higher-order factor (e.g. externalizing symptomatology), now attempting to explain the comorbidities found between the first-order factors (e.g. ADHD and ODD; Gignac, 2016). In this model, correlations between first-order factors are now constrained to zero, as these correlations are assumed to be accounted for by the higher-order

factor (Brunner et al., 2012). This is the only model, with which we can simultaneously assess associations of all first-order factors with the higher-order factor (Gignac, 2016), which is an important advantage. This model does however come with a few major drawbacks, which we will line out in the following. One at least potential drawback is that we frequently see differentially high loadings of the first-order factors onto the second-order factors, challenging the assumption that the second-order factor explains all first-order factors equally well (Brunner et al., 2012; Gignac, 2016). One of the biggest drawbacks however is the proportionality constraint, meaning that the proportion of variance in each item explained by the g- or the s-factor is constrained to be identical for all items within one first-order factor, limiting the gain of information of this model, when it comes to assessing associations between g- and s-factors on the one side and psychological symptoms or constructs on the other side (Brunner et al., 2012; Gignac, 2016; Reise, 2012). One model, assuming s- and g-factors and not having to deal with the proportionality constraint is the bifactor model (Brunner et al., 2012).

Traditional Bifactor Model

The traditional bifactor model differs in so-far from the higher-order factors model in that the g-factor in the bifactor model is modelled as another first-order factors, defined directly (and not indirectly like in higher-order factors model) by the items (Brunner et al., 2012). Contentually, the g-factor in the traditional bifactor model is equivalent to the previously discussed p-factor as a liability factor of general psychopathology (Caspi et al., 2014). The g-factor (e.g. externalizing symptomatology) is assumed to influence all observed variables; additionally, one s-factor for each dimension (e.g. ADHD-IN; ADHD-HI) is modelled (Eid et al., 2018). Correlations between all s-factors and between all s-factors and the g-factor are constrained to zero, as correlations would contradict the basic assumption of one unifying factor (Brunner et al., 2012; Heinrich et al., 2020; Reise, 2012). Importantly, s-factors in the bifactor model differ strongly from s-factors in the higher-order factors-models as in the traditional bifactor model, s-factors are formed based on residual correlations between the items and are therefore to be interpreted as residualized factors (Brunner et al., 2012; Eid et al., 2018; Gignac, 2016; Markon, 2019; Reise, 2012). This is of particular importance when interpreting bifactor-specific indices, such as omega (hierarchical) values, which provide an estimation of how much reliable variance is accounted for by g- and s-factors individually, as well as taken together (Brunner et al., 2012; Reise et al., 2013), as by definition, s-factors have lower internal consistencies than the total scores (Reise et al., 2013).

For other bifactor-specific indices that help guide a decision for or against a specific model see also Rodriguez et al. (2016). The traditional bifactor model is a promising model, in which g- and s-factors can be modelled independently from each other. It does however come with a number of limitations, which will be discussed in the following. First of all, it is important to emphasize that often bifactor models are not based on a clear definition of the g-factor, but are more exploratory in nature. This makes it very challenging to clearly identify the meaning of the g-factor as well as to compare it between studies (Eid, 2020). Another potential drawback concerns the constraint of all correlations to zero, as it seems questionable that s-factors are truly uncorrelated with the g-factor (Brunner et al., 2012). Additionally, it is unfortunate that s-factor are not allowed to correlate, as this would contradict the general assumption that the g-factor accounts for all variance common to all items. It does however seem plausible, that some of the s-factors (e.g. ADHD-IN and ADHD-HI) remain correlated, as opposed to some other s-factors (e.g. ADHD-IN and CD), even when the g-factor-accounted variance (e.g. externalizing symptomatology) has been controlled for. This leads us to the problem of domain interchangeability (meaning that a set of domains is randomly chosen from a large set of equivalent domains), which is generally assumed in traditional bifactor models, but is more than questionable in applied research, as domains are generally not selected from a large pool of equivalent domains but might differ from each other structurally (Eid et al., 2018; Heinrich et al., 2020). If domains were truly interchangeable, correlations between these should not differ from each other (Heinrich et al., 2020). On a related note, bifactor models frequently face the issues of collapsing s-factors (Eid et al., 2018; Heinrich et al., 2020) and anomalous (i.e. non-significant and/or negative) factor loadings (Burns et al., 2020; Eid et al., 2018), which according to Heinrich et al. (2020) might be “understood as a result of applying a modeling approach that requires interchangeable domains to structurally different domains” (p.14). Collapsing s-factors and anomalous factor loadings greatly challenge the interpretation of the entire model. In the case of collapsing s-factor, the meaning of the g-factor now corresponds to the meaning of the collapsed s-factor instead of to a general liability to psychopathology, which correspondingly means that s-factors change their meaning and now reflect specific variance relative to the dimensions with the collapsed s-factor (Heinrich et al., 2020). Needless to say that a comparison between studies is also impossible in case of collapsing s-factors (Heinrich et al., 2020). For further details regarding the problems with collapsing s-factors and anomalous factor loadings see Eid et al. (2018) and Heinrich et al. (2020).

Bifactor S-1 Model

As discussed, the traditional bifactor model comes with a number of great advantages, but brings along a large number of disadvantages as well, making it difficult to reliably interpret results and compare them between studies, which would be necessary to reliably identify the longed-for general liability factor of psychopathology. A slightly altered version of the bifactor model has been suggested, in which one of the specific domains is now taken as a reference domain (our new, now à priori defined g-factor) in that no s-factor is modelled for this domain (Eid et al., 2018). In this case, theoretical deliberations guide the choice of the general reference domain rather than letting the exact composition of the g-factor depend on the data as is the case in the more exploratory approach the traditional bifactor model applies (Heinrich et al., 2020). This is particularly useful, when a clear theoretical foundation exists, as to which dimension might serve as an ideal reference facet (Eid et al., 2018). Again, the s-factors are understood as residual factors (part of the domain not shared with the reference domain), therefore correlations between s-factors and the g-factor (now corresponding to our reference domain) are constrained to zero, whereas correlations between s-factor are now allowed and can be meaningfully interpreted as partial correlations, representing the commonality of two s-factors after partialing out the effect of the g-factor (Eid et al., 2018; Heinrich et al., 2020). This is one major advantage of the bifactor S-1 model. Another major advantage is that g- and s-factors do not change their meaning when adding or removing domains, allowing for a comparison between studies (Eid et al., 2018). A big shift in interpretation concerns the g-factor in the bifactor S-1 model as it is no longer assumed to represent the much longed-for general liability factor of psychopathology. Relatedly, s-factors also change their meaning. However, as Heinrich et al. (2020) put it, it might be better to know that the s-factors "represent the part of a domain that cannot be explained by the reference facet, and not the part of a domain that cannot be explained by something that researchers do not know what it actually is, as is the case with *P*" (p.15). Burns et al. (2020) have applied the bifactor S-1 model successfully to ADHD and ODD symptom ratings in children, demonstrating the straight-forward interpretability the bifactor S-1 model has to offer in terms of understanding the meaning of g- and s-factors as well as their connections to each other that. Taken together, the bifactor S-1 model appears to be a promising modelling approach, combining several advantages of other latent factor models as it considers structural differences between domains, avoids anomalous results and permits a clear interpretability of all g- and s-factors and their connections with each other (Heinrich et al., 2020).

Comparing and contrasting all these different, frequently applied latent factor models regarding their advantages and disadvantages shows that all models have their merits and answer different kind of research questions. The decision for or against a model is often made based on so-called global model-fit indices, such as the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA) and the standardized root mean square residuals (SRMR), which all have different cut-offs, suggesting at what point model-fit is considered good or adequate (Browne & Cudeck, 1992; Hooper et al., 2008; Hu & Bentler, 1999). For bifactor models additional specific indices exist (Reise et al., 2013; Rodriguez et al., 2016). Solely relying on these global model fit indices is problematic, as for example bifactor models have the tendency to overfit, due to being less restrictive (Bonifay et al., 2017). It is therefore strongly recommended to compare different kinds of à priori defined, theoretically supported models with the hypothesized model in order to make an informed decision for or against a model (Brunner et al., 2012). This is in line with Fried et al. (2021), who suggest to formulate specific hypothesis regarding *p*. Yet, the inclusion of other models might add valuable information that should not be ignored and might even generate more detailed hypotheses for future studies.

Goal of this Dissertation

In my dissertation I first aim to apply the newly developed (Eid et al., 2018) bifactor S-1 model and compare it to a range of other latent factor models (first- and higher-order correlated factors models and the traditional bifactor model). The bifactor S-1 model has been successfully applied in a community sample of children showing symptoms of ADHD and ODD (Burns et al., 2020) and my goal is to examine the structure underlying symptoms of ADHD as well as ODD in a clinical sample. In order to do this, I take the two-factorial conceptualization as assumed in the DSM-5 (APA, 2013) and the three-factorial conceptualization of ADHD as assumed in the ICD-10/ICD-11, WHO (1992, 2020) into account. In addition, I focus on the information gain of this newly developed bifactor model (article 1, Junghänel et al., 2020). In a next step, my goal is to develop and lay out specific hypotheses as to why AD symptomatology can be considered the common underlying core of both these disorders (article 2, Junghänel et al., 2022). This approach is in line with Fried et al. (2021), who strongly recommended to precisely spell out and test theories of *p*. It is important to emphasize that this study can only be understood as a first step of many, with the idea and hope in mind, that eventually this kind of research will lead to the development of theoretically supported therapeutic interventions that can be applied in children with comorbid

ADHD and ODD, as the current picking and choosing of interventions from a large variety of treatments is neither particularly evidence-based nor resource-efficient for clinicians (Meier & Meier, 2018). This dissertation is meant to make AD gain center stage, while applying the newest methodology latent factor analysis has to offer in order to stimulate and guide further research aiming to identify a classification system of mental disorders that is more parsimonious (Junghänel et al., 2022).

Summary Article 1: Applying the Bifactor S-1 Model to Ratings of ADHD/ODD Symptoms: A Commentary on Burns et al. (2019) and a Re-Analysis.

Michaela Junghänel, Klaas Rodenacker, Christina Dose & Manfred Döpfner

Contribution of Michaela Junghänel and the co-authors regarding the following article:

Contribution of Michaela Junghänel:

Michaela Junghänel developed the concept for the current study, prepared the data for analysis, and analyzed, interpreted and visualized the data. She also integrated the findings into the scientific literature and independently wrote the first draft of the manuscript. Finally, she incorporated the suggestions of the co-authors and wrote the final version of the manuscript. Additionally, she incorporated revisions requested by the journal.

Contributions of the co-authors:

Support with data interpretation: Klaas Rodenacker

Critical examination and edit of the manuscript: Christina Dose and Manfred Döpfner

All authors have critically read, revised and accepted the final version of the manuscript.

Goal of Study

In this first article of my dissertation, I applied the newly developed bifactor S-1 model (Eid et al., 2018) to ratings of ADHD and ODD symptomatology in a clinical sample of children aged 6-18 years. In this article I had several goals. First, I wanted to compare and contrast results with those of Burns et al. (2020), who has applied a bifactor S-1 model to ratings of ADHD and ODD symptomatology in a community sample. As subtypes of ADHD might not be observable in mostly healthy samples compared to clinical samples (Lee et al., 2016) and since former research often reported weak s-factors in traditional bifactor models (Arias et al., 2016; Rodenacker et al., 2016; Ullebø et al., 2012; Wagner et al., 2016), I was interested to see if the s-factor in a clinical sample accounted for more variance compared to the s-factors in the community sample by Burns et al. (2020). Second, I wanted to examine if frequently reported anomalous factors loadings and collapsing s-factors in traditional bifactor models can be eliminated when applying this new bifactor S-1 approach, leading to a more straightforward interpretability. Third, whereas Burns et al. (2020) chose ADHD-HI as the reference facet, in line with the trait-impulsivity theory (Beauchaine et al., 2010; Beauchaine & McNulty, 2013) and the conceptualization of ADHD in the DSM-5 (APA, 2013), I additionally took the three-factorial structure of ADHD into account by comparing bifactor S-1 models with difference reference facets: ADHD-HI (in accordance with the two-factorial structure of ADHD of the DSM-5; APA, 2013) and ADHD-HY/ADHD-IM (in accordance with the three-factorial structure of ADHD in the ICD-10/11; WHO, 1992, 2020).

Methods

The sample consisted of children aged 6-18 years, of which over 90% had a diagnosis of ADHD and/or ODD (for sample characteristics see Rodenacker et al., 2018). I tested bifactor S-1 models with the three reference factors ADHD-HI, ADHD-HY and ADHD-IM. To assess global model fit I considered the global model fit indices CFI, TLI and RMSEA and interpreted them according to recommended cut-offs by Hu and Bentler (1999). Additionally, I considered the bifactor-specific omega values with the cut-off recommended by Reise et al. (2013) as a measure of reliability of all factors. As not provided in Burns et al. (2020) I calculated omega statistics from their factors loadings, in order to compare them with my results.

Key Findings

I was able to replicate the findings by Burns et al. (2020), as no anomalous factor loadings were observable in any of the three bifactor S-1 models. Model fit of all assessed bifactor S-1 models were adequate. When ADHD-HI was chosen as reference factor, model fit was slightly worse than when ADHD-HY or ADHD-IM were selected as reference factor. However, only in the bifactor S-1 model with ADHD-HI as reference factor (corresponding to the conceptualization of ADHD in the DSM-5; APA, 2013) were the omega-statistics of the remaining s-factors (ADHD-IN and ODD) above the recommended cut-off by Reise et al. (2013), making the model straight-forward to interpret and supporting their consideration as reliable subscales, when diagnosing ADHD and ODD. Compared to Burns et al. (2020), I found the s-factors in our clinical sample to explain a higher proportion of the true score variance. This was particularly pronounced for the s-factor ADHD-IN, suggesting that children scoring high on ADHD-IN and lower on ADHD-HI might be a clinically important subgroup, potentially characterized by sluggish cognitive tempo, as suggested elsewhere (Ullebø et al., 2012). To conclude, I found the bifactor S-1 model in which ADHD-HI was selected as the general reference facet and ADHD-IN and ODD as the s-factors to show the best fit to the data, to be straight-forward to interpret and to be consistent with theoretical deliberations of the trait-impulsivity theory (Beauchaine et al., 2010; Beauchaine & McNulty, 2013). It is remarkable, and speaks in favor of the stable structure of ADHD and ODD symptoms, that even though different samples (clinical vs. community) were used, the structure I found was highly comparable to Burns et al. (2020).

Limitations and Suggestions for Future Research

This study comes with several limitations that need to be addressed in future research. First, I have not included external correlates, which is crucial to establish construct validity. Second, models with other reference facets, based on different theoretical considerations regarding the high comorbidity of ADHD and ODD should be tested. For this, impulsivity in particular might be of importance as I found remaining correlations between the ODD and IM s-factors. Third, ODD needs to be examined in more detail by differentiating between its subdomains (Aebi et al., 2016; Burke et al., 2014, 2021; Stringaris & Goodman, 2009). Fourth, the observed structure needs to be validated in other samples.

Summary Article 2: Irritability and Emotional Impulsivity as Core Feature of ADHD and ODD in Children.

Michaela Junghänel, Ann-Kathrin Thöne, Claudia Ginsberg, Anja Görtz-Dorten, Franziska Frenk, Kristina Mücke, Anne-Katrin Treier, Sara Zaplana Labarga, Tobias Banaschewski, Sabina Millenet, Jörg M. Fegert, Dorothee Bernheim, Charlotte Hanisch, Michael Kölch, Anne Schüller, Ulrike Ravens-Sieberer, Anne Kaman, Veit Roessner, Julian Hinz, Manfred Döpfner on behalf of the ADOPT Consortium.

Contribution of Michaela Junghänel and the co-authors regarding the following article:

Contribution of Michaela Junghänel:

Michaela Junghänel developed the concept for the current study, participated in data collection and has prepared the data for the analyses of this study. She analyzed, interpreted and visualized the results. Additionally, she integrated the findings into the scientific literature and wrote the first version of the manuscript independently. She incorporated suggestions by the co-authors and wrote the final version of the manuscript. Additionally, she incorporated revisions requested by the journal.

Contribution of the co-authors:

Study conception (ADOPT Study): Anja Görtz-Dorten, Tobias Banaschewski, Jörg M. Fegert, Charlotte Hanisch, Michael Kölch, Ulrike Ravens-Sieberer, Veit Roessner, Manfred Döpfner

Data collection: Claudia Ginsberg, Franziska Frenk, Kristina Mücke, Sara Zaplana Labarga, Dorothee Bernheim, Anne Schüller, Anne Kaman und Julian Hinz.

Study coordination and data management: Anne-Katrin Treier und Sabina Millenet.

Support with data interpretation: Ann-Kathrin Thöne

Critical examination and edit of the manuscript: Ann-Kathrin Thöne und Manfred Döpfner

All authors have critically read, revised and accepted the final version of the manuscript.

Goal of Study

In this second article I focused on AD as a potential common core of ADHD and ODD. I first aimed to assess the structure of AD as previous research has suggested AD-related symptomatology as a transdiagnostic feature and a potential candidate for the p-factor (Carver et al., 2017; Evans et al., 2017). I hypothesized to find a strong factor of irritability and emotional impulsivity (AD-II) corresponding to the specifier suggestion by Evans et al. (2017), the conceptualizations of AD symptomatology as DMDD in the DSM-5 (APA, 2013) as well as to the chronic irritability specifier to the diagnosis of ODD in the ICD-11 (WHO, 2020). As I applied a broad conceptualization of AD, I was interested to see which smaller dimensions would appear in addition to AD-II. In this article I also aimed to address some of the limitations of the first article. First, I included external correlates (emotion regulation strategies; Parents Proxy Anger Scale; Irwin et al., 2012) to assess construct validity. Second, in addition to the hypothesized bifactor S-1 model in which AD-II was modeled as the general reference factor, I examined a variety of other models based on alternative theories (first- and higher-order correlated factors models, a traditional bifactor model, bifactor S-1 models entailing reference factors related to ADHD- and ODD-symptomatology) regarding the comorbidity of ADHD and ODD. By modelling AD-II as general reference facet I now also explicitly included emotional impulsivity. Third, I took the frequently found two dimensional structure of ODD into account (Aebi et al., 2016; Burke et al., 2014, 2021) in that the irritability dimension was included in our assessment of AD, whereas I added the defiant dimension of ODD as an additional s-factor. Fourth, I used a different sample, which was more representative as it was screened in the community.

Methods

Data were collected within the multi-center ADOPT (Affective Dysregulation in Childhood – Optimizing Prevention and Treatment) project (Döpfner et al., 2019) at the baseline measurement time point. The community-screened sample consisted of 391 children between the ages of 8 and 12, that showed either particularly high AD levels (in the highest 10% of all children initially screened) or particularly low AD levels (in the lowest 10% of all children initially screened). I first applied EFA to the AD symptomatology in 50% of the sample to establish an AD-structure, which I then cross-validated with a CFA in the remaining 50% of the sample. I then applied latent factor analysis to all dimensions of AD, ADHD and ODD and assessed a range of latent factor models (first-order correlated factors model,

unidimensional model, higher-order correlated factors model, traditional bifactor model, several bifactor S-1 models with differing reference factors). Global model fit indices (CFI, TLI, RMSEA, SRMR) with their corresponding cut-offs (Browne & Cudeck, 1992; Hooper et al., 2008; Hu & Bentler, 1999) as well as bifactor-specific indices (omega-statistics and explained common variance [ECV]) and if available their corresponding cut-offs (Reise et al., 2013) were used to help guide the decision process for or against a specific model. Correlations of all dimensions with emotion regulation strategies (adaptive/maladaptive) and the Parents Proxy Anger Scale (Irwin et al., 2012) were computed to assess construct validity.

Key Findings

I identified the hypothesized core dimension of AD-II and two minor AD factors connoting positive emotionality and exuberance, likely due to our broad conceptualization of AD. As shown by applying latent factor analysis, the hypothesized bifactor S-1 model in which AD-II was modeled as general reference factor fit the data best and was straight-forward to interpret (no anomalous factor loadings, good model fit). This was still true when comparing it to the remaining bifactor S-1 models with reference factors related to ADHD- and ODD-symptomatology, as these models showed statistical and/or theoretical problems. All dimensions showed differential correlations with emotion regulation skills and the Parent Proxy Anger Scale (Irwin et al., 2012), supporting construct validity. To conclude, AD-II could be the common core underlying ADHD and ODD symptomatology, explaining a large part of the variance shared between these two disorders (about 2/3), with defiant behavior, inattention and hyperactivity/impulsivity still explaining a major part of additional variance (all s-factors together explain about 1/3 of the variance). The specifier approach taken by the ICD-11 (WHO, 2020) is supported by our results, suggesting that the structure of psychopathology is more parsimonious than currently assumed. This leads me to suggest an extension to this specifier approach, by modelling an AD-II core, to which a range of specifiers such as “with inattention”, “with defiant behavior”, “with hyperarousal” could be added.

Limitations and Suggestions for Future Research

Though this study eliminates the limitations of the first article and comes with a number of strengths, certain limitations have to be mentioned. First of all, the included age range was very narrow (8-12 years of age). Second, and related to this point, is the cross-sectional design of the study as I cannot observe if for example the number of specifiers increase with age,

what the emergence of a specific specifier depends on (e.g. specific environmental influences) and what potential protective factors (e.g. adaptive emotion regulation skills) or risk factors (e.g. maladaptive emotion regulation skills; Beauchaine et al., 2010) could be for the emergence of additional specifier. Third, I used parent ratings scales to assess all symptoms, which might be a potential drawback. Fourth, no internalizing disorder was included into our analysis, which might be important, as AD has shown major associations with internalizing mental disorders, most frequently depression (Copeland et al., 2013; Evans et al., 2017). Fifth, additional relevant external correlates for the residual ADHD- as well as ODD-dimensions, such as for example sluggish cognitive tempo or violent behavior should be included and examined further. Sixth, the observed structure needs to be assessed in samples with alternative cultural and/or linguistic backgrounds.

Discussion

In my dissertation, I question the current categorical approach, applied when classifying mental disorders according to the DSM-5 (APA, 2013) and the ICD-10/ICD-11 (WHO, 1992, 2020) that comes along with comorbidity frequencies far above chance-level (Caspi et al., 2020; Newman et al., 1998) and contrast it with a more parsimonious, dimensional classification approach. My dissertation focuses on the externalizing spectrum of mental disorders in children and adolescents, as particularly ADHD and ODD are quite common in childhood and adolescence (Canino et al., 2010; Polanczyk et al., 2015) and are found to be comorbid frequently (Willcutt, 2012). In order to do this, I applied latent factor analysis and examined a bifactor S-1 model, which is a newly developed version of the traditional bifactor model, to externalizing symptom ratings of children and adolescents and compared it to other frequently applied latent factor models (first-order/higher-order correlated factors models, unidimensional models and traditional bifactor models). Traditional bifactor models have often been applied in search for the p-factor – the general factor of psychopathology, equivalent to the general factor of intelligence – however the combination of statistical issues, such as anomalous factors loadings, collapsing s-factors, mistakenly assumed interchangeability of domains (Eid et al., 2018; Heinrich et al., 2020) and theoretical problems, such as the unclear, often exploratory nature of the identified p-factor (Kotov et al., 2017) has frequently lead to interpretation difficulties and an inability to compare identified p-factors across studies. In my first article (Junghänel et al., 2020), I have applied a bifactor S-1 model to ratings of ADHD- and ODD-symptomatology in children and adolescents under routine care conditions and found this model to eliminate the above-mentioned statistical issue in addition to (by definition) the theoretical problem, as the general reference factor is chosen à priori. The two-dimensional conceptualization of ADHD (in line with the DSM-5; APA, 2013) proved to be more stable than the three-dimensional conceptualization of ADHD (in line with the ICD-10/ICD-11; WHO, 1992, 2020). The ADHD-HI domain was found to account for a significant part of shared variance between ADHD and ODD, whereas the s-factors ADHD-IN and ODD remained stable, important dimensions. In my second article (Junghänel et al., 2022), I focused on the structure of AD, and in particular the main AD component, irritability and emotional impulsivity and its potential role in explaining the frequent comorbidities between ADHD and ODD. Additionally, I addressed some of the major limitations of my first article, by including external correlates to assess construct validity, taking the frequently found two-dimensional structure of ODD into account (Aebi et

al., 2016; Burke et al., 2014, 2021), using a more representative sample and comparing results of a variety of latent factor models. Again, the bifactor S-1 model eliminated anomalous factor loadings and the clear definition of g- and s-factors allowed for a straight-forward interpretability, leading me to suggest AD-II as the common core of ADHD and ODD symptomatology, to which specifiers, such as "with inattention", "with defiant behavior" or "with hyperarousal" could be added.

Advantages and Disadvantages of the Bifactor S-1 Model

When applying and interpreting the bifactor S-1 model, it should be emphasized, that this model does not initially aim to identify *the* general factor of psychopathology, but instead models an à priori chosen reference domain, that is hypothesized to explain a major part of shared variance of all included symptoms. Additionally s-factors, hypothesized to account for additional variance are included in this model (Burns et al., 2020; Eid et al., 2018; Heinrich et al., 2020). The advantage of this model is the clear interpretability of the reference factor, which does not change in its interpretation depending on the included s-factors, as long as the items of the reference domain remain the same. This invites a comparison between studies. While abandoning the search for the general psychopathology factor might be disappointing at first, it needs to be pointed out, that the problem with current p-factor research is frequently, that the p-factor is determined in an explorative way, differs strongly among studies, depending on the symptoms included in the specific studies and is often of unclear nature for that reason (Eid et al., 2018; Heinrich et al., 2020; Kotov et al., 2017). With the bifactor S-1 model, we know exactly what the reference domain stands for. In addition, due to the elimination of anomalous factor loadings and collapsing s-factor we also know exactly what the additional s-factors stand for and we can even assess remaining partial correlations between the s-factors, as we do not assume that all correlations between s-factors are necessarily accounted for by the g-factor (Eid et al., 2018; Heinrich et al., 2020). This flexible, straight-forward approach might facilitate the search for common core factors, maybe even eventually *the one* common core factor (*if* it exists), explaining a large amount of variance that a range of disorders share, and with that offering an explanation for comorbidity. Pettersson et al. (2021) hypothesizes the p-factor to be of similar importance as the g-factor of general intelligence has shown to be as it might serve as a first reliable measurement for clinicians, indicating overall distress and helping them to predict prognosis and how much treatment might be needed. At the same time it is likely, that specific dimensions in addition to the general factor remain important and it is on us to examine which particular dimensions

these might be and which specific etiological or prognostic associations they might have (Hartman, 2021). The flexible approach of the bifactor S-1 model allows us to examine specific g- and s-factor in a top-down fashion, which lets us "lump" (into the g-factor) *and* "split" (into s-factors) where we need (Hartman, 2021). Consistent with this, Pettersson et al. (2021) hypothesizes the remaining s-factors to differentiate better between patients and to suggest a clearer target for treatment as compared to now, where individuals with a wide spread of symptoms likely display higher scores on a large amount of psychiatric scales. As we know exactly, what the reference factor represents, it would also be possible to combine it with the RDoC approach (Cuthbert & Insel, 2013; Insel et al., 2010) and examine potential neural circuits underlying this relevant dimension. This could be of particular importance as the p-factor has been suggested to be observable quite early on in life and to influence a range of additional symptoms appearing later in life (Caspi et al., 2014). Knowing about underlying neurological associations might eventually allow for an earlier prevention, before a clinically relevant symptom pattern or a number of comorbidities arise. In addition, clearly defined s-factor could potentially be associated with person-specific influences (Lahey et al., 2021) that might act as risk or protective factors for a specific combination of symptoms or symptom complexes (Hartman, 2021), again allowing for earlier prevention.

While in my first article (Junghänel et al., 2020), I only applied the omega-statistics as bifactor-specific indices with the cut-off recommended by Reise et al. (2013), I chose to additionally calculate the ECV and to focus less on the omega-cut-off values in the second article. The reasons for this being that I agree with Hartman (2021), who argues that we should *expect* s-factors in latent factor models (in the way they are currently assessed) to show lower factors loadings, larger standard error and less stability. According to Hartman (2021), this only reflects what we already know, namely, that after accounting for the variance of the dominant g-factor a "chaotic covariance structure of high instability remains" (p.72). One solution, if relying solely on omega-statistics and their cut-offs would be to dismiss s-factors in most cases as they are not reliable enough. Another way to look at it though is to understand the weaker s-factors as a result of inadequate measurements for these and to make it our task to develop strong measures, independent from the general factor, that are still reliable and valid (Hartman, 2021). The ECV has the advantage that it does not give us a yes/no answer of whether to include a specific s-factor or not, like the omega-statistics with its cut-offs indicating reliability, but provides us with a rough idea of how important this particular s-factor might be when accounting for the remaining variance. In an additional next

step, strong measures would have to be designed. Then a reassessment of the reliability of the s-factors, while at the same time accounting for the g-factor seems like a "fairer" approach for the s-factors. The development of stronger measures of relevant s-factors seems of crucial importance in order to assess specific associations with external correlates. This has to be our goal, if we are truly interested in establishing a more parsimonious approach of psychopathology, that is reliable and valid and gives us clearer instructions for necessary treatment.

Associations of ADHD, ODD and AD Dimensions

Results from both articles show, that ADHD and ODD were often comorbid with each other as well as with AD symptomatology (Junghänel et al., 2020, 2022). In article one (Junghänel et al., 2020), more than 90% of the children and adolescents had an ADHD and/or ODD diagnosis (42% ADHD only, 20% ODD only, 31% comorbid ADHD and ODD; Rodenacker et al., 2018). In article two (Junghänel et al., 2022), where I focused on the overlap of ADHD and ODD with AD symptomatology, 100% of children with an ODD diagnosis (93), as well as almost 100% of children with an ADHD diagnosis (61/62) showed strong AD symptomatology as well.

In both articles, ADHD and ODD shared a large part of common variance (Junghänel et al., 2020, 2022), in article one captured by ADHD-HI and in article two after further development of my hypotheses by AD-II. What both reference factors – ADHD-HI and AD-II – from both studies share is the strong impulsive component. The difference lies mainly in the nature of the impulsivity captured. In ADHD-HI, the assessed impulsivity is more physical in nature, resembling behavioral inhibition (Factor et al., 2014), compared to the rather emotional impulsivity assessed in AD-II (Junghänel et al., 2022). This is important, as in article one (Junghänel et al., 2020), we found remaining correlations between ADHD-IM and ODD in one of the bifactor S-1 models, suggesting an additional, potentially impulsive component responsible for the remaining association between ADHD-IM and ODD that was not adequately captured by the reference factor ADHD-HY. Berg et al. (2015) discuss the multidimensional nature of impulsivity in their review and call impulsivity an "umbrella concept, that refers to several conceptually and empirically separable traits […] associated with poor planning skills, difficulty maintaining attention, and risk-taking behaviors" (p.1129). The exact number and nature of these traits is yet to be determined (Berg et al., 2015), emotional impulsivity as one of these dimensions however, has been discussed in a number of studies and has been associated frequently with ADHD and ODD (Anastopoulos et

al., 2011; Barkley & Fischer, 2010; Factor et al., 2014; Rosen & Factor, 2015). Regarding the association with ADHD, Factor et al. (2014), point out that the current conceptualization of ADHD does not adequately capture emotional impulsivity, which has been shown to play an in important yet neglected role in ADHD, as it has shown strong associations with functional impairment (Anastopoulos et al., 2011) as well as comorbid diagnoses (mostly ODD) in addition to the ADHD diagnosis (Anastopoulos et al., 2011; Factor et al., 2014). In my second article (Junghänel et al., 2022), I therefore adapted the reference factor from ADHD-HI to AD-II, as the focus on *emotional* impulsivity instead of *physical* impulsivity appeared to account for the frequently found comorbidities between ADHD and ODD even better. As it accounted for 70% of the shared common variance of ADHD and ODD, it appears to be a promising candidate for the common core of these disorders (Junghänel et al., 2022).

The identified common core factor AD-II does not only consist of emotional impulsivity but also of irritability, a concept, closely associated with emotional impulsivity (Barkley & Fischer, 2010). The strong connection between irritability and ODD is rather obvious, as one of the ODD dimensions frequently identified is termed irritability (Burke et al., 2014, 2021; Rowe et al., 2010). More recently, the importance of irritability in children with ADHD has been highlighted as well. Eyre et al. (2017) found at least one irritability symptom in a vast majority of children with an ADHD diagnosis (53% showed the maximum of three symptoms) and 31% of children with an ADHD diagnosis to show a 3-month prevalence of DMDD. Karalunas et al. (2019) identified an ADHD+irritability subtype, neither reducible to the ADHD+ODD-, nor to the ADHD+DMDD-combination. The identified subtype showed high external validity and appeared to be more stable than the DMDD diagnosis, in addition, it was associated with an increased risk for negative outcomes (Karalunas et al., 2019). Stability over time and prognostic value of a diagnosis or a subtype are of crucial importance for clinicians and therefore for classification systems of mental disorders, which led Karalunas et al. (2019) to suggest the addition of an irritability specifier to the diagnosis of ADHD, similar to the chronic irritability specifier to the diagnosis of ODD, which currently captures AD symptomatology in the ICD-11 (WHO, 2020).

Taken together, both my studies show that impulsivity can capture a large part of the variance shared between ADHD and ODD. As impulsivity is a construct of multidimensional nature (Berg et al., 2015), the refinement of my hypothesis, to model a combination of *emotional* impulsivity and chronic irritability as the common core of ADHD and ODD

(Junghänel et al., 2022) appears to be promising in the light of previously discussed associations between emotional impulsivity as well as irritability with ADHD and ODD.

Whereas I assessed a clinical sample in my first article (Junghänel et al., 2020), my second sample consisted of children screened in the community, with either particularly high or particularly low AD levels (Junghänel et al., 2022). In both studies I found the s-factors to explain an important part of the remaining variance. According to the omega-values, the s-factors in the clinical sample were stronger, which is in line with Lee et al. (2016), stating that subtypes are more frequently observable in clinical samples. However, also in article two (Junghänel et al., 2022) we found the s-factors to account for a large part of additional variance (about 30% of the total variance). The two remaining ADHD components (ADHD-IN and ADHD-HI) explained the major part of this remaining variance (21% of the 30%). The s-factors ADHD-IN and ADHD-HI also showed strong partial correlations (r = .58), suggesting that an additional ADHD-specific component might exist, which I have not captured in my model. For future research it would be interesting to assess external correlations of the remaining s-factors, such as for ODD with violent behavior (Althoff et al., 2014) or disruptive, aggressive behavior (Burke et al., 2021) or for ADHD-IN with sluggish cognitive tempo (Hartman et al., 2004; Lee et al., 2016). Potentially, this would yield interesting results as to which items of the s-factors capture these residual dimensions best, ideally leading to the development of stronger, hence more stable, measures of the s-factors in question (Hartman, 2021).

Categorical versus Dimensional Classification of Mental Disorders

How do these results help us when attempting to answer the question whether a categorical or dimensional classification system is more appropriate? First, my results show that ADHD and ODD, two disorders frequently diagnosed in childhood and adolescence, share a large part of common variance, which can be interpreted as an indicator that a more parsimonious approach than the current approach of frequently diagnosing comorbid ADHD *and* ODD (Willcutt, 2012) seems justified (Junghänel et al., 2020; Junghänel et al., 2022). Second, my results suggest that emotional impulsivity and irritability, conceptualized as the main AD component AD-II, might be the common connecting core of these two externalizing disorders. When talking about dimensional classification systems, typically two points are implied, that I would like to discuss further.

The first aspect deals with identifying the relevant dimension(s) underlying a range of symptoms. In my case, this corresponds to the AD-II dimension explaining ADHD and ODD symptoms. I therefore suggest AD-II as a potential candidate for the p-factor (Caspi et al., 2014), though at this point our results are limited to ADHD and ODD symptomatology. A few aspects speak in favor of AD-II being a good p-factor candidate, even beyond ADHD- and ODD symptomatology. First, AD-II is similar to the suggested p-factor of *impulsive responsivity to emotion* by Carver et al. (2017), as they found "the majority of […] published associations for p [to be] consistent with a general tendency to react impulsively" (p.883). They suggest that people who are by disposition more reactive to emotions are also more vulnerable to psychopathology and often experience more difficulties in life in general, depending on a range of other factors. This emotion-related impulsivity has been associated with psychopathology particularly strongly compared to impulsivity that is not emotion-related (Berg et al., 2015; Carver et al., 2017). The second reason for AD-II being a good p-factor candidate is that the combination of emotional impulsivity with irritability has the potential to be even more strongly transdiagnostic in nature than emotional impulsivity or *impulsive reaction to emotion* on its own, as in particular irritability has been strongly associated with internalizing disorders as well (Copeland et al., 2013; Evans et al., 2017; Leibenluft, 2011; Waldman et al., 2021). Future research is needed to assess the associations between AD-II and depressive and anxious symptomatology in order to gain information regarding whether or not AD-II might serve as a good p-factor candidate, indicating liability for psychopathologies within the externalizing *and* the internalizing spectrum of mental disorders.

The second important aspect generally implied when referring to dimensional systems is that dimensional classification systems understand symptomatology to appear on a dimension (hence the name), ranging from "normal" to "highly pathological" behavior. This stands in contrast to categorical systems, such as the DSM-5 (APA, 2013) or the ICD-10/ICD-11 (WHO, 1992, 2020) understanding a mental disorder as being either present or absent. Though a combination of both approaches is possible by applying a cut-off to a dimensional symptomatology, in our case that might mean considering AD symptomatology as being either clinically relevant (if above the cut-off) or clinically irrelevant (if below the cut-off), or classifying AD symptomatology as mild, moderate or severe, it still shows a different underlying understanding of mental disorders. As Carragher et al. (2015) point out, the addition of severity level might serve "as a subtle reminder, that we are not dealing with

natural categories" here (p. 345). Regarding ODD, Barry et al. (2013) have suggested for ODD to be assessed and diagnosed along a continuum, as they conclude that ODD symptomatology is, to some extent, found in typical development as well, and does not constitute behavior that is *qualitatively* (but rather *quantitatively*) distinct. A similar finding for ADHD symptomatology has been observed by Marcus and Barry (2011), who conducted a set of taxometric analysis and found a dimensional latent structure of ADHD across a number of different analysis. This has also been suggested for AD symptomatology (Brotman et al., 2017). Though I may not draw any conclusions regarding whether a categorical or a dimensional diagnostic approach of AD-II is more useful as I did not specifically compare these two approaches, the positive correlations between AD-II symptomatology and maladaptive emotion regulation strategies in addition to negative correlations with adaptive emotion regulation strategies that I found indicate that increasing AD-II levels might be associated with an increasing amount of problems, which is in agreement with dimensional approaches (Junghänel et al., 2022). Future research is necessary to assess at what point on the identified AD-II-dimension cut-offs indicating the need for therapeutic support would be helpful for clinicians as well as patients.

What would specific advantages and disadvantages be of adopting this approach when diagnosing ADHD and ODD in the future? First, as mentioned before, the dimensional approach serves as a reminder that we are in fact dealing with natural categories (Carragher et al., 2015) instead of qualitatively distinct behavior, which might reduce stigma for children with mental health issues. Second, and strongly related, the elimination of comorbid diagnosis could further reduce stigma for affected children and their families. One can only imagine that being given the diagnoses ADHD *and* ODD stigmatizes a child more as a "problem child" than being told to show moderate or severe AD-II. Third, it might be more helpful for clinicians to know that a child shows severe AD-symptomatology, than being given a patient with the diagnoses ADHD and ODD, naturally leading to the question, where one should start therapeutically. In line with Pettersson et al. (2021) the separation in general factor and subfactors could be helpful for clinicians, as the general factor might indicate the amount of overall distress, give a first prognosis and suggest the intensity of treatment needed, whereas high subfactor scores could indicate suitable targets for further treatment. Fourth, knowing about the p-factor could lead to the development of early, transdiagnostic prevention programs (Caspi et al., 2020), potentially preventing or at least reducing impairment through mental disorders later in life. Caspi et al. (2020) report associations of the p-factor with poor

neurocognitive functioning already at the age of three, which would mean, that transdiagnostic prevention programs could be employed very early on in life. Delivering the identified AD-II dimension as a potential p-factor candidate to the RDoC research (Cuthbert & Insel, 2013; Insel et al., 2010) could lead to interesting findings regarding brain circuits underlying this dimension. There are also a number of disadvantages, or at least challenges, with my suggested approach. First, even if we find the clinically relevant dimensions, it would likely take some time until we as clinicians get used to applying them, as currently we are so used to thinking and diagnosing in categories (Carragher et al., 2015). Second, it will have to be demonstrated that employing dimensions instead of categories is clinically superior (Zimmerman, 2021). Third, there might be some disorders showing very unique features that cannot be captured adequately by the p-factor and might need differential treatment (Carragher et al., 2015). This problem however could be solved by applying subfactors or specifiers in addition to the p-factor (such as is possible in the bifactor S-1 model I favored in both my studies), which might indicate differences between patients even better after accounting for the shared variance captured by the p-factor (Pettersson et al., 2021).

In light of all discussed (dis-)advantages, I interpret my results in favor of a dimensional classification approach of mental disorders and suggest AD-II as a potential common core of ADHD and ODD, which should be explored more in regards to other (internalizing) mental disorders. In the following I will discuss limitations of both articles included in my dissertation and will make suggestions on how to address these in future research.

Limitations and Suggestions for Future Research

First, the samples in both articles were not representative. In the first article (Junghänel et al., 2020) a clinical sample was used in which over 90% of children and adolescents had an ADHD and/or ODD diagnosis. In the second article, the sample was more representative, however, children with particularly high (in the top 10%) of particularly low (in the bottom 10%) AD levels were selected from a large pool of children fulfilling inclusion criteria regarding age and current living situation. Additionally, even though all families with children between the ages of 8 and 12 were contacted, it is possible that the families returning the questionnaires differ systematically from the families not returning the questionnaire (i.e. in terms of time, attitude towards psychotherapy etc.). Systematically assessing these differences and examining potential associations with symptomatology would therefore be a necessary next step in order to make more general statements about the structure of

psychopathology in children and adolescents. The age range in this sample was also very narrow, as only children between the ages of 8-12 years were included. This will definitely have to be assessed in future studies, however it has been suggested that a large number of children and adolescents already show noticeable problems related to the p-factor early on in life (Caspi et al., 2020).

A second major limitation is the cross-sectional design of both studies. Longitudinal designs would be important to assess a few crucial points. If the p-factor is supposed to represent liability, it would necessarily have to be observable in some form or to some extent very early on in life. This would be in line with Caspi et al. (2020), who found poor neurocognitive functioning already at the age of three, that was associated with high p-factor scores later in life. RDoC-related research could be helpful to identify brain circuits underlying the suggested common core factor AD-II. With a longitudinal design it would be possible to assess if children, which later on show high scores on the AD-II dimension already differ systematically from other children at a young age. It might then also be possible to identify and select children at risk, which could be highly relevant, as it has been suggested that emotion-related impulsivity might predict symptom onset as well as progression (Carver et al., 2017). In addition, a longitudinal design would let us examine which person-specific factors might lead to further specifiers appearing later on in life (Lahey et al., 2021), with that identifying potential risk and protective factors for children showing a high AD-II score. If we knew more about potential risk and protective factors, effective prevention programs, for example for schools, could be established and employed. As I found AD symptomatology to strongly correlated with emotion (dys-)regulation, it would be interesting to see if children with high AD-II scores profit particularly strongly from prevention programs teaching a functional use of emotion regulation strategies.

Third, in both articles, the parent-ratings of the child's/adolescent's AD, ADHD and ODD symptomatology were used. This approach was chosen for the analyses, as most questionnaires employed are only validated for children and adolescents aged eleven or older, reason for this being, that younger children often encounter difficulties to report reliably on their own behavior. As studies assessing interrater-reliability have shown only moderate correspondence between self- and other informant reports (Achenbach et al., 1987; De Los Reyes et al., 2015), using a mix of questionnaires for our analyses filled out be the child/adolescent *and* the parent did not seem recommendable. It has long been recognized, that the integration of reports by multiple informants (e.g. child, parent, teacher) is crucial

when assessing psychopathology in children and adolescents, as it has been shown that the behavior of children varies strongly across contexts (De Los Reyes et al., 2013; Dirks et al., 2012), therefore making it improbable that a single informant could thoroughly and adequately inform about a child's symptomatology (Achenbach, 2020; De Los Reyes et al., 2013, 2015; Dirks et al., 2012). In the future it would be of great interest to examine if the observed structure of AD, ADHD as well as ODD symptoms can also be found when evaluated by the child/adolescent itself (i.e. is stable across multiple informants).

Fourth, latent factor analysis is only one method to understand comorbidity. Another very prominent approach with a different understanding of comorbidity is network analysis, which focuses on the relation between symptoms without modeling latent factors (Borsboom & Cramer, 2013). The main assumption here is that symptoms directly influence one another (Borsboom & Cramer, 2013; van Bork et al., 2017), and that symptoms which are common to several disorders (i.e. in our case items assessing irritability/emotional impulsivity) might consequently activate the networks of several disorders (i.e. ADHD *and* ODD; Borsboom, 2017; Cramer et al., 2010). This shows an entirely different understanding of comorbidity, as this approach does not understand comorbidity as an artefact of the diagnostic system, resulting from overlapping criteria (Cummings et al., 2014), but rather as an intrinsic feature of mental disorders (Borsboom, 2017). I did not compare these two approaches in my dissertation and it should be kept in mind that the existence of underlying latent factors (i.e. dimensions) is only one approach of understanding the comorbid presentation of two or more mental disorders.

Fifth, the reason for frequent unstable residual s-factors is yet unclear. In the first article (Junghänel et al., 2020) I found stable s-factors according to the recommended cut-off for the omega (hierarchical) statistics (Reise et al., 2013). However, the sole reliance on omega statistics would have led me to disregard the s-factors in the second article (Junghänel et al., 2022). Reasons for this observed instability is important. Are s-factors truly less stable, potentially because they are less genetically influenced than the p-factor (Lahey et al., 2021)? Or is the instability an artefact of the statistical organization of the data (Arseneault, 2021) and it has to be expected that the residual s-factors in a bifactor model *as they are currently assessed* are less stable and show lower factor loadings and larger standard errors (Hartman, 2021)? In the latter case, designing stronger measures of the residual s-factors, which demonstrate high internal construct validity would be necessary in order to examine clear association with other external correlates (Hartman, 2021).

Sixth, and already briefly addressed before, we did not include mental disorders within the internalizing spectrum (i.e. depression/anxiety) in our model. The separation between externalizing and internalizing spectrum when identifying a general liability factor of psychopathology might be unnecessary, as Caspi et al. (2020) found patients with *any* disorder at time point one to show an elevated risk for *all* other disorders at time point two, independent if the disorder was part of the internalizing or the externalizing spectrum. A lot of research, like this one, is cross-sectional in nature and in these cases, comorbidity can only be assessed if we understand it as two or more disorders being present at the same point in time or retrospectively. However, if we want to identify a general liability factor for psychopathology we would also be interested to examine which disorders appear over the course of a life-time, no matter if they are present at the same time or not. This would be particularly important if we are interested in developing early prevention programs for patients at risk to develop a comorbid disorder later in life. Previous research suggests that AD-II might be a suitable common core factor for internalizing disorders, as associations between mental disorders within the internalizing spectrum and irritability (Burke et al., 2021; Copeland et al., 2013; Evans et al., 2017) as well as cognitive impulsiveness in reaction to emotion (as opposed to other aspects of impulsiveness) have been reported (Carver et al., 2017).

Seventh, and this is a major limitation, it is of crucial importance to talk about whether or not this dimensional approach will help clinicians to provide better therapy to their patients – as this *has* to be the major goal of a good classification system. Opinions differ whether or not the bifactor approach has the potential to truly improve treatment for patients seeking psychological help. One the one hand, a dimensional approach could be very useful, as the clinical implication here is that it should be feasible to target the commonalities of two or more disorders with the same intervention (Meier & Meier, 2018). This would clearly be a great advantage as patients with different diagnoses could potentially be treated simultaneously in a group setting (at least as a first step in a stepped-care approach), as common elements underlying all these disorders would be targeted (Meier & Meier, 2018). In our case this would correspond to patients with high AD scores receiving group treatment as a first step, then patients with remaining problems and/or a number of additional specifiers (i.e. “inattention”) could proceed to more intensive/specialized training. The amount of treatments designed to be transdiagnostic has increased rapidly and promising results have been found when applying these to treat comorbid disorders (Neacsiu et al., 2014; Norton et al., 2004,

2013). One has to keep in mind though, that general factors such as empathy, positive outcome expectancy and goal setting might already be "transdiagnostic" in that they might ameliorate symptomatology of a large range of disorders, which does not depend on specific dimensions or categories targeted (Meier & Meier, 2018). This thought is in line with Zimmerman (2021), who expects similar outcomes between two groups assessed with categorical versus dimensional approaches, partly due to positive treatment responses in a large number of patients due to non-specific aspects of treatment. In addition to that, he highlights the existence of an additional group that is generally treatment resistant (no matter how they are diagnosed) and concludes that in his opinion due to these two crucial points, diagnostic precision through the application of a more accurate dimensional systems could improve outcome in 25% of patients at most. He further hypothesizes that due to a lack of difference in employed treatment in most cases, it will be hard to demonstrate superiority of the dimensional approach, which would be necessary in order to justify the extensive relearning process for clinicians associated with it. One further point of criticism regarding the use of dimensional systems compared to categorical systems has been mentioned by Arseneault (2021), who emphasizes that we do need to identify critical points (i.e. cut-offs), indicating at what point professional help is needed. While this is certainly true, it does not necessarily mean that dimensional models should be disregarded. First of all, this mainly targets one point of dimensional systems, namely the assumption that behavior ranges from "normal" to "highly pathological" and does not touch the second point, dealing with the identification of crucial dimensions. As outlined before, dimensional systems are a reminder that we are not dealing with natural categories – an important reminder, potentially reducing stigma of mental disorders. Flexibly adding cut-offs to a dimension, after learning about its association with impairment to decide at what point professional help is needed, is still a possibility when applying a dimensional approach.

From what we know so far, a model that summarizes information and retains specificity appears to be most promising (Arseneault, 2021; Hartman, 2021). This is exactly what the bifactor S-1 model does. Additionally, the combination of a dimensional approach with cut-offs, indicating the need for and the intensity of needed treatment appears reasonable. Once crucial dimensions of psychopathology have been identified and confirmed in a large number of different, representative samples, it will have to be assessed whether patients diagnosed with dimensional versus categorical classification systems receive (a) different treatment and most importantly (b) show a better outcome in terms of less impairment, less

comorbidity and less symptomatology. These are major steps that will likely take decades and were not all goals of this dissertation. The goal of this dissertation was to contribute to the identification of potential crucial dimensions of mental disorders, and I did this, by suggesting AD-II as a common core factor for ADHD and ODD, proposing additional specifier and laying out promising ideas for further research.

Summary

In this dissertation I focused on the classification system of mental disorders by examining associations between ADHD and ODD symptomatology in children, applying latent factor analysis.

In my first article (Junghänel et al., 2020), I applied a newly developed bifactor model (Burns et al., 2020; Eid et al., 2018) – the bifactor S-1 model – in a clinical sample of children aged 6-18 years of age. This model allows for the à priori definition of a general reference factor, which is opposed to the traditional bifactor model in which a general factor is extracted in a more exploratory way. I compared models with difference reference factors according the trait-impulsivity theory (Beauchaine et al., 2010; Beauchaine & McNulty, 2013) and the two- (DSM-5; APA, 2013) vs. three-dimensional (ICD-10/ICD-11; WHO, 1992, 2020) conceptualization of ADHD. Key findings were the following: First, the model showed no statistical problems, such as anomalous factor loadings or collapsing s-factor, frequently found in the traditional bifactor model, strongly facilitating its interpretation. As the general reference factor was defined à priori and associations between s-factors could be assessed, the bifactor S-1 model allowed for a straight-forward interpretation of all included dimensions and their associations with one another. Second, I found the model with ADHD-HI as the general reference factor, which is in line with the two-dimensional conceptualization of ADHD (DSM-5; APA, 2013) to fit the data best. Third, I found two additional stable s-factors, accounting for the remaining variance, constituting ODD and ADHD-IN symptomatology.

In the second article (Junghänel et al., 2022) I further elaborated my hypothesis regarding the general reference factor of ADHD and ODD by including AD symptomatology, with that also taking the multidimensional nature of impulsivity into account (Berg et al., 2015) by differentiating between physical and emotional impulsivity. In this article, I addressed several limitations of my first article. I included a more nuanced view of ODD symptomatology, by differentiation between the two frequently found dimensions of ODD-irritability and ODD-defiant behavior (Aebi et al., 2016; Burke et al., 2014; Rowe et al., 2010), used a more representative sample, assessed construct validity, and compared the bifactor S-1 models to a large range of other latent factor models. Key findings were the following: First, a main component representing irritability and emotional impulsivity emerged when assessing the structure of AD. In addition, I found two minor factors

constituting positive emotionality and exuberance. Second, the bifactor S-1 model with AD-II as general reference factor fit best in statistical and theoretical terms. About 2/3 (70%) of the variance shared between ADHD and ODD could be explained by AD-II. The remaining third of the variance (30%) were explained by all s-factors taken together. Third, differential correlations between g- and s-factors with emotion regulation skills and the Parent Proxy Anger Scale (Irwin et al., 2012) supported construct validity.

In sum, the bifactor S-1 model eliminated frequently found statistical problems and was clearly interpretable due to the general reference factor, which was defined à priori, as well as a result of the possibility to assess partial correlations between the residual s-factors. I found ADHD and ODD to share a large part of common variance that was mainly explained by irritability and emotional impulsivity. These results lead me to suggest AD-II as the common core of ADHD and ODD symptomatology, responsible for the frequently found comorbidities between them. In conclusion, I would like to propose an adaptation and extension of the specifier approach the ICD-11 (WHO, 2020) has already taken for ODD and AD symptomatology, by adding a specifier of chronic irritability to the ODD diagnosis. My suggestion is to understand AD-II as the core component of ADHD and ODD symptomatology. To this core, specifiers such as “with inattention”, “with hyperarousal” or “with defiant behavior” could be added. An examination regarding the extension of this approach into the internalizing spectrum, including mood and anxiety disorders seems promising. My results speak in favor of a more parsimonious, dimensional classification approach of mental disorders and will hopefully encourage further research in this area.

Zusammenfassung

Der Fokus meiner Dissertation lag auf den Klassifikationssystemen psychischer Störungen. Ich habe latente Faktorenanalyse angewandt, um die Assoziationen zwischen Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung (ADHS) und oppositionellem Trotzverhalten (ODD) zu untersuchen.

In meinem ersten Artikel (Junghänel et al., 2020) habe ich eine neu entwickelte Variante des Bifaktor Modells – das Bifaktor S-1 Modell (Burns et al., 2020; Eid et al., 2018) – in einer klinischen Stichprobe von Kindern im Alter von 6-18 Jahren angewandt. Bei diesem Modell ist die à priori Definition eines allgemeinen Referenzfaktors möglich, was im Gegensatz zum traditionellen Bifaktor-Modell steht, bei dem typischerweise ein allgemeiner Faktor auf eher explorative Art und Weise extrahiert wird. Ich habe Modelle mit verschiedenen Referenzfaktoren verglichen, die im Einklang mit der Trait-Impulsivitäts-Theory (Beauchaine et al., 2010; Beauchaine & McNulty, 2013), sowie der zwei- (DSM-5; APA, 2013) bzw. dreifaktoriellen (ICD-10/ICD-11; WHO, 1992, 2020) Konzeptualisierung von ADHS stehen. Dies waren die wichtigsten Ergebnisse: Erstens, keines der Bifaktor S-1 Modelle zeigte anormale Faktorladung oder kollabierende s-Faktoren, was beides häufig in traditionellen Bifaktor-Modellen vorkommt. Dies hat die Interpretation aller miteinbezogenen Dimensionen in Kombination mit dem à priori definierten Referenzfaktor und der hier möglichen Erfassung partieller Korrelationen zwischen den s-Faktoren stark vereinfacht. Zweitens, das Modell mit ADHS-Hyperaktivität/Impulsivität als Referenzfaktor, welches im Einklang mit der zweifaktoriellen Konzeptualisierung von ADHS (DSM-5; APA, 2013) steht, passte am besten auf die Daten. Drittens, ich habe zwei weitere stabile s-Faktoren gefunden, welche OPP und ADHS-Unaufmerksamkeit repräsentieren und die verbleibende Varianz erklären.

In meinem zweiten Artikel (Junghänel et al., 2022), habe ich meine Hypothese hinsichtlich des Referenzfaktors von ADHS und OPP weiter ausgearbeitet, indem ich Affektive Dysregulation (AD) miteinbezogen habe. Damit habe ich auch die mehrdimensionale Beschaffenheit von Impulsivität inkludiert (Berg et al., 2015) und habe im Folgenden zwischen eher körperlicher und eher emotionaler Impulsivität differenziert. In diesem Artikel habe ich zudem die Limitationen meines ersten Artikels adressiert. Dafür habe ich eine differenziertere Sichtweise auf OPP angewandt, welche zwischen den zwei häufig gefundenen Dimensionen OPP-Irritabilität und OPP-Trotzverhalten unterscheidet (Aebi et al., 2016; Burke et al., 2014; Rowe et al., 2010), eine repräsentativere Stichprobe verwendet, die Konstruktvalidität erfasst

und die Bifaktor S-1 Modelle mit einer Reihe von anderen latenten Faktorenmodellen verglichen. Dies waren die wichtigsten Ergebnisse: Erstens, bei der Untersuchung der Struktur von AD habe ich einen Hauptfaktor gefunden, der Irritabilität und emotionale Impulsivität (AD-II) darstellt, sowie zwei weitere kleine Faktoren, die positive Emotionalität und Überschwang darstellen. Zweitens, das Bifaktor S-1 Modell mit AD-II als Referenzfaktor konnte etwa 70% der gemeinsamen Varianz von ADHS und OPP erklären, während alle s-Faktoren zusammen die weiteren 30% erklären konnten. Drittens, Konstruktvalidität wurde durch unterschiedlich hohe Korrelationen der verschiedenen Dimensionen mit Emotionsregulationsfähigkeiten sowie der etablierten Parent Proxy Anger Scale (Irwin et al., 2012) gestützt.

Zusammengefasst zeigen meine Ergebnisse, dass das Bifaktor S-1 Modell häufig gefundene statistische Anomalitäten beseitigt und eindeutig interpretierbar ist, da der Referenzfaktor à priori definiert wird und die Möglichkeit besteht partielle Korrelationen zwischen den s-Faktoren zu berechnen. Zudem habe ich gefunden, dass ADHS und OPP einen großen Teil an Varianz teilen, der hauptsächlich durch Irritabilität und emotionale Impulsivität erklärt werden konnte. Ich schlage somit AD-II als gemeinsame Basis von ADHS und OPP vor, welche für die häufig gefundenen Komorbiditäten zwischen diesen zwei psychischen Störungen verantwortlich ist. Ich rege daher eine Erweiterung und Anpassung des Spezifikatoren-Ansatzes vor, welches die ICD-11 (WHO, 2020) bereits für OPP und AD Symptomatik etabliert hat, indem sich bei Bedarf die Spezifikation „mit chronischer Irritabilität“ der OPP Diagnose zuordnen lässt. Mein Vorschlag beinhaltet, AD-II als die Basis von ADHS und OPP Symptomatik zu verstehen, zu der sich dann weitere Spezifikatoren wie „mit Unaufmerksamkeit“, „mit Übererregbarkeit“ oder „mit Trotzverhalten“ bei Bedarf zuordnen lassen. Eine Untersuchung hinsichtlich der Erweiterung dieses Ansatzes auf das internalisierende Spektrum, inklusive affektiven- und Angststörungen scheint vielversprechend. Meine Ergebnisse sprechen sich für eine sparsamere, dimensionale Klassifikation psychischer Störungen aus und stimulieren hoffentlich weitere Forschung auf diesem Gebiet.

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Attachment: Publications, Supplementary Material, Eidesstattliche Erklärung

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Applying the Bifactor *S*-1 Model to Ratings of ADHD/ODD Symptoms: A Commentary on Burns et al. (2019) and a Re-Analysis

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Abstract

To examine the construct validity of attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD), the bifactor *S*-1 approach has been applied as an alternative to the fully symmetrical bifactor models in order to eliminate anomalous results and to allow for an unambiguous interpretation of g- and s-factors. We compared and contrasted our results with those of Burns et al. (2019) and extended their analyses by taking into account a two- vs. a three-factor structure of ADHD. Data from our previous research were reanalyzed and reinterpreted in accordance with the bifactor *S*-1 approach, constructing different models with hyperactivity (HY), impulsivity (IM) or hyperactivity/impulsivity (HI) as the general factor. No anomalous results were observed. All factor loadings were significant. Our results were comparable to those reported by Burns et al. (2019), although items from the specific subscales inattention (IN) and ODD accounted for more variance in our sample. Model fit for our HI model was comparable to that in Burns et al. (2019). In our sample, model fit was best when solely HY *or* IM was chosen as a general reference factor. However, in these cases, the remaining specific factor IM or HY was weakly defined. Overall, we were able to replicate the results found by Burns et al. 2019), although our factor loadings on the g-factor were slightly lower and our specificity regarding IN and ODD was slightly higher. Our results support a two-factor structure of ADHD/ODD in a clinical population.

Keywords Attention-deficit/hyperactivity disorder · Oppositional defiant disorder · Trait-impulsivity model · Bifactor models

With great interest, we read the article by Burns et al. (2019) on the application of a relatively new methodological approach, the bifactor *S*-1 model, to the factorial structure of symptoms of attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). In recent years, many research efforts have focused on examining ADHD and/or ODD ratings using so-called fully symmetrical bifactor models (cf. Arias et al. 2016; Burns et al. 2014). However, as Burns et al. (2019) point out, several authors have demonstrated that the application of fully symmetrical bifactor models to ratings of ADHD/ODD symptoms often leads to anomalous or inadmissible results, including (close-to-)zero or negative factor variances and/or factor loadings (Heinrich et al. 2018; Rodenacker et al. 2016, 2017, 2018; Ullebø et al. 2012). These anomalous results have made it difficult to unambiguously interpret the model-implied general factor (g-factor) and the specific factors (s-factors) as well as their relation to one another (for a deeper understanding of this problem, see Eid et al. 2017). In response to their criticism of applying fully symmetrical bifactor models to ratings of ADHD/ODD symptoms, Burns et al. (2019) propose applying the bifactor *S*-1 model for examining the hierarchical structure of ADHD/ODD symptoms, which clearly defines the meaning of the g-factor by modeling one of the first-order factors as general reference factor (for the items of this factor, no specific factor is specified). As a result, the g-factor in *S*-1 models has a different meaning than in fully symmetrical models. The g-factor in *S*-1 models describes how well the reference domain (e.g., hyperactivity/impulsivity [HI]) is able to predict the manifestation of individual differences in an s-factor (e.g., inattention [IN] or ODD). The s-factors (e.g., IN and ODD) account for a unique part of symptom variance that is not shared with the g-factor (e.g., HI).

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Applying both a fully symmetrical bifactor model and a bifactor *S*-1 model to mother, father and teacher ratings of ADHD/ODD symptoms in a large community sample of Spanish children, Burns et al. (2019) were able to show that – in contrast to the symmetrical bifactor model – the bifactor *S*-1 model did not yield any anomalous or inadmissible results and allowed for an unambiguous interpretation of the g-factor and the s-factors.

In our opinion, Burns et al. (2019) offer new and interesting insights into the structure of ADHD/ODD symptoms. We especially welcome this work as it enabled us to gain a new perspective on the results of our own previous research. In previous studies (Rodenacker et al. 2016, 2017, 2018), we first applied a fully symmetrical bifactor model to ADHD and ODD symptoms with either two specific ADHD symptom domain factors according to the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association [APA], 2013) or three specific ADHD symptom domain factors according to the 10th edition of the International Classification of Diseases (ICD-10, World Health Organization 1992). Due to anomalous results, we developed a so-called incomplete bifactor model by excluding the specific hyperactivity (HY) factor (Rodenacker et al. 2016, 2017, 2018). We chose this procedure as this specific factor did not account for any substantial true score variance over and above the shared common variance explained through the g-factor in the symmetrical model. Although the *S*-1 model and our incomplete bifactor model are nearly identical in statistical terms, there is a crucial difference concerning their development: While Burns et al. (2019) emphasize that the construction of their *S*-1 model was theory-driven, the development of our incomplete model was (at least in the beginning) data-driven. In our opinion, both approaches have their merits. The data-driven construction of new models may contribute to the development of new theories or to the refinement of existing theories. However, newly developed models surely have to be cross-validated in different samples and embedded in theoretical contexts. Hence, in our latest work (Rodenacker et al. 2018), we developed and tested a bifactor model not only based on the anomalous results of previous research, but also with reference to the trait-impulsivity theory. This theory also provides the theoretical for the bifactor *S*-1 model examined in Burns et al. (2019). It assumes that there are two different neural systems underlying ADHD symptoms (Beauchaine et al. 2010; Beauchaine and McNulty 2013). First, there is the mesolimbic reward pathway, which in the case of dysfunction leads to the development of hyperactivity/impulsivity symptoms. Based on this pathway, inattention symptoms develop only secondarily. Depending on the presence of environmental risk factors, dysfunctions in this pathway might also increase the risk of developing ODD and conduct disorder (CD) later in life. The second pathway concerns frontal structures. Dysfunctions in this pathway might lead directly to the inattention symptoms typical for ADHD, but are not associated with trait-impulsivity or an increased risk of developing ODD or CD. Burns et al. (2019) chose the HI factor as reference factor in their bifactor *S*-1 model, as this choice is consistent with the trait-impulsivity theory and because previous research on the factor structure of ADHD frequently found the specific HY and/or the impulsivity (IM) factor to collapse or to be inadequately defined (Burns et al. 2014; Rodenacker et al. 2016, 2017, 2018; Ullebø et al. 2012). This model is consistent with the two-domain conception of ADHD symptoms in the DSM-5.

Our incomplete model differs in three main points from the *S*-1 model of Burns et al. First, in contrast to Burns et al. (2019), we did not consider HI as common factor, but drew an additional distinction between an HY and IM factor, consistent with the three-domain conception of ADHD in the ICD-10. We excluded the specific HY factor, which was the psychometrically worst latent variable in previous research, from the model. As a result, the items of the HY domain were only allowed to load on the g-factor in our model. Second, as Burns et al. (2019) correctly argued, so far, we have not adapted the interpretation of our g-factor when constructing an incomplete instead of a fully symmetrical bifactor model (the g-factor in our model needs to be interpreted as an HY general reference factor). Third, the sample used by Burns et al. (2019) consisted of a randomly selected group of students, whereas we analyzed a clinical sample of children and adolescents aged 6–18 years, in which more than 90% of the patients were diagnosed with ADHD and/or ODD (Rodenacker et al. 2018). Thus, the results found by Burns et al. (2019) concern the factor structure of ADHD/ODD symptoms in a predominantly healthy population, whereas we may draw conclusions about the factor structure in a clinical population of children and adolescents with ADHD and/or ODD. S-factors might account for more variance in a clinical sample, in which distinct subtypes of ADHD have been identified (Lee et al. 2016). However, in non-clinical samples, most previous analyses of bifactor models have found a strong g-factor and weakly defined ADHD subdomains (Arias et al. 2016; Ullebø et al. 2012; Wagner et al. 2016), often only accounting for a small amount of variance after controlling for the variance explained by the g-factor (e.g. Arias et al. 2016; Lee et al. 2016; Rodenacker et al. 2016; Wagner et al. 2016).

Reanalysis in a Clinical Sample

We reanalyzed and reinterpreted our data from Rodenacker et al. (2018) based on this new insight regarding *S*-1 models as an alternative to fully symmetrical bifactor models provided by Burns et al. (2019) (for sample characteristics, see the original article). We aimed to compare and contrast our results with those of Burns et al. (2019), to extend the research on the

factor structure of ADHD and ODD by examining and comparing bifactor *S*-1 models consistent with the DSM-5 versus the ICD-10 conception of ADHD and ODD and to consider the results from a broader theoretical perspective. As we used a clinical sample, we expected that our results might differ from those reported by Burns et al. (2019) regarding the variance accounted for by the g-factor vs. the s-factors. It is conceivable that the s-factors are of higher relevance in a clinical sample and explain more variance, as distinct subtypes that have been identified (Lee et al. 2016) might not be observable in a mostly healthy sample.

We analyzed different models, with either HY, IM or HI as the general reference factor. All analyses were conducted using Mplus 8 (Muthén and Muthén 2017). We compared and contrasted model fit indices, factor loadings and true score variances from our original models (first-order model with three correlated factors: IN, HI, ODD, fully symmetrical bifactor model with three s-factors: IN, HI, ODD, incomplete bifactor model with three s-factors: IN, IM, ODD [in this model, the former HI factor was divided into an HY and an IM factor and only the HY factor was chosen as general reference factor]; Rodenacker et al. 2018), our new bifactor *S*-1 models and the bifactor *S*-1 model by Burns et al. (2019). As Burns et al. (2019) did not provide the true score variances, we computed them from the factor loadings given in their article for the purpose of comparison with our models. Burns et al. (2019) tested their *S*-1 model in subsamples of mothers, fathers and teachers. Unless otherwise indicated, we chose the results for the mother sample as comparison, as most of the ratings were provided by mothers in our sample. If not mentioned otherwise, the models for mothers and fathers in Burns et al. (2019) did not differ significantly from one another in matters relevant for this comparison.

To evaluate model fit, we considered the Comparative Fit Index (CFI), the Root Mean Square Error of Approximation (RMSEA), and the Tucker-Lewis Index (TLI). As suggested in Hu and Bentler (1999), model fit was considered as good when the RMSEA was equal to or below 0.05 and the TLI and CFI were equal to or above 0.95. Model fit was considered as adequate in the case of an RMSEA value below 0.08 and a TLI and CFI value above 0.90.

To further evaluate the different models, we used Omega statistics as measures of reliability of the g-factor and the s-factors. In a bifactor model, Omega (ω) describes the amount of variance in all items or item subsets accounted for by the g-factor and the s-factors taken together. Omega hierarchical displays the amount of variance in all items or item subsets accounted for by either the g-factor or an s-factor (cf. Brunner et al. 2012; Reise 2012). We use the abbreviation ω_H to indicate the amount of variance attributable to the g-factor and the abbreviation ω_S to indicate the amount of variance attributable to an s-factor. As Burns et al. (2019) did not provide Omega statistics, we computed them from the factor loadings given in their article for the purpose of comparison with our models. Reise et al. (2013) recommend a ω_H value regarding the g-factor and ω_S values regarding the s-factors of 0.50 or preferably 0.75.

Detailed tables displaying the results of our reanalyses regarding model fit, factor loadings and omega statistics, true score variance and consistency and specificity are provided in the online supplement of this article (Tables S1 to S4). Notably, and similar to the results of Burns et al. (2019), we did not have any problems with anomalous results such as negative or non-significant factor loadings in any of the three bifactor *S*-1 models that we examined. Thus, we were able to replicate the finding that the application of the *S*-1 model helps to eliminate these results and to facilitate the interpretation of the models. All of our three bifactor *S*-1 models provided an adequate fit to the data, with the models with HY or IM as reference domain demonstrating slightly better model fits than the model with HI as reference domain (Table S1). All factor loadings in these three models were significant, and reliability was good in all models, with ω values regarding the respective g-factor between 0.95 and 0.96 and for the s-factors ranging from 0.88 to 0.98. The s-factors IN and ODD were well defined in all models, with ω_S values between 0.58 and 0.71 for IN and 0.68 and 0.79 for ODD. In the models using IM or HY, respectively, as reference factor, the reliability of the specific HY and IM domains as indicated by ω_S remained, with 0.39 and 0.41, below the recommended cut-off of 0.50 (Reise et al. 2013). For the items of the IN domain, the s-factor explained much more variance than the g-factor in all three models (Table S2). This is interesting, as the IN subdomain has been found to be associated with sluggish cognitive tempo (SCT), while the inattention part of ADHD represented through the g-factor has not (Lee et al. 2016). This suggests that the specific inattention part, represented by the subdomain, captures different, highly relevant aspects of inattention. Ullebø et al. (2012) found similar results regarding the IN subdomain and also suggest that individuals with high scores on the inattention subdomain and low scores on the general hyperactivity factor might represent a clinically important subgroup, being characterized by SCT. Along with the finding that the IN domain accounts for a large amount of reliable variance, this is in accordance with the trait-impulsivity theory, as Burns et al. (2019) already stated in their article.

Although the model with HI as reference factor had a slightly worse fit compared to the models using either HY or IM as reference factor, its fit was still in an adequate range and it had additional advantages. This model did not yield any weakly defined s-factors and is straightforward to interpret regarding the core component of ADHD as defined by the DSM-5. The low ω_S values of the HY and IM factors when the respective other one was modeled as the general reference factor support the idea that they share a large amount of variance, which is already mostly accounted for by the g-factor, and that they should therefore not be modeled as distinct subdomains, but rather be considered as one factor according to the DSM-5. In

conclusion, we favor a bifactor *S*-1 model with HI as general reference factor and the two s-factors IN and ODD, due to its statistically sound structure, the straightforward interpretability, and the consistency with theoretical considerations of the trait-impulsivity theory. This is the bifactor *S*-1 model equivalent to the one Burns et al. (2019) examined in their study.

The fragile nature of the specific HY and IM factor that we found in the models using IM or HY, respectively, as reference factor is in line with the results presented by Burns et al. (2019) for their fully symmetrical bifactor model. The authors report that the anomalous loading patterns in their fully symmetrical bifactor model differed across raters, meaning that different components were affected for mothers and fathers (HY for mothers vs. IM for fathers), leading to a differing meaning of the g-factor across sources. By eliminating these anomalous results using the bifactor *S*-1 approach, the interpretation of the g-factor and the s-factors is much clearer, does not vary across sources and is therefore comparable between studies.

Compared to Burns et al. (2019), we found lower loadings on the g-factor (Table S2) and, accordingly, a lower true score variance for items from all domains (Table S3). Moreover, we found the consistencies of the specific items to be lower and the specificities to be higher (Table S4), which was also reflected in the ω_H and ω_S values for the separate subdomains. This means that in our clinical sample, the s-factors explained a higher proportion of the true score variance than they did in the sample of Burns et al. (2019).

As we applied the same statistical method as Burns et al. (2019), but used a different sample**,** differences regarding the true score variance as well as the consistencies and specificities might be due to the different manifestations of ADHD and ODD symptomatology in clinical vs. community samples. Whereas Burns et al. (2019) examined the factor structure of ADHD and ODD in a mostly healthy sample, in our sample, more than 90% of the patients were diagnosed with ADHD and/or ODD. Given this great difference between the two samples, the similarity of the results is still remarkable, and indicates a highly stable factor structure of ADHD and ODD over the entire symptom spectrum.

Compared to data-driven approaches, a major advantage of testing theory-driven models as in Burns et al. (2019) lies in the more straightforward interpretation of the g-factor, as it is a priori and unmistakably defined. When constructing bifactor *S*-1 models, these considerations are especially important, as the choice of the appropriate reference factor is highly relevant. However, the trait-impulsivity theory is just one of several theories aiming to explain the underlying core dysfunctions and etiology of ADHD. According to this theory, the core component of ADHD differs between subgroups and could be either HI (following dysfunctions in pathway one) or IN (following dysfunctions in pathway two). As such, it is challenging to accurately capture both subgroups in one bifactor *S*-1 model, in which a single reference domain has to be chosen. It is important to keep in mind that other cognitive or motivational theories of ADHD (for a detailed overview see Sonuga-Barke 2004), would potentially suggest other symptom groups as the general reference domain, or, in the case of the dual pathway theory (Sonuga-Barke 2003), would make it difficult to decide on one symptom group to be taken as a general reference factor, as they suggest that both are equally important in the development of ADHD.

Conclusion

In conclusion, we welcome the study by Burns et al. (2019) to help to shed more light on the factor structure of ADHD and ODD and to introduce a methodologically sound bifactor model to the psychological research on ADHD and ODD. Similarly to our previously introduced incomplete bifactor model (Rodenacker et al. 2018), the construction of the bifactor *S*-1 models eliminates anomalous results and additionally facilitates the interpretation of the results by suggesting a clear interpretation of the g-factor and its relation to the s-factors. In the present analyses, overall, we were able to replicate the findings of Burns et al. (2019) regarding the elimination of anomalous results, model fit indices, factor loadings and true score variances in a bifactor *S*-1 model of ADHD and ODD symptomatology with HI as general reference factor. Similar to Burns et al. (2019), we found that the specific IN and ODD factors accounted for a large amount of reliable variance, which supports the consideration of the respective subscales when diagnosing ADHD and ODD. Nevertheless, our factor loadings and true score variances were generally slightly lower and our s-factors accounted for more true score variance compared to Burns et al.'s (2019) findings, which might be due to the different samples. In our analyses, we additionally aimed to disentangle the two- vs. three-factor structure of ADHD. Although the model fit indices for the models with either HY or IM as reference factor were slightly better than those for the model with HI as reference factor, we found that the s-factors HY or IM were weakly defined when only one of them was chosen as reference factor. Our results therefore support previous findings regarding the generally fragile nature of the HY and IM factor (Arias et al. 2016; Rodenacker et al. 2016, 2017, 2018; Ullebø et al. 2012; Wagner et al. 2016).

Future research should extend the recent findings in several important ways. First, it would be helpful to more closely examine external correlates, such as SCT and social and academic impairment, which could establish construct validity through distinct correlates with the subdomains. Burns et al. (2019) have already assessed social and academic impairment as well as peer rejection and found unique correlations with the subdomains, albeit not consistently across raters. Second, models based on other theories should be tested, such as with IN as the core component of ADHD, which would be

consistent with the second pathway of the trait impulsivity theory, or based on other cognitive theories of ADHD. Third, it might be interesting to examine ODD more closely; that is, to differentiate between the subdomains "headstrong", "irritable" and "vindictiveness" (Stringaris and Goodman 2009) and to ascertain whether or not distinct correlations can be found. In our models with HY as reference factor, we found a moderate correlation between the s-factors IM and ODD. While this correlation has to be interpreted with caution due to the weakly defined IM factor, this result is nevertheless quite interesting, as impulsivity can be seen as a bridge between ADHD and ODD. Impulsivity plays a major role in both disorders but is usually expressed in different ways (intruding/interrupting in ADHD vs. emotional impulsivity in ODD). The remaining correlation between these two domains suggests that there might be an underlying factor responsible for both kinds of impulsivity that goes beyond the variance already explained through the general HY factor. Future research incorporating a more differentiated factor structure of ODD could shed more light on the interpretation of the impulsivity factor and the relation between ODD and ADHD symptomatology. Fourth, further research is necessary to establish whether results concerning the factor structure of ADHD and ODD are stable across different samples regarding age, gender and symptom manifestations as well as across different raters (father vs. mother vs. teacher vs. self-rating). The bifactor *S*-1 approach applied in Burns et al. (2019) makes it possible to reliably draw comparisons across studies, ultimately allowing for broader statements regarding the factor structure of ADHD in different samples. This is a major advantage for research.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval and Informed Consent The parents of all participating children and adolescents provided informed consent, with ethical approval for the study obtained from the Ethics Committee of the University Hospital of Cologne.

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Table S1

Model Fit Indices and Correlations Between the First-Order Factors or the Specific Factors

Model (Reference factor)	CFI	RMSEA (90% CI)	TLI	Significant correlations
		Models examined by Rodenacker et al. (2018)		
First order model with correlated factors	-	.075 (.072, .078)	.92	IN-HI: .57 IN-ODD: .26 HI-ODD: .50
Fully symmetrical bifactor model	-	.060 (.058, .063)	.95	n/a
Incomplete bifactor model	-	.064 (.061, .066)	.94	n/a
		S-1 model by Burns et al.		
HI	.97	.067 (.065, .070)[a]	-	none
		Newly constructed *S*-1 models		
HI	.93	.071 (.067, .074)	.92	none
HY	.96	.053 (.049, .057)	.96	IM-ODD: .36
IM	.96	.057 (.053, .061)	.95	IN-HY: .41

Note. CFI = comparative fit index, RMSEA = root mean square error of approximation, CI = confidence interval, TLI = Tucker Lewis index, IN = inattention, HI = hyperactivity/impulsivity, ODD = oppositional defiant disorder, HY = hyperactivity, IM = impulsivity, n/a = not applicable (correlations in the respective model were constrained to zero).

[a] results for the mother sample.

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Table S2

Standardized Factor Loadings (Standard Error) and Omega Statistics for all Bifactor S-1 Models

Symptoms	HI-model			HY-model				IM-model				HI-model (Burns et al.)		
	G-HI	S-IN	S-ODD	G-HY	S-IM	S-IN	S-ODD	G-IM	S-HY	S-IN	S-ODD	G-HI	S-IN	S-ODD
ADHD-HY														
Fidgets/Squirms	.73 (.02)			.76 (.02)	-			.53 (.03)	.60 (.04)			.87 (.01)		
Leaves seat	.75 (.02)			.78 (.02)	-			.57 (.03)	.55 (.04)			.90 (.01)		
Runs/Climbs	.82 (.02)			.85 (.02)	-			.67 (.03)	.52 (.04)			.86 (.01)		
Loud/Noisy	.76 (.02)			.80 (.02)	-			.63 (.03)	.48 (.04)			.81 (.01)		
Driven/On the go	.70 (.03)			.73 (.03)	-			.60 (.03)	.39 (.04)			.89 (.01)		
ADHD-IM														
Talks too much	.61 (.03)			.43 (.03)	.58 (.04)			.65 (.03)	-			.74 (.01)		
Blurts	.69 (.02)			.57 (.03)	.46 (.03)			.73 (.02)	-			.84 (.01)		
Does not wait turn	.81 (.02)			.65 (.03)	.56 (.03)			.85 (.02)	-			.87 (.01)		
Interrupts/Intrudes	.85 (.02)			.69 (.03)	.60 (.03)			.90 (.01)	-			.84 (.01)		
ADHD-IN														
Close attention	.28 (.04)	.70 (.02)		.30 (.04)		.69 (.03)		.20 (.04)		.72 (.02)		.54 (.02)	.64 (.01)	
Sustain attention	.56 (.03)	.55 (.03)		.60 (.03)		.51 (.03)		.48 (.03)		.63 (.03)		.59 (.02)	.71 (.01)	
Listen	.52 (.03)	.44 (.03)		.55 (.03)		.41 (.03)		.46 (.04)		.50 (.03)		.64 (.02)	.56 (.02)	
Follow through	.42 (.03)	.77 (.02)		.44 (.03)		.75 (.02)		.32 (.04)		.81 (.02)		.61 (.02)	.64 (.02)	
Organization	.39 (.03)	.60 (.03)		.41 (.03)		.59 (.03)		.32 (.04)		.64 (.03)		.55 (.02)	.70 (.02)	
Avoids tasks	.41 (.04)	.67 (.03)		.43 (.04)		.66 (.03)		.33 (.04)		.70 (.03)		.55 (.02)	.69 (.02)	
Loses things	.27 (.04)	.54 (.03)		.28 (.04)		.54 (.03)		.21 (.04)		.56 (.03)		.54 (.02)	.57 (.02)	
Easily distracted	.58 (.03)	.53 (.03)		.62 (.03)		.50 (.03)		.52 (.03)		.60 (.03)		.62 (.02)	.68 (.01)	
Forgetful	.30 (.04)	.56 (.03)		.32 (.04)		.56 (.03)		.24 (.04)		.59 (.03)		.54 (.02)	.70 (.02)	

Symptoms	HI-model			HY-model				IM-model				HI-model (Burns et al.)		
	G-HI	S-IN	S-ODD	G-HY	S-IM	S-IN	S-ODD	G-IM	S-HY	S-IN	S-ODD	G-HI	S-IN	S-ODD
ODD														
Argues	.31 (.04)		.65 (.03)	.24 (.05)			.68 (.03.)	.35 (.04)			.63 (.30)	.55 (.02)		.60 (.02)
Loses temper	.28 (.04)		.76 (.02)	.21 (.05)			.78 (.02)	.32 (.04)			.74 (.03)	.61 (.02)		.61 (.02)
Defies/Refuses	.43 (.04)		.60 (.03)	.39 (.04)			.63 (.03)	.47 (.04)			.58 (.03)	.63 (.02)		.57 (.02)
Annoys others	.38 (.04)		.60 (.03)	.33 (.04)			.63 (.03)	.43 (.04)			.57 (.04)	.61 (.02)		.53 (.02)
Blames others	.40 (.04)		.55 (.03)	.35 (.05)			.58 (.03)	.44 (.04)			.52 (.03)	.59 (.02)		.57 (.02)
Annoyed by others	.37 (.04)		.76 (.02)	.28 (.05)			.80 (.02)	.41 (.04)			.74 (.03)	.56 (.02)		.60 (.02)
Angry/Resentful	.38 (.04)		.84 (.02)	.29 (.04)			.87 (.02)	.42 (.04)			.82 (.02)	.51 (.02)		.70 (.02)
Spiteful/Vindictive	.30 (.05)		.71 (.03)	.24 (.05)			.72 (.03)	.33 (.05)			.69 (.04)	.47 (.03)		.66 (.02)
ω	.95	.91	.92	.95	.88	.91	.92	.96	.98	.91	.92	.98[a]	.97[a]	.95[a]
ωH	.72	.30	.20	.47	.47	.33	.13	.38	.59	.20	.24	.82[a]	.42[a]	.44[a]
ωS	-	.62	.73	-	.41	.58	.79	-	.39	.71	.68	-	.55[a]	.50[a]

Note. g = general factor, s = specific factor, HY = hyperactivity, IM = impulsivity, HI = hyperactivity/impulsivity, ADHD = attention-deficit/hyperactivity disorder, ODD = oppositional defiant disorder, ω = omega (amount of variance accounted for by the g-factor and the s-factors taken together), ωH = omega hierarchical general (amount of variance accounted for by the g-factor), ωS = omega hierarchical subscale (amount of variance accounted for by the s-factors).

[a]not presented in original article by Burns et al.; computed based on the factor loadings reported there

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Table S3

True Score Variance (Standard Error) for all S-1 Models

Symptoms	Burns et al.[a]	Our models		
	HI-model	HI-model	HY-model	IM-model
ADHD-HI				
Fidgets/Squirms	.76	.53 (.03)	.58 (.03)	.65 (.04)
Leaves seat	.81	.56 (.03)	.60 (.03)	.63 (.03)
Runs/Climbs	.74	.67 (.03)	.73 (.03)	.72 (.03)
Loud/Noisy	.66	.58 (.03)	.64 (.03)	.63 (.03)
Driven/On the go	.79	.49 (.04)	.53 (.04)	.51 (.04)
Talks too much	.55	.37 (.03)	.52 (.04)	.42 (.03)
Blurts	.71	.47 (.03)	.53 (.03)	.54 (.03)
Does not wait turn	.76	.65 (.03)	.73 (.03)	.73 (.03)
Interrupts/Intrudes	.71	.72 (.03)	.82 (.03)	.82 (.03)

Symptoms	Burns et al.[a]	Our models		
	HI-model	HI-model	HY-model	IM-model
ADHD-IN				
Close attention	.70	.57 (.03)	.57 (.03)	.56 (.03)
Sustain attention	.85	.62 (.03)	.63 (.03)	.63 (.03)
Listen	.72	.46 (.03)	.47 (.03)	.47 (.03)
Follow through	.78	.76 (.02)	.76 (.02)	.76 (.02)
Organization	.79	.51 (.03)	.52 (.03)	.51 (.03)
Avoids tasks	.78	.61 (.03)	.61 (.03)	.61 (.03)
Loses things	.62	.37 (.03)	.37 (.03)	.36 (.03)
Easily distracted	.85	.62 (.03)	.63 (.03)	.63 (.03)
Forgetful	.78	.41 (.03)	.41 (.03)	.40 (.03)
ODD				
Argues	.66	.51 (.04)	.52 (.04)	.51 (.04)
Loses temper	.74	.65 (.03)	.65 (.03)	.65 (.03)
Defies/Refuses	.72	.55 (.03)	.55 (.03)	.55 (.03)
Annoys others	.65	.51 (.03)	.51 (.03)	.51 (.03)
Blames others	.67	.46 (.04)	.47 (.04)	.47 (.04)
Annoyed by others	.67	.72 (.03)	.72 (.03)	.72 (.03)
Angry/Resentful	.75	.85 (.02)	.85 (.02)	.85 (.02)
Spiteful/Vindictive	.66	.59 (.04)	.58 (.04)	.59 (.04)

Note. HI = hyperactivity/impulsivity, HY = hyperactivity, IM = impulsivity, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, ODD = oppositional defiant disorder.

[a] not presented in original article by Burns et al.; computed based on the factor loadings reported there.

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Table S4

Consistency-Specificity Values for ADHD-IN and ODD Symptoms in the HI-Models

Symptoms	HI-model Burns et al.		Our HI-model	
	Consistency	Specificity	Consistency	Specificity
ADHD-IN				
Close attention	.41	.59	.14	.86
Sustain attention	.40	.60	.51	.49
Listen	.56	.44	.58	.42
Follow through	.47	.53	.23	.77
Organization	.39	.61	.30	.70
Avoids tasks	.39	.61	.27	.73
Loses things	.47	.53	.20	.80
Easily distracted	.45	.55	.54	.46
Forgetful	.38	.62	.22	.78
ODD				
Argues	.45	.55	.19	.81
Loses temper	.50	.50	.12	.88
Defies/Refuses	.55	.45	.34	.66
Annoys others	.57	.43	.29	.71
Blames others	.52	.48	.35	.65
Annoyed by others	.46	.54	.19	.81
Angry/Resentful	.35	.65	.17	.83
Spiteful/Vindictive	.34	.66	.15	.85

Note. HI = hyperactivity/impulsivity, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, ODD = oppositional defiant disorder.

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Irritability and Emotional Impulsivity as Core Feature of ADHD and ODD in Children

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Abstract

The categorical approach of diagnosing mental disorders entails the problem of frequently occurring comorbidities, suggesting a more parsimonious structure of psychopathology. In this study, we therefore aim to assess how affective dysregulation (AD) is associated with attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) in children. To assess AD in children aged 8–12 years ($n = 391$), we employed the parent version of a newly constructed parent rating scale. Following item reduction, we conducted exploratory and confirmatory factor analyses to establish a factorial structure of AD. One core dimension was identified, comprising irritability and emotional impulsivity, and two smaller dimensions, comprising positive emotionality and exuberance. Subsequently, we examined five different latent factor models – a unidimensional model, a first-order correlated factor model, a second-order correlated factor model, a traditional bifactor model, and a bifactor S-1 model, in which the first-order factor AD-Irritability/Emotional Impulsivity (II) was modeled as the general reference factor. A bifactor S-1 model with the *a priori* defined general reference domain AD-II provided the best fit to our data and was straightforward to interpret. This model showed excellent model fit and no anomalous factor loadings. This still held true, when comparing it to bifactor S-1 models with ADHD/ODD-related reference factors. Differential correlations with emotion regulation skills and the established Parent Proxy Anger Scale validate the interpretation of the different dimensions. Our results suggest that irritability/emotional impulsivity might be a common core feature of ADHD and ODD.

Keywords ADHD · ODD · Affective dysregulation · Irritability · Emotional impulsivity · Bifactor models

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The classification of psychiatric symptoms into categorical mental disorders, as is currently the case in the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association [APA], 2013) and the 11th editions of the International Classification of Diseases (ICD-11; World Health Organization [WHO], 2020), is useful for many reasons. It is useful for research, as the nosology makes it possible to integrate empirical findings, allows for communication and comparison of research findings, and guides further empirical studies. Ultimately, moreover, this research guides treatment. The categorical approach of classifying mental disorders brings the advantages of identifying risk and protective factors, of enabling prognoses, and of deciding which form of treatment has the best chance of success for a particular disorder (Caspi et al., 2014; Malhi & Bell, 2019).

However, one of the major challenges inherent in the categorical approach is the observation that comorbidities are the rule rather than the exception. Newman et al., (1998) found that comorbidities roughly conform to the rule of 50%, describing that half of individuals meeting diagnostic criteria for one disorder also meet criteria for a second disorder at the same time; and of these, 50% meet the criteria for a third mental disorder, and so on. Approaches attempting to explain this non-negligible number of comorbidities have focused either on the underlying etiological mechanisms in a bottom-up fashion (e.g. Research Domain Criteria [RDoC] by the National Institute of Mental Health; Cuthbert & Insel 2013; Insel et al., 2010) or on the observed pattern of covariation among psychopathological symptoms and traits in a top-down fashion (e.g. Hierarchical Taxonomy of Psychopathology [HiTOP]; Kotov et al., 2017). Though the RDoC and HiTOP differ in their approach to the reorganization of psychopathological symptom complexes, they pursue the same goal of moving away from diagnostic categories (Michelini et al., 2021). The neuroscientific RDoC approach aims to elucidate biobehavioral systems underlying a range of mental disorders (Cuthbert & Insel, 2013; Insel et al., 2010; Michelini et al., 2021). The approach is based on the assumption that mental disorders are, in fact, disorders of brain circuits, whose (dys-) functions can be assessed with neuroscientific tools, which will ultimately yield biosignatures that improve the understanding of the associations between symptom complexes (Insel et al., 2010). The extensively researched dimensional framework HiTOP (Kotov et al., 2017) aims to incorporate broader dimensions, potentially explaining comorbidity, as well as specific dimensions, accommodating heterogeneity *within* a disorder as well as symptom overlap *between* disorders (Kotov et al., 2017; Michelini et al., 2021). As both approaches come with some disadvantages (RDoC: large number of symptoms requiring clinical attention are missing (Michelini et al., 2021); HiTOP: underlying biological mechanisms are not considered), Michelini et al. (2020) proposed a promising RDoC-HiTOP interface, in which psychometrically robust clinical targets are suggested by HiTOP and can then be examined in terms of potential biological underpinnings.

While a more parsimonious, dimensional approach of classifying mental disorders comes with many advantages, it is not without criticism (Carragher et al., 2015; Ruggero et al., 2019; Zimmerman, 2021). As the ultimate goal of psychopathological classification systems is the selection and application of the ideal treatment, a restructuring of the classification system would have to improve precisely this process. The necessary relearning and retraining would require a significant amount of time and money (Zimmermann 2021, Carragher 2014) and it remains to be seen whether this process is worth the increased resources, as patients may show a considerable response to non-specific treatment aspects, independent of their diagnosis (Zimmermann, 2021). Related to this, due to time constraints, it might be difficult for acute settings to employ a fully dimensional approach (Ruggero et al., 2019). While an accurate classification system is undoubtedly of interest, and the relearning process would only be a temporary issue, the impact it would have on clinical outcomes will have to be evaluated.

In this article, we focus on affective dysregulation (AD) and how it might potentially explain the frequently observed comorbidities between attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). AD is a criterion for many diagnoses in children in the DSM-5 (APA, 2013) and the ICD-11 (WHO, 2020), and is therefore often characterized as a transdiagnostic dimension (Evans et al., 2017). It is generally understood as entailing an affective component (anger) and a behavioral component (aggression; Leibenluft & Stoddard 2013). Precise definitions differ, and range from rather narrow to very broad conceptualizations, leading to reported prevalence rates between 0.8% and 6.6% (Brotman et al., 2006; Copeland et al., 2013; Holtmann et al., 2008). Though the terms irritability, AD, and emotion dysregulation are sometimes used interchangeably when referring to the same or a highly similar construct (Evans et al., 2017; Leibenluft, 2011; Shaw et al., 2014), the mere definition of AD based on irritability is rather restricted, as emotion dysregulation and AD generally also include at least an impulsive component. In their influential review on irritability, Evans et al., (2017) summarized that irritability constitutes a diagnostic or at least an associated feature of a large number of psychological conditions, in particular but not limited to internalizing disorders such as depression and general anxiety disorder, and may be able to explain a good proportion of the frequently found comorbidities in children and adolescents. The authors also concluded that

particularly children with a combination of irritability with anger and temper outbursts, which corresponds to the definition of AD as entailing an affective as well as a behavioral component, show a pattern of correlates and outcomes differentiating them from other children with the same diagnosis. These findings led them to suggest that instead of an independent diagnosis for the combination of irritability and temper outbursts, a more parsimonious solution is needed (Evans et al., 2017). Compared to irritability, AD might therefore serve as an even more transdiagnostic concept, as irritability shows especially strong associations with disorders within the internalizing spectrum (Evans et al., 2017), whereas temper outbursts and impulsivity are, by definition, associated features of externalizing disorders. The addition of impulsivity might also be of particular interest given that in the majority of published associations, the p-factor is interpreted as a general tendency to react impulsively (Carver et al., 2017). AD-associated impulsivity differs from ADHD-associated impulsivity insofar as it contains an emotional component (e.g. *"often loses temper"*) that is not inherent in ADHD-associated impulsivity (e.g. *"talks a lot"*), which is so closely associated with hyperarousal that they are counted as *one* dimension in the DSM-5 (APA, 2013). The concept of emotional impulsivity has previously been suggested as an additional feature to the two established ADHD dimensions inattention and hyperactivity/impulsivity (Barkley & Fischer, 2010) and has been defined as "impatience, low frustration tolerance, hot-temperedness, quickness to anger, irritability and easily emotional excitability" (Barkley & Fischer, 2010, p.503), which closely corresponds to our definition of AD-associated impulsivity In this article, AD-associated emotional impulsivity and ADHD-associated impulsivity will therefore be treated as two separate constructs, in line with the general notion that impulsivity might be more of an "umbrella concept" (Berg et al., 2015, p.1129) referring to a large yet not definitively known number of dimensions (Berg et al., 2015).

AD symptomatology still poses a challenge for diagnostic classification systems (Evans et al., 2017). It is related to the DSM-5 (APA, 2013) diagnosis of disruptive mood dysregulation disorder (DMDD; Waltereit et al., 2019) as both concepts include chronic irritability as well as emotional impulsivity. As opposed to the broad, dimensional concept of AD symptomatology, DMDD is defined as a distinct categorical disorder. This has been strongly criticized (Evans et al., 2017; Lochman et al., 2015), as studies have failed to show a clear distinction from numerous other disorders, and mainly from ODD and ADHD (Evans et al., 2017). Based on recommendations by Lochman et al., (2015) and Evans et al., (2017), the ICD-11 (WHO, 2020) refrained from including AD symptomatology as a distinct categorical disorder and instead added a specifier of chronic irritability to the ODD diagnosis. The categorical conceptualization in the ICD-11 (WHO, 2020) and the DSM-5 (APA, 2013) is therefore countered by a dimensional view insofar as AD symptomatology is seen as a feature that is present in most individuals to varying degrees as well as a transdiagnostic feature in several psychopathological symptom complexes (Brotman et al., 2017). The same most likely holds true for ODD and ADHD symptomatology, as has been shown by a number of studies applying latent factor analyses in clinical and community samples to examine the underlying structure of these two disorders Burns et al., 2020; Junghänel et al., 2020; Thöne et al., 2021).

Previous studies assessing the comorbidities between ADHD and ODD have frequently applied bifactor or higher-order factor models to specify a common overarching factor Burns et al., 2020; Junghänel et al., 2020; Thöne et al., 2021; Waldman et al., 2021). Of the two ODD dimensions, particularly the dimension of defiant behavior has often being linked to ADHD (Evans et al., 2017; Stringaris & Goodman, 2009; Waldman et al., 2021). Emotion regulation, a concept that is inherent to all of the aforementioned symptom complexes, might explain the strong associations that are frequently found on this level (Ambrosini et al., 2013; Shaw et al., 2014). Similarly, in their trait-impulsivity theory, Beauchaine et al., (2010) suggested, that impulsivity combined with emotion dysregulation might be predecessors of the comorbid expression of ADHD and ODD.

Due to its particularly close associations with ADHD and ODD, AD may help to explain the frequently found comorbidities between these two disorders (Willcutt, 2012). To date, DMDD is the best researched conceptualization of chronic irritability and resembles the broad AD conceptualization to some extent, as it includes irritability as well as emotional impulsivity. For DMDD, strong associations with ODD and ADHD have been reported (Copeland et al., 2013; Evans et al., 2017; Leibenluft, 2011; Mayes et al., 2016; Mulraney et al., 2016). ADHD has been associated with the two main components of AD symptomatology separately as well. Barkley & Fischer (2010) identified emotional impulsivity as an important add-on concept to the ADHD diagnosis. In addition, (Karalunas et al., 2019) found ADHD symptomatology to be associated with irritability and identified a subgroup of children with ADHD and irritability that could not be reduced to the combination of ADHD and ODD. This finding led the authors to suggested a specifier of irritability to the ADHD diagnosis. Similarly, Eyre et al., (2017) showed that almost all children with an ADHD diagnosis displayed at least one symptom of irritability. Regarding AD and ODD symptomatology, the chronicity of irritability and the severity of temper tantrums supposedly differentiate AD from ODD. The close proximity between DMDD and ODD can also be seen in the DSM-5, where a

diagnostic hierarchy does not allow for an additional ODD diagnosis once the criteria for DMDD are fulfilled. The reason for the frequent diagnostic and clinical overlap between ODD and DMDD might lie in the current conceptualization of ODD. A number of independent research groups have demonstrated that ODD consists of at least two different dimensions – irritability and defiant/argumentative behavior, which lead to differential outcomes (Evans et al., 2017). The current conceptualization of ODD appears to confound these two frequently co-occurring but distinct dimensions (Runions et al., 2016). This combination of dimensions into a categorical diagnosis is a hallmark of the current classification systems and is contrasted by approaches attempting to explain comorbidities between diagnoses by reorganizing symptoms constituting DSM-5/ICD-11 diagnosis into dimensions and to model their associations with one another (e.g. HiTOP; Kotov et al., 2017). Further research is necessary to examine the exact composition of AD symptomatology and its association with dimensions of ODD and ADHD. As we were interested in the broad spectrum of AD and not in a specific diagnosis or conceptualization, for the purpose of the present study it seemed most appropriate to employ a broad definition of AD symptomatology, assessing all potentially associated features.

One method to address the research question of how (dimensions of) mental disorders are related to one another is latent factor analysis. Latent factor analysis can highlight core dimensions accounting for observed symptomatology and comorbidity between disorders (Eaton, 2015) and has been applied with increasing frequency within psychological research in recent years. Applying latent factor analysis also enables us to move away from diagnostic-level to symptom-level analyses, contributing to the development of potentially more valid and parsimonious nosologies (Eaton, 2015). In particular, higher-order factor models and bifactor models, which decompose true score variance and assign it to a general (g-) or a specific (s-) factor have provided useful insights into the latent factor structure of psychopathology (Eid et al., 2017). The variety of latent factor models come with advantages and disadvantages, provide differential information, and can therefore answer different kinds of research questions. In the following, we will briefly outline the kind of models, also employed in this study, that are frequently used to answer research questions related to the overall theme of examining associations between dimensions of mental disorders. First-order correlated factor models (CFO) are a good basis for higher-order models and yield initial insights into how the dimensions or disorders are related to one another (Eid, 2020). In our case, assessing AD, ODD, and ADHD dimensions, such models would allow us to examine to what degree these dimensions are correlated. If high correlations between dimensions are found, the CFO ends up being an incomplete model, as correlations between dimensions are left unmodeled. These correlations can then either be accounted for in a unidimensional model (i.e., assuming that an externalizing spectrum, which has been found frequently, though with different subfacets (Kotov et al., 2017; Krueger et al., 2005) captures the corresponding symptomatology better than the distinct diagnoses ADHD, ODD and AD) or a model that assumes some sort of overarching factor. The models with an overarching factor are either higher-order factor models, such as the HiTOP (Kotov et al., 2017), or bifactor models, which model an additional first-order factor instead of a higher-order factor, which is associated with the *items* from all dimensions. The idea behind bifactor models is that *"only with the general factor variance removed can we have a clear window into the remaining covariance patterns among the symptoms in our measure. Only with specific measures unconfounded by the general factor can we have a clear window into the etiological or prognostic associations"* (Hartman, 2021, p.72). However, despite the popularity of these models, it often remains unclear what this so-called g-factor really stands for (Heinrich et al., 2020). Additional problems include the proportionality constraint in the higher-order factors model, which describes the problem that, by definition, all items from one dimension show the same association ratio with the lower- and higher-order factors (Brunner et al., 2012; Gignac et al., 2016), whereas in bifactor models, associations between the s-factors as well as between the g- and the s-factors cannot be assessed as they are constrained to zero. In addition, bifactor models often yield weakly defined s-factors and interpretation difficulties of the factors and their relation to one another arise due to anomalous factor loadings, such as negative or non-significant factor loadings or variances (Burns et al., 2020; Eid et al., 2017). A relatively new version of a bifactor model – the bifactor S-1 model – offers a solution to the aforementioned problems. By modeling one of the s-factors as a general reference factor (i.e. there is no s-factor modeled for items of the reference factor), there are now "pure indicators of the general factor" (Markon, 2021, p.67). The choice of this reference factor should be theoretically derived or correspond to a domain of greater interest (Eid, 2020). The remaining s-factors are orthogonal to this general reference factor and are allowed to correlate with each other (Eid et al., 2017). The bifactor S-1 model therefore allows for a straightforward interpretation of the g- and s-factors as well as their relations to one another. The initial goal of bifactor models to identify a general overarching psychopathology factor has to be dismissed when applying this version of the bifactor model. Nevertheless, it appears that traditional bifactor models cannot reach this goal either, and frequently lead to

a misinterpretation of the g-factor as a general psychopathology factor (Heinrich et al., 2020).

Aim of Study

The current study aimed to establish a factorial structure of AD and to examine how AD relates to the defiant dimension of ODD and ADHD. As ODD has been shown to consist of a defiant and an irritable dimension and irritability is a main component of AD, we only assessed associations of AD with the defiant dimension of ODD (ODD-D). For ADHD, associations with inattention (ADHD-IN) and hyperactivity/impulsivity (ADHD-HI) were examined. In a first step we assessed item descriptive statistics and item redundancy in order to shorten the AD questionnaire, which in its original form was a combination of items from several existing questionnaires assessing AD. This was followed by exploratory (EFA) and confirmatory factor analyses (CFA) to establish the factorial structure of AD. In a next step, we examined five different latent factor models (Fig. 1): (a) a unidimensional model, corresponding to the idea of a general externalizing spectrum, in which all items from all dimensions loaded onto one common overarching factor (Uni), (b) a correlated factor model with the aforementioned correlated first-order factors (CFO) to assess the associations between dimensions, (c) a second-order factor model, in which these first-order factors loaded onto one second-order factor (SOF), (d) a bifactor model, in which the items from all dimensions loaded onto one general factor as well as onto *one* specific factor (BI), and (e) a bifactor S-1 model, in which we suggest the core AD-factor, which we presumed to be describing irritability/emotional impulsivity, as a candidate for the general reference factor (BI S-1). Importantly, at this step, any dimension of interest could have been chosen as the general reference factor. We chose the core AD dimension, as previous studies have frequently found measures of emotional impulsivity, a concept closely related to AD symptomatology, to be strongly associated with the p-factor and to predict the onset and progression of symptoms (Carver et al., 2017). In line with this, Beauchaine et al., (2010) suggested emotion dysregulation/impulsivity as a predecessor of the combined presentation of ADHD and ODD, contributing to the hypothesis of AD core symptomatology serving as an ideal candidate for the general reference factor of AD, ODD and ADHD. Subsequently, we examined the associations of the different dimension with emotion regulation skills, assessed using the German FRUST questionnaire as well as the Parent Proxy Anger Scale, which is part of the Patient-Reported Outcomes Measurement Information System (PROMIS; Irwin et al., 2012). In a final step, in order to validate our assumption of AD core symptomatology serving as an ideal reference factor, we tested competing hypotheses, i.e. models with ODD-D, ADHD-IN, or ADHD-HI as alternative general reference factors. Since AD symptomatology is currently conceptualized as a specifier to the ODD diagnosis in the ICD-11 (WHO, 2020), it seemed important to assess ODD-D as a general factor as well. As for ADHD, the finding of Karalunas et al., (2019) regarding a specifier of irritability to the ADHD diagnosis and the results of Barkley & Fischer (2010) of emotional impulsivity being an important add-on concept to the ADHD diagnoses, led us to the decision to also test the hypotheses of both ADHD dimensions serving as the general reference factor.

Hypotheses

1) Item reduction: As items were taken from several existing questionnaires assessing broad AD symptomatology in children, we expected the results of item descriptive statistics and item redundancy to allow for a shortening of the newly developed 38-item AD questionnaire before further validating it.
2) Factorial structure of AD: In line with the conceptualization of AD symptomatology as a specifier of irritability/anger suggested by Evans et al., (2017) and as DMDD (DSM-5, APA, 2013), we expected to identify an AD-core factor, defined by items describing irritability and emotional impulsivity. As items were taken

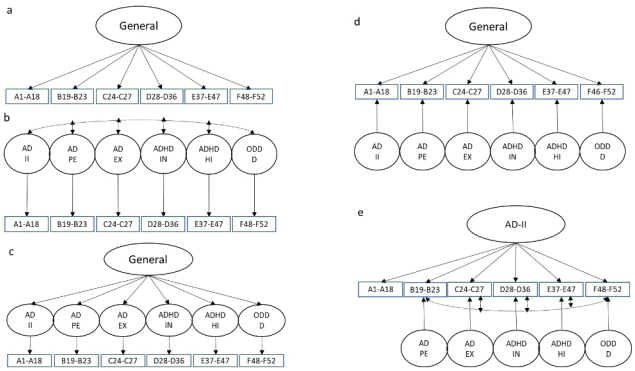

Fig. 1 Latent Factor Models of AD, ADHD and ODD Dimensions. (**Fig. 1a** Unidimensional model (Uni), **b** Factor model with six correlated first-order factors (CFO), **c** Factor model with six correlated first-order factors and one second-order factor (SOF), **d** Bifactor model (BI) **e** Bifactor S-1 model (BI S-1)
Note. In 1b and 1e, all first-order factors are allowed to correlate (indicated by dotted arrows). Item numbers are displayed in the boxes and residuals are not shown for clarity of presentation. AD = affective dysregulation, II = irritability/emotional impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension)

Table 1 Sample and Descriptive Statistics

Sample Statistics		
Total sample		*n* = 391
Age: mean (*SD*)		10.64 (1.33)
Male: *n* (%)		220 (56)
Group [*n* (%)]		
AD		244 (62)
NoAD		147 (38)
Diagnoses [*n* (%)]		
DMDD		41 (11)
ODD		93 (24)
ADHD		62 (16)
ADHD, combined type		19 (5)
ADHD, predominantly inattentive type		30 (8)
ADHD, predominantly hyperactive-impulsive type		13 (3)
CD		6 (2)
MD		5 (1)
Comorbid diagnoses [*n* (%)]		
AD + ODD		93 (24)
AD + ADHD		61 (16)
AD + ADHD, combined type		19 (5)
AD + ADHD, predominantly inattentive type		29 (7)
AD + ADHD, predominantly hyperactive-impulsive type		13 (3)
AD + CD		6 (2)
AD + MD		5 (1)
Dimensional Statistics		
Dimension (*n* = 386–390)	M (*SD*)	α
AD-irritability/emotional impulsivity	1.13 (0.55)	0.96
AD-positive emotionality	2.23 (0.49)	0.72
AD-exuberance	0.89 (046)	0.81
ADHD-inattention	0.93 (0.79)	0.94
ADHD-hyperactivity/impulsivity	0.56 (0.63)	0.92
ODD-defiant dimension	0.68 (0.64)	0.84
FRUST-adaptive emotion regulation strategies	2.00 (0.72)	0.89
FRUST-maladaptive emotion regulation strategies	1.61 (1.00)	0.78
PROMIS	1.40 (0.93)	0.91

Note. AD = affective dysregulation, ADHD = attention-deficit/hyperactivity disorder, DMDD = disruptive mood dysregulation disorder, ODD = oppositional defiant disorder, CD = conduct disorder, MD = major depressive episode, M = mean, *SD* = standard deviation, α = Cronbach's Alpha, FRUST = "Questionnaire on the regulation of unpleasant moods in children" (Fragebogen zur Regulation unangenehmer Stimmungen von Kindern), PROMIS = Patient-Reported Outcomes Measurement Information System - Parent Proxy Anger Scale

from several questionnaires, assessing a broad range of AD symptomatology in children, we expected to find smaller AD-factors in addition to that core factor.

3) Latent factor analysis of AD, ADHD and ODD symptomatology: We expect the extension of our theoretically derived bifactor S-1 model to AD symptomatology with AD-core symptoms as 'pure indicators' of the reference factor to fit the data best in terms of global model fit indices, the significance of factor loadings and general interpretability. This is consistent with previous studies showing the clear interpretability of the bifactor S-1 model when assessing the structure of ADHD and ODD (Burns et al., 2020; Junghänel et al., 2020; Thöne et al., 2021). On top of that, this approach adds to previous findings reporting high comorbidities between AD, ADHD and ODD symptomatology (Evans et al., 2017; Eyre et al., 2017; Mulraney et al., 2016) and a strong association of irritability with ADHD and ODD (Ambrosini et al., 2013; Shaw et al., 2014).
4) Divergent and convergent validity: We expected to find differential correlations of all AD, ADHD and ODD dimensions with emotion regulation strategies as well as the PROMIS Parent Proxy Anger Scale, thus supporting the external validation of our model.
5) Comparison of reference factors: Based on studies suggesting measures of emotional impulsivity, which are strongly related to AD core symptomatology, as an ideal candidate for our general reference factor (Beauchaine et al., 2010; Carver et al., 2017), we expected the bifactor S-1 model with the reference factor AD-II to provide a better fit to the data in terms of explained common variance of the reference factor, global model fit, and the pattern of factor loadings, compared to models with the reference factors ODD-D, ADHD-IN or ADHD-HI.

Methods

Participants and Procedure

Data collection took place within the ongoing ADOPT (Affective Dysregulation in Childhood—Optimizing Prevention and Treatment; Döpfner et al., 2019) study. The *ADOPT* project is a multicenter research project encompassing Seven study centers located in Germany and the six subprojects coordination, epidemiology, neurobiology, online, treatment and institution. It entails several measurement time points (Döpfner et al., 2019). The aim of this multicenter study was to optimize clinical diagnostics, prevention and treatment of AD and investigates an evidence-based, individualized treatment program based on behavioral interventions for children with AD. For the

present study, we analyzed baseline data (T1) of a sample screened in the community, collected between August 2018 and September 2019, which included 391 children aged 8–12 years ($M = 10.6$, $SD = 1.3$; 56.3% males). An initial screening, obtained through a parent screening questionnaire, categorized participants into an AD and a NoAD group. Children with AD symptoms in the top 10% of the sample were allocated to the AD group, children with AD symptoms in the bottom 10% of the sample were allocated to the NoAD group (for further details regarding the screening procedure see Otto et al., 2022). All families in the AD group were then offered further participation in the ADOPT study. A random sample was drawn from the NoAD group. Clinical child and parent interviews (Görtz-Dorten, Döpfner & Thöne, 2022) were conducted with participating families. AD/NoAD group assignment was confirmed through the clinical parent interview. Main inclusion criteria include the age of the child (8;0–12;11 at T1), the residence of the child (child lives with at least one natural or adoptive parent), and clinician-rated AD symptomatology of the child (based on the clinical parent interview) as well as the families' willingness and ability to participate in the study. As can be seen in Tables 1 and 244 (62.4%) of study participants were categorized into the AD group. Regarding additional diagnoses, 41 study participants (10.5%) fulfilled the DSM-5 criteria for DMDD, 93 (23.8%) for ODD, 62 (15.9%) for ADHD, six (1.5%) for CD and five (1.3%) for a major depressive episode (MD). In almost 100% of ODD, ADHD, MD and CD comorbid AD was present (Table 1). All diagnoses were based on clinical interviews (Görtz-Dorten, Döpfner & Thöne, 2022), which are part of the Diagnostic System of Mental Disorders in Children and Adolescents based on the ICD-10 and DSM-5 [*Diagnostik-System für psychische Störungen nach ICD-10 und DSM-5 für Kinder und Jugendliche – III*] (DISYPS–III; Döpfner & Görtz-Dorten (2017).

Measures

FBB-ADHS/FBB-SSV

The mothers or fathers of the 391 participants completed the German Symptom Checklist for Attention-Deficit/Hyperactivity Disorder (*Fremdbeurteilungsbogen für Aufmerksamkeitsdefizit-/Hyperaktivitätsstörungen;* FBB-ADHS) as well as the Symptom Checklist for Disruptive Behavior Disorders (*Fremdbeurteilungsbogen für Störungen des Sozialverhaltens*; FBB-SSV) from the DISYPS-III (Döpfner & Görtz-Dorten, 2017). Both rating scales are based on the symptom criteria of the DSM-5 and ICD-10. The FBB-ADHS assesses IN symptoms with nine items and HI symptoms with eleven items. All items are rated on a 4-point Likert scale (0 [not at all) – 3 [very much]). The FBB-SSV assesses ODD, CD and callous-unemotional symptoms. Following the questionnaire's instruction to assess CD and CU symptoms only for children aged eleven or older, we only used the items assessing ODD for the present study. To avoid symptom overlap, and as we were interested in how dimensions of psychopathology (similar to HiTOP; Kotov et al., 2017) might be able to explain comorbidities between current diagnostic categories such as ADHD and ODD, we only considered five of the items, which assess the defiant dimension of ODD (ODD-D), as the other three ODD symptoms of the FBB-SSV assessing the irritability dimension were already included in the assessment of AD. Both questionnaires have shown good psychometric properties in terms of reliability and validity of the scale scores (Döpfner et al., 2008; Erhart et al., 2008; Görtz-Dorten et al., 2014).

DADYS parent rating

The parent version of the DADYS [**D**iagnostikum für **A**ffektive **DYS**regulation (Diagnostic System for Affective Dysregulation)] is used to assess AD and comprises 38 items from several existing questionnaires assessing irritability/anger and affective dysregulation/emotional regulation in general. Items from the DADYS included in the present analysis were taken from the Emotion Regulation Checklist (Shields & Cicchetti, 1997), the FBB-SSV (Döpfner & Görtz-Dorten, 2017) and the Affective Reactivity Index (Stringaris et al., 2012). All items are rated on a 4-point Likert scale (0 [not at all) – 3 [very much]).

PROMIS parent Proxy anger scale

The Parent Proxy Anger Scale is part of the Patient-Reported Outcomes Measurement Information System (PROMIS; Irwin et al., 2012) and entails five items assessing rage and anger in children. Items are rated by the parent on a 5-point Likert scale, ranging from 0 (never) to 4 (almost always). The scale score of the Anger Scale has shown good psychometric properties (Varni et al., 2012).

FRUST

The "Questionnaire on the regulation of unpleasant moods in children" (Fragebogen zur Regulation unangenehmer Stimmungen von Kindern; FRUST) by (Görtz-Dorten et al., 2019, unpublished manuscript) is adapted from the FEEL-KJ questionnaire (Grob & Smolenski, 2005), which is frequently applied to assess emotion regulation skills in children and adolescents. As opposed to the FEEL-KJ, which assesses the regulation skills for fear, anger and

sadness separately with 30 item per emotion ("If I am scared, I…"/"If I am angry, I…"/"If I am sad, I…") the FRUST assesses the regulation of unpleasant emotions in general with 30 items all together ("If I feel bad, I…") We additionally designed and validated a parent version of the FRUST, which was used in this study. This version contained only 14 items, as we excluded items assessing primarily internal processes that are difficult to observe from the outside. The FRUST showed good psychometric qualities (Junghänel et al., in preparation). Items are rated on a 5-point Likert-scale ranging from 0 (almost never) to 4 (almost always). A two-factorial structure of the FRUST has been established, consisting of one adaptive emotion regulation factor (FRUST-A; 10 items) and one maladaptive emotion regulation factor (FRUST-M; 4 items) (Junghänel et al., in preparation). The internal consistency was good, with Cronbach's $\alpha = 0.89$ for FRUST-A and $\alpha = 0.78$ for FRUST-M (Junghänel et al., in preparation).

Statistical analyses

For descriptive analyses and calculations of internal consistency, we used SPSS version 26. All other analyses were conducted using Mplus version 8.4 (Muthén & Muthén, 2017). We used the weighted least square mean and variance adjusted (WLSMV) estimator (delta parameterization), which is suggested for modeling ordinal data and does not assume normally distributed variables (Li, 2016). Missing data were handled with the Mplus default strategy *pairwise deletion.* Due to increasing computational demands in analyses with five or more factors, the full information maximum likelihood (FIML) estimation has been found to be impracticable (Forero & Maydeu-Olivares, 2009). In addition, global model fit cannot be assessed in most cases with Mplus when fitting ordinal factor analysis models with FIML (Shi et al., 2020). In the case of a low number of missing values, high reliability of the scales, as well as the assumption that missing values are missing completely at random, which all held true in our study, pairwise deletion has been found to work well (Shi et al., 2020; Tsikriktsis, 2005). The amount of missing data per item was below 1% for all items. Covariance coverage was above 0.995 for all items.

For the SOF model, we applied the Schmid-Leiman transformation, which is a mathematical transformation of the standardized factor loadings that can be used to estimate the direct impact of the first-order and the higher-order factors on manifest item scores in higher-order factor models (Brunner et al., 2012; Gignac, 2016). For the impact of the higher-order factor, the standardized factor loading of each item was multiplied by the factor loading of the corresponding first-order factor on the second-order factor. To estimate the impact of the specific first-order factor, the factor loading of each item was multiplied by the standard deviation of the corresponding factor (for a detailed explanation see Brunner et al., 2012).

To evaluate model fit, we predominantly relied on the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA) and the standardized root mean square residuals (SRMR). For model fit to be considered good, the CFI and TLI should be ≥ 0.95 (Hu & Bentler, 1999) and RMSEA and SRMR should be ≤ 0.05 (Browne & Cudeck, 1992; Hooper et al., 2008). For adequate model fit, RMSEA and SRMR should be ≤ 0.08 (Browne & Cudeck, 1992; Hooper et al., 2008). Differences of > 0.010 in CFI would indicate a significant difference between global model fit (Cheung & Rensvold, 2002).

There is a vast array of indices that can be used for the evaluation of dimensionality in bifactor models on the item level, the factor level, and the model level (for a detailed summary and explanation see Rodriguez et al., 2016). Frequently reported are the omega statistics. Omega (ω) describes the amount of reliable variance accounted for by the g-factor and all s-factors taken together, whereas omega hierarchical (ωH) describes the amount of reliable variance accounted for by the g-factor (Brunner et al., 2012; Reise, 2012). Omega hierarchical subscale (ωHS) is the equivalent to ωH for the individual s-factors. It has been recommended that ωH/ωHS should not be lower than 0.50 or ideally 0.75 in order to be interpreted reliably (Reise et al., 2013). Another important index supporting the correct evaluation of dimensionality is the explained common variance (ECV), which indicates the proportion of all common variance explained by that factor. Bifactor-relevant indices were calculated with the help of the Bifactor Indices Calculator by Dueber (2017).

Analytic Plan

Item reduction of the DADYS Questionnaire

For item reduction several criteria were considered: Skewness, kurtosis, usage of all response categories, distribution of responses (how often was an item answered with 0 or 1), item-item-correlations $r < .30$, item-item-correlations $r > .80$, redundancy of item content assessed by three clinical raters, and correspondence with DSM-5 criteria for DMDD. For the clinical assessment of item redundancy each of the three clinical raters created item pools, consisting of items that they regarded to assess the same content. Items were excluded for three reasons: 1) *Exclusion due to saliences in*

descriptive statistics. Items were excluded if they showed at least four of the following saliencies: (a) Skewness/kurtosis larger than one/two standard deviation(s) (counted as one and two saliences, respectively), (b) not all response categories were used, (c) salient distribution of responses (> 90% answered this item with 0 or 1), (d) the item correlated with more than 50% (= 17 items) of the other items with $r < .30$. 2) *Exclusion due to item redundancy*. Items were excluded if they correlated with at least one other item with $r > .80$ and the content was additionally rated as redundant by at least two of the three clinical raters. The selection regarding which of the redundant items was kept in the questionnaire was based on two aspects: If one of the redundant items was a DSM-5 criterion for DMDD, this item was selected to remain in the questionnaire. If none of the items rated as redundant were DSM-5 criteria for DMDD, the item with the highest number of item-item correlations $r > .80$ was kept in the questionnaire. 3) *Exclusion due to low associations with the other items*. Items were excluded if they correlated with more than 90% (= 34 items) of the other items with $r < .30$. If the to-be-excluded item was a DSM-5 criterion for DMDD, it was kept in the questionnaire. As our assessment of AD symptomatology is similar to, yet broader than DMDD, we aimed for our AD construct to fully include DMDD and therefore decided to keep the previously evaluated DSM-5 criteria for DMDD in our questionnaire whenever possible.

Exploratory and confirmatory factor analysis of the DADYS Questionnaire

For EFA and CFA, items were treated as ordinal and the WLSMV estimator was used. In a first step, the sample was divided randomly in two groups, each containing $n = 195$ children. We then applied EFA in one half of the sample with a principal axes factor analysis, as we did not expect for all variance to be explained by the extracted factors. We chose the oblique GEOMIN rotation, which allows for correlations between factors. EFA is advantageous in situations where no clear established structure for a construct exists, as cross-loadings are freely estimated, which can provide novel insight into the data structure. We then applied CFA in the other half of the sample in order to examine if the previously extracted factorial structure of AD can be confirmed. We additionally let Mplus calculate modification indices (MI) to suggest potential changes to our model, which were carefully checked and assessed with regard to content-based meaningfulness in every case.

Confirmatory factor analysis of AD, ADHD and ODD dimensions

In a next step, items from all AD-dimensions, ADHD-IN, ADHD-HI and ODD-D were tested in a unidimensional model (Uni), a first-order correlated factor model (CFO), a second-order correlated factor model (SOF), a traditional bifactor model (BI) and a bifactor S-1 model (BI S-1) to examine how AD symptomatology was associated with ADHD and ODD dimensions (Fig. 1).

Associations with External correlates

To externally validate our factorial structure, correlations of the dimensions of our optimally fitting model with the PROMIS Parents Proxy Anger Scale as well as with the FRUST-A and the FRUST-M were calculated.

Comparison of reference factors

In addition to the previously examined BI S-1 model, which had AD-II as reference factor, we additionally tested three bifactor S-1 models with ODD-D (BI S-1; ODD-D), ADHD-IN (BI S-1; ADHD-IN) and ADHD-HI (BI S-1; ADHD-HI) as reference factors.

Results

Descriptive information

Descriptive information can be found in Table 1. Internal consistencies for all scales, including the reduced ODD-D scale, were good ($\alpha \geq 0.72$).

Item reduction

The exclusion process with all its criteria can be found in Table S1. Seven items (10, 12, 16, 18, 19, 24 and 30) fulfilled the criteria for previously described salient descriptive statistics. We excluded all of these items with the exception of item 30 ("*prolonged physically aggressive tantrums*") as this item was included in the DSM-5 as a criterion for DMDD and therefore of interest to keep in the questionnaire. Four items (8, 26, 36, 37) were excluded due to item redundancy. Of the items that correlated highly with one another we kept item 29 as this was included in the DSM-5 as a criterion for DMDD. One item (23) was excluded as it showed low item-item correlations of $r < .30$ with at least 90% of the other items. Altogether, we excluded eleven items and reduced the DADYS questionnaire from 38 to 27

items selected fur further validation, thus confirming our first hypothesis.

Factorial structure of AD

The three factor-solution of the EFA in one half of the sample was the first that yielded good model-fit (CFI = 0.988, TLI = 0.985, RMSEA = 0.056 (90% CI: 0.046-0.066), SRMR = 0.052) and clear interpretability of the factors. Factor 1 constitutes the AD-core factor and comprises 18 items describing irritability and emotional impulsivity (AD-II). Factor 2 is composed of five items, describing positive emotionality (AD-PE), whereas the four items of factor 3 characterize exuberance (AD-EX), which in distinction to impulsive behavior is free of value and also comprises positive outbursts of emotions. Factor loadings can be found in Table S2. We found significant correlations ($p < .05$) between AD-II and AD-PE ($r = -.50$) and AD-II and AD-EX ($r = .38$).

We then assessed the three-factor structure applying CFA in the other half of the sample. Items 4, 5, 9 and 11 were recoded to load positively on their respective factor. Model fit indices were in an adequate to good range, expect for the RMSEA (CFI = 0.964, TLI = 0.961, RMSEA 0.090 (90% CI: 0.083-0.098), SRMR = 0.077) and all items loaded significantly ($p < .001$) on their corresponding factor (Table S3). A careful inspection of the MI's revealed that the two largest MI's suggested allowing for the additional residual correlations between items 3 ("*responds positively to adults*") and 7 ("*responds positively to peers*") [MI: 145.48], as well as between items 5 (*"Calms down after being angry"*) and 34 ("*Stays angry"*) [MI: 66.74], due to their very close proximity in content. After this adaptation, model fit indices were all in an adequate to good range (CFI = 0.978, TLI = 0.976, RMSEA = 0.071 (90% CI: 0.062-0.079), SRMR = 0.066). Items 3 and 7 as well as items 5 and 34 correlated significantly ($p < 0001$), with $r = .70$, and $r = .67$, respectively. The internal consistency for all dimensions was good to excellent, with Cronbach's α ranging from 0.72 for AD-PE to 0.96 for AD-II. This stable three-factor solution with an AD-core factor (AD-II), as well as two additional smaller AD-factors (AD-PE; AD-EX) confirms our second hypothesis.

Table 2 Comparison of Model Fit Indices

Model	χ^2 *(df)*	CFI	TLI	RMSEA (90% CI)	SRMR
Uni	4670.937* (1272)	0.918	0.914	0.083 (0.080, 0.085)	0.095
CFO	2451.262* (1257)	0.971	0.970	0.049 (0.046, 0.052)	0.060
SOF	2728.702* (1266)	0.965	0.963	0.054 (0.052, 0.057)	0.069
BI	2436.548* (1220)	0.971	0.968	0.051 (0.048, 0.053)	0.061
BI S-1 (Ref. = AD-II)	2294.591* (1228)	0.974	0.972	0.047 (0.044, 0.050)	0.055
BI S-1 (Ref. = ODD-D)	2103.000* (1215)	0.979	0.977	0.043 (0.040, 0.046)	0.049
BI S-1 (Ref. = ADHD-IN)	2274.056* (1219)	0.975	0.972	0.047 (0.044, 0.050)	0.052
BI S-1 (Ref. = ADHD-HI)	2148.761* (1221)	0.978	0.976	0.044 (0.041, 0.047)	0.050

Note. χ^2 = Chi-Square, *df* = degrees of freedom, CFI = comparative fit index, TLI = Tucker-Lewis index, RMSEA = root mean square error of approximation, CI = confidence interval, SRMR = standardized root mean square residuals, Uni = unidimensional model, CFO = factor model with six correlated first-order factors, SOF = factor model with six correlated first-order factors and one second-order factor, BI = bifactor model, BI S-1 = bifactor S-1 model (see Fig. 1), Ref. = reference factor. AD-II = affective dysregulation – irritability/emotional impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity

* = $p < .001$

Latent factor analysis including ADHD and ODD

In terms of global model fit, the third hypothesis was not supported. As indicated by the model fit indices alone, the global model fit of the CFO, the SOF, the BI and the BI S-1 did not differ significantly from one another. Only the unidimensional model yielded inadequate fit and will therefore not be discussed further (Table 2). For the CFO model (Table S4), all factor loadings on their respective factor were high and significant ($p < .001$). All six factors correlated strongly ($r = -.48$ to $r = .85$) and significantly ($p < .001$) with each other (Table 3). For the SOF model (Table S4), all factor loadings on the first-order factors were high and significant ($p < .001$). As shown in Table S5, all first-order factors loaded significantly ($p < .001$) on the second-order factor (-0.70 for AD-PE to 0.90 for AD-II). All first-order factors showed significant ($p < .001$) residual variance (0.18 for AD-EX to 0.51 for AD-PE). The Schmid-Leiman transformation (Table S6) showed that in total, item loadings were stronger on the second-order factor than on the first-order factors (*Mean[M]* = 0.61 for the second-order vs. $M = .30$ for the s-factors). An examination of the g/s loading ratios revealed that this pattern was not equally pronounced for all dimensions: The g/s loading ratio was highest for the dimension AD-EX (2.16), followed by AD-II (2.01) and ODD-D (1.99). The ratio was closer to 1 for the dimension

Table 3 Correlations Between the First-Order Factors

Variable	1	2	3	4	5	6
1. AD-II	–					
2. AD-PE	−0.71*	–				
3. AD-EX	0.84*	−0.56*	–			
4. ADHD-IN	0.70*	−0.54*	0.69*	–		
5. ADHD-HI	0.73*	−0.48*	0.82*	0.79*	–	
6. ODD-D	0.85*	−0.64*	0.74*	0.67*	0.71*	–

Note. AD = affective dysregulation, II = irritability/emotional impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension

* = $p < .001$

AD-HI (1.66), ADHD-IN (1.40) and AD-PE (-0.98). The BI model yielded some anomalies, such as non-significant factor loadings and negative residual items variances (Table S7). The bifactor-specific indices ωH/ ωHS and ECV indicated a strong g-factor and weakly defined s-factors. ωHS ranged between 0.19 for ODD-D to 0.44 for AD-PE, thus remaining below the recommended cut-off of 0.50 by Reise et al., (2013). The ECV for all s-factors combined lay at 29% and ranged from 2% for ODD-D to 8% for AD-II. The BI S-1 model showed significant factor loadings ($p < .001$) and no anomalies (Table S8). Similar to the BI model, ωH/ ωHS and ECV suggested a strong g-factor as well as weak s-factors (Table 4). Compared to the BI model, ωHS values were higher, albeit still below the recommended cut-off and ranged between 0.26 for ODD-D and 0.47 for ADHD-IN. The ECV for all s-factors combined lay at 30%, with values ranging from 3% for AD-PE, AD-EX and ODD-D to 0.11% for ADHD-HI. As shown in Table S9, some residual correlations between the first-order factors remained significant ($p < .05$), the two highest being the correlation between the two ADHD dimensions ($r = .58$) and the correlation between ADHD-HI and AD-EX ($r = .56$). The good model fit of the BI S-1 model, the significant factor loadings of items from all dimensions on the reference factor AD-II, the absence of anomalous factor loadings, the remaining significant residual correlations as well as the *a priori* defined general reference factor and the resulting straightforward interpretation of g- and s-factors and their relation to one another, support our third hypothesis that the BI S-1 model captures the data best.

Convergent and divergent validity

To assess convergent and divergent validity, we included the PROMIS Parent Proxy Anger Scale, FRUST-A and FRUST-M in our BI S-1 model and computed (residual) correlations. (Table S10). The PROMIS Parent Proxy Anger Scale correlated significantly ($p < .05$) with all other residual factors, except for ODD-D. The highest correlation was found with the reference factor AD-II ($r = .90$), all other correlations were small and ranged from $r = .08$ to $r = .14$. FRUST-A correlated significantly negative ($p < .05$) with the reference factor AD-II ($r = -.71$) as well as the residual factors AD-PE ($r = -.46$) and the PROMIS Scale ($r = -.59$). FRUST-M correlated significantly ($p < .05$) with all residual factors, as well as the PROMIS scale and FRUST-A. The highest correlations were found with AD-II ($r = .82$), PROMIS ($r = .74$) and FRUST-A ($r = -.77$). All other correlations were small and ranged from $r = -.08$ to $r = .18$. The observed differential correlations of the PROMIS scale and emotion regulation strategies with all AD, ADHD and ODD dimensions confirm our fourth hypothesis.

Comparison of reference factors

Out of the four BI S-1 models, the model with ODD-D as reference factor showed the highest ECV of the reference factor (72%), followed by the models with AD-II (70%), ADHD-HI (64%) and ADHD-IN (60%) as reference factors. The models did not show significant differences in model fit. The BI S-1 model with ODD-D as reference factor showed non-significant and negative factor loadings on the s-factor AD-II. Taken together, our fifth hypothesis can be confirmed, as the model explaining the most common variance (BI S-1; ODD-D) showed a number of anomalous factor loadings, with that limiting straight-forward interpretability of all factors.

Discussion

In the present study, we performed item reduction of the DADYS questionnaire, investigated the factorial structure of AD in a sample screened in the community, including children with and without AD, assessed associations between AD, ADHD and ODD dimensions, examined convergent and divergent validity of our dimensions and compared bifactor S-1 models with different reference factors.

To establish a factorial structure of our broadly defined AD concept, we first excluded eleven items from the questionnaire due to salient item descriptive statistics or item redundancy. Item redundancy was to be expected as the

questionnaire was created by combining items from several questionnaires assessing AD. Some other items (e.g. "*takes pleasure in distress of others*") that were excluded due to salient descriptive statistics may have been too negatively connotated for our sample consisting of relatively young children (8–12 years), about half of whom did not meet criteria for AD.

Through EFA and CFA, including the process of cross-validation, we established a stable factorial structure of AD, which comprised one core-factor, describing irritability/emotional impulsivity, as well as two smaller AD factors, representing positive emotionality and exuberance. This core factor of irritability and emotional impulsivity is in line with the chronic irritability/anger specifier suggested by Evans et al., (2017). Similarly, the core criteria of DMDD are severe temper tantrums and persistent irritability or anger, corresponding to our core-factor AD-II. In our sample, we could not find the differentiation between the trait component of chronic irritability and the state component of temper tantrums. In view of the fact that the conceptualization and assessment of AD have not yet been unequivocally determined, our results contribute to the standardization of this concept and suggest a broader conceptualization, with a strong core factor of irritability and emotional impulsivity.

In order to examine how AD relates to the externalizing disorders ADHD and ODD and to assess whether irritability/emotional impulsivity might be the common core feature underlying ADHD and ODD symptomatology, we specified our hypothesized bifactor S-1 model with 'pure indicators' of AD core symptomatology as general reference factor as a potential improvement to the traditional bifactor model, as well as four additional latent factor models to test for competing theories such as the assumption of an externalizing spectrum (Achenbach & Edelbrock, 1981) or a HiTOP-like organization of symptoms and dimensions (Kotov et al., 2017). High correlations between all the factors observed in the CFO suggested a common factor connecting these disorders within the externalizing spectrum, which we subsequently assessed with a variety of models. The good to adequate model fit of all models apart the unidimensional model should never be understood as a "decision-maker" *for* a model, but can only help us in our decision process, mainly by excluding models with an inadequate fit. This especially holds true for bifactor models, which tend to overfit (Bonifay et al., 2017) as a result of being less restrictive compared to other latent factor models. The unidimensional model showing an inadequate fit suggests that a model capturing general and well as specific aspects is better suited. Results from the higher-order and the bifactor models demonstrate that the specification of a common factor is justified. Compared to higher-order factor models, bifactor models come with the advantage that here, s-factors model dimensionality *beyond* the general factor, whereas in higher-order factor models, the lower-order factors model dimensionality *within* the general factor (Hartman, 2021). A problem arose from the varying loading ratios for g- and s-factors in the SOF model, as well as the differential ECV in the traditional bifactor model, which indicated that the associations of the individual domains with g differed, giving rise to the question of what this second-order g-factor really represents. As frequently observed in bifactor models (Eid et al., 2017), anomalous factor loadings in the traditional bifactor model impeded the interpretation of g- and s-factors. ECV values indicated that the g-factor did not represent all dimensions equally well, changing its meaning from a truly general factor to a mainly AD-II/ODD defined factor. Moreover, we found weakly defined s-factors, with ωHS values below the recommended cut-off of .50 (Reise et al., 2013). Though omega statistics are popular bifactor-specific indices, they are not without criticism (for a detailed discussion regarding problems with the ωHS see Rodriguez et al., 2016). According to the ωHS values alone, s-factors are often considered unstable and are dismissed for that reason. Hartman (2021), however, pointed out that unstable s-factors are only a reflection of what has already been well established from factor analytic effort – namely that dimensions free from the dominant general factor frequently display a "chaotic covariance structure of high instability" (p. 72). Their suggestion is to use bifactor models in a top-down fashion, choosing theoretically derived relevant item clusters, and in a next step to the design strong measures of these s-factors, instead of dismissing weak s-factors altogether. Interpreting the usefulness of specific factors for clinical practice or the individual research question based on other indicators such as the ECV, in combination with theoretical considerations, might therefore be a necessary first step, instead of solely relying on predefined global cut-off values. In the BI model, the ECV for all s-factor combined lay at 29% after partialling out the influence of the g-factor, therefore explaining an important part of the variance. These findings suggest that the s-factors are still meaningful, despite not meeting the cut-off criteria for ωHS. As we were interested in how well AD-core symptomatology defined through irritability and emotional impulsivity was able to explain the associations between ADHD and ODD, we applied the bifactor S-1 model with AD-II symptoms as pure indicators of the general reference factor. The bifactor S-1 model combines some of the advantages of a first-order correlated factor model and the traditional bifactor model: It retains the straightforward interpretability of all factors and their relation to each other of the CFO, and allows for the specification of a factor that explains variance common to all other s-factors, albeit to different extents, of the traditional bifactor model. In the bifactor S-1 model, all other s-factors can

be interpreted in relation to the general reference domain. Additionally, residual correlations can be interpreted meaningfully as partial correlations. Significant factor loadings and no anomalous results were observed, which greatly facilitated interpretation and is in accordance with previous studies pointing to a facilitated and less ambiguous interpretation of bifactor S-1 models compared to traditional bifactor models (Burns et al., 2020; Junghänel et al., 2020). Regarding the omega statistics, a similar, though slightly improved pattern compared to the traditional bifactor model was observed. All s-factors combined explained 30% of the variance in this model, after partialling out the influence of the reference factor, with ADHD-IN (10%) and ADHD-HI (11%) explaining the largest amount of the residual variance. The particularly high residual correlations between ADHD-IN and ADHD-HI showed that beyond the irritable/impulsive component, there is a remaining ADHD-specific component. Taken together, this model nicely demonstrates that the broadly defined AD-II factor captures important aspects of both ADHD dimensions, as well as the ODD-D dimension. At the same time, important disorder-specific aspects and dimensions remain and help us obtain a more nuanced picture of the associations between the symptom complexes. The differentiation of g- and s-factors brings along a number of potential advantages for research and clinical practice. Measures based on an array of problems tend to be particularly good prognosticators (Achenbach, 2021) and might be especially valuable for improving the diagnostic process (Lahey et al., 2021). In line with this, Pettersson et al., (2021) hypothesizes that the g-factor of psychopathology might be as useful for the psychiatric domain as the g-factor of intelligence has proven to be for the educational domain. It has been suggested that the g-factor, capturing correlations between different psychopathological dimensions, might result mainly from nonspecific etiological factors (Lahey et al., 2017) – a hypothesis that potentially provides a great target for further RDoC research. The s-factors might differentiate better between patients, especially those with a broad range of problems, who frequently show elevated scores on a range of scales (Pettersson et al., 2021). Longitudinal studies that examine the differential pathways, including specific protective and risk factors for people scoring high on different s-factors, are necessary.

The observation of remaining significant ODD and ADHD aspects that appear to be mostly free of irritability, emotional impulsivity, and emotion dysregulation is interesting. In future research, it would be worthwhile to examine more closely what these residual symptom complexes represent and what they are associated with. With regard to ODD, our results strengthen previous findings (Evans et al., 2017; Runions et al., 2016) reporting a distinction between a defiant and an irritable dimension of ODD, thus showing differential pathways and associations. Compared to the irritable dimension, the defiant dimension, which we identified as an important ODD-remaining aspect, has been shown to be associated more strongly with the odds of violence as an adult (Althoff et al., 2014) as well as disruptive and aggressive behavior (Burke et al., 2021). Regarding the ADHD remaining aspects that are free of irritability and emotional impulsivity, research has suggested particularly strong associations of the ADHD-IN dimension with sluggish cognitive tempo (Hartman et al., 2004), two concepts that are significantly related to academic functioning (Langberg et al., 2014). One could hypothesize that the ADHD-HI dimension now mainly captures hyperactivity and physical impulsivity, which is in line with the general understanding of impulsivity as a multidimensional construct (Berg et al., 2015). In future studies, it would be of great interest to ascertain, whether these residual ADHD- and ODD-specific aspects are now associated even more strongly with their respective external correlates. In a next step, and in line with Hartman (2021), strong measures that specifically assess these aspects could be designed.

To assess the convergent and divergent validity of our bifactor S-1 model, we added the three factors PROMIS Parent Proxy Anger Scale, FRUST-A and FRUST-M to our model and computed correlations with the reference factor as well as residual correlations with all s-factors. The strong correlation of the established PROMIS Parent Proxy Anger Scale with our AD-II reference factor, in combination with the low correlations of this scale with all other factors of our model, suggest that in line with our assumption, all scales except for AD-II indeed measure distinct constructs, independent of anger/rage. The correlations between the dimensions and emotion regulation strategies differ greatly in strength, with AD-II showing strong correlations with both, FRUST-A and FRUST-M. This constitutes an important validation of our model, because emotion dysregulation, has been strongly associated with ADHD and ODD (Ambrosini et al., 2013; Shaw et al., 2014) and corresponds here to our reference factor AD-II, while showing only small correlations with most of the remaining factors. Interestingly, there is one exception, as AD-PE correlate moderately with FRUST-A but not with FRUST-M. This supports the frequent finding that adaptive and maladaptive emotion regulation are distinct and not simply opposing constructs (Aldao & Nolen-Hoeksema, 2010; Grob & Smolenski, 2005). Indeed, the use of adaptive emotion regulation strategies might even be a protective factor for ADHD- and ODD-related symptomatology, beyond the mere absence of maladaptive emotion regulation strategies. Correspondingly, adaptive emotion regulation strategies, such as reappraisal

Table 4 Omega Statistics and Explained Common Variance of all Bifactor S-1 Models

AD-II	AD-II	AD-PE	AD-EX	ADHD-IN	ADHD-HI	ODD-D
ω	**0.98**	0.78	0.91	0.97	0.96	0.91
ωH	**0.90**					
ωHS		0.41	0.33	0.47	0.46	0.26
ECV	**0.70**	0.03	0.03	0.10	0.11	0.03
ODD-D						
ω	0.98	0.76	0.91	0.96	0.96	**0.98**
ωH						**0.90**
ωHS	0.11	0.40	0.26	0.43	0.35	
ECV	0.06	0.03	0.03	0.09	0.08	**0.72**
ADHD-IN						
ω	0.97	0.77	0.89	**0.98**	0.96	0.92
ωH				**0.82**		
ωHS	0.51	0.54	0.43		0.35	0.51
ECV	0.19	0.04	0.04	**0.60**	0.08	0.06
AD-HI						
ω	0.97	0.77	0.87	0.96	**0.98**	0.92
ωH					**0.85**	
ωHS	0.48	0.61	0.18	0.37		0.45
ECV	0.18	0.05	0.02	0.07	**0.64**	0.05

Note. Values for reference domain are marked in bold. AD = Affective dysregulation, II = irritability/emotional impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension, ω = omega (amount of variance accounted for by the g- and s-factors taken together), ωH = omega hierarchical (amount of variance accounted for by the g-factor), ωHS = omega hierarchical subscale (amount of variance accounted for by the s-factors), ECV = explained common variance (proportion of all common variance explained by that factor; for specific factors, the ECV computes the strength of a specific factor relative to all explained variance only of all items, even those not loading on the specific factor).

and acceptance, have been associated with fewer symptoms of psychopathology (Aldao et al., 2010, Braet et al., 2014).

In a final step, we compared bifactor S-1 models with different reference factors in order to examine, if AD-II indeed was the ideal core component of ADHD and ODD as hypothesized. All models yielded a good model fit and explained between 60% (ADHD-IN) to 72% (ODD-D) of the common variance (Table 4). The high ECV of all four BI S-1 models supports the general suggestion of a more parsimonious structure of psychopathology, as intended by the specifier approach in the ICD-11 (WHO, 2020) for ODD and AD symptomatology, instead of frequently diagnosing several comorbid disorders, such as ADHD *and* ODD or ADHD *and* DMDD. Despite the high ECV for the respective general factor, we observed a large gain of information through the assessment of all additional dimensions, indicated by the ECV for the s-factors in all models (Tables S8, S11-S13). Based on model fit alone, no model could be excluded at this point, which is unsurprising given the high correlations between all dimensions. The model with ODD-D as general reference factor demonstrates a slightly higher ECV (72%) than the model with AD-II (70%) as general reference factor. However, in the ODD-D model, the s-factor AD-II collapsed, as indicated by several anomalous factor loadings as well as the outstandingly low ωHS value (ωHS = 0.11), exacerbating straightforward interpretability and suggesting that this model does not capture the data in the most ideal way. The model with AD-II as reference factor explains a similar amount of common variance (70%) and shows no anomalous factor loadings or collapsing s-factors, and is straightforward to interpret. As pointed out above, a model should never be chosen based on statistical indices alone. The model with AD-II is derived based on theoretical considerations and fits well. The model with ODD-D as general reference factor shows good model fit, but does come with a number of statistical and theoretical problems, suggesting that AD-II might be a better reference factor than ODD-D: First, it has been suggested that the defiant dimension of ODD appears only later in life, as a consequence of impulsivity and weak emotion regulation skills (Beauchaine et al., 2010). Therefore, conceptualizing ODD-D as core feature, might not capture the problem behavior of all children, especially not of younger ones. This will have to be assessed in longitudinal studies. Second, when conceptualizing irritability/emotional impulsivity as core factor, this more parsimonious structure could potentially be extended into the internalizing spectrum. This seems worth examining for several reasons. In the DSM-5, DMDD is classified as a unipolar mood disorder, and previous studies have shown a strong association of AD symptomatology with internalizing symptomatology such as depression/dysthymia and anxiety (Copeland et al., 2013; Evans et al., 2017; Leibenluft, 2011; Waldman et al., 2021). The high correlations of AD-II with emotion regulation also suggest a potential association with

the internalizing spectrum, as mood and anxiety disorders have been strongly associated with more maladaptive and less adaptive emotion regulation strategies (Carthy et al., 2010; Silk et al., 2003). We therefore suggest an adaption and extension of the specifier approach. Our results indicate that irritability/emotional impulsivity could be the crucial factor explaining the high correlations frequently found between ADHD and ODD and could therefore be modelled as the core factor for ADHD- and ODD-related symptomatology. Related specifiers, such as "*with inattention*", "*with defiant behavior*" and "with *hyperarousal*" could be added to this core factor, leading to a more parsimonious structure of psychopathology instead of major symptom overlap and a number of comorbid diagnoses.

Limitations

One limitation of our study is that due to the relatively small age range of the children (8–12 years), we are unable to draw any conclusions about emotional impulsivity and irritability in children beyond that age range. In future research, it will be important to assess whether emotional impulsivity and irritability are similarly strongly associated with an array of disorders in other age groups. This is important as the AD-II factor showed a strong correlation with adaptive and maladaptive emotion regulation strategies, which has been suggested to be age dependent, potentially in the sense of a maladaptive shift, describing a reduction of adaptive strategies during adolescence (Cracco et al., 2017). Additionally, it will be important to assess whether similar results can be obtained in samples from other cultural and linguistic backgrounds. Another limitation is that we employed a parent rating scale to examine the associations between the relevant dimensions as our only measure, which will have to be expanded upon in future studies. We did not include any internalizing disorders (e.g. anxiety or depressive disorders) in our analyses. AD, however, has often been referred to as a transdiagnostic dimension, and it would be of great interest to examine how it relates to disorders in the internalizing spectrum. Furthermore, external correlates of the residual ADHD and ODD dimensions, such as sluggish cognitive tempo and violent behavior, should be examined in future research. Due to our cross-sectional study design, we cannot draw any conclusions relating to the onset and progression of AD and other externalizing disorders or the identification of risk or protective factors. It would be interesting to examine if the number of specifiers increases with age in irritable/emotionally impulsive children and if so, which specifiers appear at what time. Moreover, it would be useful to examine whether adaptive emotion regulation strategies might be a protective factor and maladaptive strategies a general liability factor for irritability and emotional impulsivity, as this could result in targeted prevention programs at an early age.

Conclusions

In the present study, we examined the factorial structure of AD, and found a stable structure, entailing one core-component, describing irritability/emotional impulsivity and two smaller factors describing positive emotionality and exuberance. We found a bifactor S-1 model with AD-II as general reference factor to lead to a straightforward interpretation of the associations between all dimensions. AD-II captured a major part of the shared variance of all AD, ADHD and ODD dimensions and at the same time, all dimensions explained important additional variance. Correlations with external correlates validated our model. When comparing models with different reference factors, AD-II as reference factor captured the data better than models with ADHD-/ODD-dimensions as reference factor. Our results support the specifier-approach adopted by the ICD-11 for ODD and AD symptomatology. We suggest an adaptation and extension of this approach in the future by selecting irritability/emotional impulsivity as the core diagnostic dimension, to which specifiers such as "*with inattention*" "*with defiant behavior*" "*with hyperarousal*", could be added. Additional specifiers, potentially extending into the internalizing spectrum should be examined in future studies and could eventually lead to an even more parsimonious structure of psychopathology. Our results suggest that comorbidities arise largely through how diagnoses are currently determined in the DSM-5 (APA, 2013) and the ICD-11 (WHO, 2020) – namely the combination of several domains within one disorder. The specifier approach might offer us a more accurate, richer, less stigmatizing and at the same time more parsimonious description of patients (Ruggero et al., 2019), which could additionally improve communication in research and clinical practice and lead to better treatment approaches.

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Authors' contributions Anja Görtz-Dorten, Tobias Banaschewski, Jörg M. Fegert, Charlotte Hanisch, Michael Kölch, Ulrike Ravens-Sieberer, Veit Roessner and Manfred Döpfner contributed to the study conception and design. Michaela Junghänel, Claudia Ginsberg, Franziska Frenk, Kristina Mücke, Sara Zaplana Labarga, Dorothee Bernheim, Anne Schüller, Anne Kaman and Julian Hinz performed data collection. Anne-Katrin Treier and Sabina Millenet coordinated the study and were responsible for data management. Analysis were performed by Michaela Junghänel. Statistical analyses were reviewed by Ann-Kathrin Thöne. The first draft of the manuscript was written by Michaela Junghänel and revised by Ann-Kathrin Thöne and Manfred Döpfner and all authors critically reviewed and approved the final manuscript.

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Declarations

Conflicts of Interest/Competing interests Manfred Döpfner and Anja Görtz-Dorten are authors of DISYPS and receive royalties.

Ethics approval Approval was obtained from the ethics committee of the University of Cologne (18–033), the University Hospital of Dresden (EK 35409218), the University Hospital of Mannheim (2018-554 N-MA) and the University Hospital of Ulm (297/18). The procedures used in this study adhere to the tenets of the Declaration of Helsinki.

Trial registration ADOPT Online: German Clinical Trials Register (DRKS) DRKS00014963. Registered 27 June 2018.

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Supporting Information for *Irritability and Impulsivity as a Core Feature of ADHD and ODD in Children*

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Table S1

Criteria for Item Reduction of the DADYS Questionnaire

Item	Item description	Skewness	Kurtosis	Not all response categories were used	> 90% not clinically relevant (response 0/1)	Number of item-item correlations < .3 **(bold if > 50%)**	Number of item-item correlations >.8	Of those with item-item correlation > .8, additionally rated as redundant by at least 2 out of 3 clinicians	Of those that fulfill criteria for exclusion: DSM-5 criteria for DMDD?	Excluded
1	Is a happy child					9				
2	Exhibits mood swings					7	1			
3	Responds positively to adults			Yes	Yes	**32**				
4	Switches easily between activities					9				
5	Calms down after being angry					12				
6	Easily frustrated					9				
7	Responds positively to peers			Yes	Yes	**32**				
8	Prone to temper tantrums					9	3	Yes	No	✓
9	Can wait for good things					11				
10	Takes pleasure in distress of others	XX	XX		Yes	**32**				✓
11	Can modulate excitement in high-energy situations					11				
12	Whiny/Clingy	X	X		Yes	**27**				✓
13	Prone to exuberant/ disruptive outbursts of emotions					7				
14	Angry about limit-setting					6				
15	Verbalize emotions					14				
16	Sad/Listless	X	XX		Yes	14				✓
17	Overly exuberant when engaging others to play					18				
18	Displays flat affect	XX	XX		Yes	**33**				✓

19	Responds negatively to friendly approaches by peers	XX	XX	Yes	Yes	**34**					✓
20	Impulsive					9					
21	Empathetic/Caring					**27**					
22	Displays exuberance others find intrusive		X			15					
23	Appropriate negative emotions when others are hostile					**37**					✓
24	Inappropriate emotions when engaging others in play	X	XX		Yes	13					✓
25	Demands must be met immediately					7					
26	Quickly angry/ Temper tantrums					7	7	Yes			✓
27	Often irritable					6	4				
28	Often offended					7	1				
29	Prolonged verbally aggressive tantrums					8	1	Yes	Yes		
30	Prolonged physically aggressive tantrums	X	XX		Yes	16			Yes		
31	Chronically irritable mood	X	X		Yes	10					
32	Easily annoyed by others					7					
33	Often loses temper					6	4				
34	Stays angry					10					
35	Is mostly angry	X		Yes	Yes	9					
36	Is often angry					7	5	Yes			✓
37	Loses temper easily					8	4	Yes			✓
38	Irritability causes problems					6					

Note. X = skewness/kurtosis larger than one standard deviation. XX = skewness/kurtosis larger than two standard deviations. Excluded items are highlighted in grey.

Table S2

Exploratory Factor Analysis of the DADYS Questionnaire

Item description	Factor 1	Factor 2	Factor 3
Is a happy child	-.37*	**.53***	.10
Exhibits mood swings	**.94***	.12*	.04
Responds positively to adults	.07	**.86***	-.01
Switches easily between activities	**-.45***	.36*	-.04
Calms down after being angry	**-.83***	.06	-.25*
Easily frustrated	**.79***	.00	.16*
Responds positively to peers	-.01	**.90***	.10
Can wait for good things	**-.61***	.12	-.14
Can modulate excitement in high-energy situations	-.41*	.26*	**-.42***
Prone to exuberant/disruptive outbursts of emotions	.61*	-.15*	**.30***
Angry about limit-setting	**.81***	.03	.10
Verbalizes emotions	-.19*	**.40***	-.17
Overly exuberant when engaging others to play	.15*	.02	**.71***
Impulsive	**.70***	.08	.31*
Empathetic/Caring	-.01	**.61***	-.08
Displays exuberance others find intrusive	.01	-.13	**.89***
Demands must be met immediately	**.71***	-.02	.22*
Often irritable	**.99***	.09	.01
Often offended	**.95***	.05	.01
Prolonged verbally aggressive temper tantrums	**1.02***	.24*	-.09
Prolonged physically aggressive tantrums	**.80***	.07	-.00
Chronically irritable mood	**.72***	-.23*	-.18*
Easily annoyed by others	**.75***	.00	.19*
Often loses temper	**.93***	.02	.06
Stays angry	**.98***	.00	-.39*
Is mostly angry	**.75***	-.28*	-.18*
Irritability causes problems	**.61***	-.17*	.34*

Note. The extraction method was principal axis factoring with an oblique (GEOMIN) rotation.

Assigned items are marked in bold.

Table S3

Standardized Factor Loadings (Standard Error) for the Confirmatory Factor Analysis of the Correlated Factors Models with Three AD Factors

Item	Factor loading (*SE*)
AD- Irritability/Impulsivity	
Exhibits mood swings	.89* (.02)
Switches easily between activities	.59* (.05)
Calms down after being angry[a]	.63* (.05)
Easily frustrated	.77* (.04)
Can wait for good things	.70* (.04)
Angry about limit-setting	.78* (.03)
Impulsive	.81* (.03)
Demands must be met immediately	.77* (.04)
Often irritable	.98* (.01)
Often offended	.93* (.01)
Prolonged verbally aggressive tantrums	.87* (.02)
Prolonged physically aggressive tantrums	.68* (.04)
Chronically irritable mood	.80* (.03)
Easily annoyed by others	.84* (.03)
Often loses temper	.93* (.02)
Stays angry[a]	.77* (.03)
Is mostly angry	.81* (.03)
Irritability causes problems	.87* (.02)
AD-Positive Emotionality	
Is a happy child	-.84* (.06)
Responds positively to adults[b]	-.36* (.09)
Responds positively to peers[b]	-.35* (.09)
Verbalizes emotions	-.66* (.06)
Empathetic/caring	-.60* (.06)
AD-Exuberance	
Can modulate excitement in high-energy situations	.81* (.04)
Prone to exuberant/disruptive outbursts of emotions	.96* (.02)
Overly exuberant when engaging others to play	.70* (.05)
Displays exuberance others find intrusive	.81* (.03)
Residual correlations	
[a]	.67*
[b]	.70*

Note. SE = standard error. AD = affective dysregulation.

* = $p < .001$.

[a,b] Residuals of these items were allowed to correlate.

Table S4

Standardized Factor Loadings (Standard Error) for the Unidimensional Model (Uni), the First-Order Correlated Factor Model (CFO) and Original Standardized Factor Loadings for the Second-Order Correlated Factor Model (SOF) before the Schmid-Leiman Transformation

Item	Uni	CFO	SOF
AD- Irritability/Impulsivity			
Exhibits mood swings	.86* (.02)	.89* (.02)	.89* (.02)
Switches easily between activities	.59* (.03)	.63* (.04)	.62* (.04)
Calms down after being angry[a]	.61* (.03)	.65* (.04)	.65* (.04)
Easily frustrated	.79* (.02)	.82* (.02)	.82* (.02)
Can wait for good things	.71* (.03)	.75* (.03)	.75* (.03)
Angry about limit-setting	.80* (.02)	.83* (.02)	.83* (.02)
Impulsive	.79* (.02)	.83* (.02)	.83* (.02)
Demands must be met immediately	.77* (.03)	.80* (.03)	.80* (.03)
Often irritable	.94* (.01)	.96* (.01)	.96* (.01)
Often offended	.90* (.01)	.92* (.01)	.92* (.01)
Prolonged verbally aggressive tantrums	.81* (.02)	.84* (.02)	.84* (.02)
Prolonged physically aggressive tantrums	.65* (.03)	.69* (.03)	.69* (.03)
Chronically irritable mood	.71* (.03)	.75* (.03)	.75* (.03)
Easily annoyed by others	.81* (.02)	.84* (.02)	.84* (.02)
Often loses temper	.91* (.01)	.94* (.01)	.94* (.01)
Stays angry[a]	.70* (.03)	.74* (.03)	.74* (.03)
Is mostly angry	.74* (.03)	.78* (.03)	.78* (.03)
Irritability causes problems	.85* (.02)	.89* (.02)	.89* (.02)
AD-Positive Emotionality			
Is a happy child	-.56* (.04)	.81* (.04)	.81* (.04)
Responds positively to adults[b]	-.31* (.05)	.46* (.07)	.46* (.07)
Responds positively to peers[b]	-.30* (.05)	.46* (.07)	.45* (.07)
Verbalizes emotions	-.50* (.04)	.71* (.05)	.72* (.05)
Empathetic/caring	-.44* (.04)	.63* (.05)	.63* (.05)
AD-Exuberance			
Can modulate excitement in high-energy situations	.72* (.03)	.83* (.03)	.83* (.03)
Prone to exuberant/disruptive outbursts of emotions	.82* (.02)	.94* (.02)	.95* (.02)
Overly exuberant when engaging others to play	.58* (.04)	.66* (.04)	.65* (.04)
Displays exuberance others find intrusive	.68* (.03)	.78* (.03)	.77* (.03)
ADHD-Inattention			
Overlooks details	.72* (.03)	.82* (.02)	.81* (.02)
Sustained attention	.81* (.02)	.90* (.02)	.90* (.02)
Does not listen	.72* (.03)	.82* (.03)	.82* (.03)
Incomplete execution of tasks	.82* (.02)	.91* (.02)	.91* (.02)
Difficulties with organization	.77* (.02)	.86* (.02)	.86* (.02)
Aversion to cognitive effort	.80* (.02)	.90* (.02)	.90* (.02)
Loses things	.65* (.03)	.75* (.03)	.75* (.03)

Easily distracted	.83*(.02)	.92* (.02)	.93* (.02)
Forgetful	.74* (.03)	.83* (.02)	.83* (.02)
ADHD-Hyperactivity/Impulsivity			
Fidgets/squirms	.73* (.03)	.80* (.02)	.80* (.03)
Gets up	.72* (.03)	.79* (.03)	.79* (.03)
Walks/climbs	.80* (.03)	.86* (.02)	.86* (.02)
Inner restlessness	.69* (.04)	.77* (.04)	.77* (.04)
Difficulties playing quietly	.79* (.03)	.87* (.02)	.87* (.02)
Extreme agitation	.86* (.02)	.92* (.02)	.93* (.02)
Frequently on the go	.80* (.03)	.87* (.02)	.87* (.02)
Blurts	.73* (.03)	.81* (.03)	.81* (.03)
Difficulties waiting for own turn	.78* (.02)	.87* (.02)	.87* (.02)
Interrupts/disturbs	.81* (.02)	.91* (.02)	.91* (.02)
Talks a lot	.62* (.03)	.70* (.04)	.70* (.04)
ODD-D			
Argues with adults	.76* (.02)	.86* (.02)	.86* (.02)
Does not follow rules	.81* (.02)	.91* (.02)	.92* (.02)
Annoys others	.71* (.03)	.81* (.03)	.81* (.03)
Accuses others	.75* (.02)	.86* (.02)	.87* (.02)
Spiteful/vindictive	.61* (.04)	.70* (.04)	.69* (.04)
Residual correlations			
[a]	.68*	.65*	.65*
[b]	.73*	.69*	.70*

Note. CFO = first-order correlated factor model, SOF = second-order correlated factor model, AD = affective dysregulation, ADHD = attention-deficit/hyperactivity disorder, ODD-D = oppositional defiant disorder – only defiant dimension.

* = $p < .001$.

[a,b] Residuals of these items were allowed to correlate.

Table S5

Standardized Factor Loadings (Standard Error) of the First-Order Factors on the Second-Order Factor and Residual Variances (Standard Error) for the Second-Order Correlated Factor Model (SFO)

Dimension	Factor Loading on Second-Order Factor	Residual Variance
External		
AD-Irritability/Impulsivity	.90* (.01)	.20* (.03)
AD-Positive Emotionality	-.70* (.04)	.51* (.05)
AD-Exuberance	.91* (.02)	.18* (.04)
ADHD-Inattention	.81* (.02)	.34* (.04)
ADHD-Hyperactivity/Impulsivity	.86* (.02)	.27* (.03)
ODD-D	.89* (.02)	.20* (.03)

Note. AD = affective dysregulation, ADHD = attention-deficit/hyperactivity disorder, ODD-D = oppositional defiant disorder – only defiant dimension.

* = p <.001.

Table S6

Standardized Factor Loadings as Obtained by Applying the Schmid-Leiman Transformation to the Second-Order Factor Model

Item	Item Loading on 2nd-order factor (g)	Item Loading on 1st-order factor (s)
AD- Irritability/Impulsivity		
Exhibits mood swings	.79	.39
Switches easily between activities	.56	.28
Calms down after being angry[a]	.58	.29
Easily frustrated	.73	.36
Can wait for good things	.67	.33
Angry about limit-setting	.75	.37
Impulsive	.74	.37
Demands must be met immediately	.72	.36
Often irritable	.86	.43
Often offended	.82	.41
Prolonged verbally aggressive tantrums	.75	.37
Prolonged physically aggressive tantrums	.62	.31
Chronically irritable mood	.67	.33
Easily annoyed by others	.75	.37
Often loses temper	.84	.42
Stays angry[a]	.66	.33
Is mostly angry	.70	.35
Irritability causes problems	.79	.39
M (*SD*)	.72 (.08)	.36 (.04)
Ratio (g/s)	2.01	
AD-Positive Emotionality		
Is a happy child	-.57	.57
Responds positively to adults[b]	-.32	.33
Responds positively to peers[b]	-.31	.32
Verbalizes emotions	-.50	.51
Empathetic/caring	-.44	.45
M (*SD*)	-.43 (.11)	.44 (.11)
Ratio (g/s)	-.98	
AD-Exuberance		
Can modulate excitement in high-energy situations	.75	.35
Prone to exuberant/disruptive outbursts of emotions	.86	.40
Overly exuberant when engaging others to play	.59	.28
Displays exuberance others find intrusive	.70	.32
M (*SD*)	.73 (.11)	.34 (.05)
Ratio (g/s)	2.16	
ADHD-Inattention		
Overlooks details	.66	.47
Sustained attention	.73	.52
Does not listen	.67	.48
Incomplete execution of tasks	.74	.53
Difficulties with organization	.70	.50
Aversion to cognitive effort	.73	.52
Loses things	.61	.43
Easily distracted	.75	.54
Forgetful	.67	.48

M (*SD)*	.70 (.05)		.50 (.04)
Ratio (g/s)		1.40	
ADHD-Hyperactivity/Impulsivity			
Fidgets/squirms	.69		.41
Gets up	.68		.41
Walks/climbs	.74		.45
Inner restlessness	.66		.40
Difficulties playing quietly	.75		.44
Extreme agitation	.79		.48
Frequently on the go	.75		.45
Blurts	.70		.42
Difficulties waiting for own turn	.75		.45
Interrupts/disturbs	.78		.47
Talks a lot	.60		.36
M (*SD)*	.72 (.06)		.43 (.03)
Ratio (g/s)		1.66	
ODD-D			
Argues with adults	.74		.37
Does not follow rules	.82		.41
Annoys others	.72		.36
Accuses others	.77		.39
Spiteful/vindictive	.62		.31
M (*SD)*	.73 (.07)		.37 (.04)
Ratio (g/s)		1.99	
Total M*(SD)*	.61 (.35)		.30 (.07)

Note. M = mean, *SD* = standard deviation, AD = affective dysregulation, ADHD = attention-deficit/hyperactivity disorder, ODD-D = oppositional defiant disorder – only defiant dimension.

[a,b] Residuals of these items were allowed to correlate.

Table S7

Standardized Factor Loadings (Standard Error), Omega Statistics and Explained Common Variance for the Bifactor Model (BI)

Item	G External	S AD-II	S AD-PE	S AD-EX	S ADHD-IN	S ADHD-HI	S ODD-D
AD-Irritability/Impulsivity							
Exhibits mood swings	.77* (.03)	.46* (.04)					
Switches easily between activities	.58* (.04)	.21* (.06)					
Calms down after being angry[a]	.56* (.04)	.38* (.05)					
Easily frustrated	.75* (.03)	.31* (.04)					
Can wait for good things	.73* (.03)	.09 (.06)					
Angry about limit-setting	.79* (.03)	.21* (.04)					
Impulsive	.79* (.02)	.18* (.05)					
Demands must be met immediately	.75* (.03)	.23* (.05)					
Often irritable	.85* (.02)	.45* (.04)					
Often offended	.80* (.02)	.47* (.04)					
Prolonged verbally aggressive tantrums	.69* (.03)	.56* (.04)					
Prolonged physically aggressive tantrums	.56* (.04)	.49* (.06)					
Chronically irritable mood	.60* (.04)	.55* (.05)					
Easily annoyed by others	.77* (.03)	.31* (.04)					
Often loses temper	.84* (.02)	.40* (.04)					
Stays angry[a]	.58* (.04)	.57* (.04)					
Is mostly angry	.60* (.04)	.64* (.06)					
Irritability causes problems	.84* (.02)	.22* (.04)					
AD-Positive Emotionality							
Is a happy child	-.57* (.04)		.46* (.06)				
Responds positively to adults[b]	-.32* (.06)		.46* (.07)				
Responds positively to peers[b]	-.30* (.06)		.55* (.07)				
Verbalizes emotions	-.51* (.04)		.39* (.06)				

Empathetic/caring	-.44* (.05)	.54* (.06)			
AD-Exuberance					
Can modulate excitement in high-energy situations	.76* (.03)		.10 (.06)		
Prone to exuberant/disruptive outbursts of emotions	.86* (.02)		.17* (.05)		
Overly exuberant when engaging others to play	.58* (.04)		.45* (.09)		
Displays exuberance others find intrusive	.69* (.04)		.81* (.15)		
ADHD-Inattention					
Overlooks details	.65* (.03)			.53* (.04)	
Sustained attention	.74* (.03)			.49* (.04)	
Does not listen	.68* (.04)			.41* (.04)	
Incomplete execution of tasks	.74* (.03)			.52* (.04)	
Difficulties with organization	.67* (.03)			.60* (.04)	
Aversion to cognitive effort	.76* (.03)			.40* (.04)	
Loses things	.56* (.04)			.59* (.04)	
Easily distracted	.78* (.03)			.45* (.04)	
Forgetful	.64* (.03)			.58* (.04)	
ADHD-Hyperactivity/Impulsivity					
Fidgets/squirms	.66* (.03)				.52* (.04)
Gets up	.66* (.04)				.47* (.05)
Walks/climbs	.70* (.04)				.56* (.05)
Inner restlessness	.65* (.04)				.43* (.06)
Difficulties playing quietly	.74* (.03)				.45* (.04)
Extreme agitation	.77* (.03)				.54* (.04)
Frequently on the go	.72* (.03)				.53* (.04)
Blurts	.72* (.03)				.35* (.05)
Difficulties waiting for own turn	.79* (.03)				.27* (.05)
Interrupts/disturbs	.82* (.02)				.25* (.04)
Talks a lot	.61* (.04)				.31* (.05)
ODD-Defiant Dimension					
Argues with adults	.77* (.03)				.41* (.04)

Does not follow rules	.82* (.02)						.44* (.05)
Annoys others	.72* (.03)						.46* (.06)
Accuses others	.78* (.03)						.20* (.05)
Spiteful/vindictive	.62* (.05)						.38* (.08)
ω	.99	.98	.78	.92	.97	.96	.92
ωH/ ωHS	.89	.21	.44	.20	.32	.25	.19
ECV	.71	.08	.03	.03	.07	.06	.02

Note. G = general factor, S = specific factor, AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, ODD-D = oppositional defiant disorder – only defiant dimension, ω = omega (amount of variance accounted for by the g- and s-factors taken together), ωH = omega hierarchical (amount of variance accounted for by the g-factor), ωHS = omega hierarchical subscale (amount of variance accounted for by the s-factors), ECV = explained common variance (proportion of all common variance explained by that factor; for specific factors, the ECV computes the strength of a specific factor relative to all explained variance only of all items, even those not loading on the specific factor).

* = p <.001.

[a,b] Residuals of these items were allowed to correlate.

Table S8

Standardized Factor Loadings (Standard Error) for the Bifactor S-1 Model (BI S-1) with AD-II as Reference Factor

Item	Reference AD-II	S AD-PE	S AD-EX	S ADHD-IN	S ADHD-HI	S ODD-D
AD-Irritability/Impulsivity						
Exhibits mood swings	.89* (.02)					
Switches easily between activities	.63* (.04)					
Calms down after being angry[a]	.65* (.04)					
Easily frustrated	.82* (.02)					
Can wait for good things	.75* (.03)					
Angry about limit-setting	.83* (.02)					
Impulsive	.83* (.02)					
Demands must be met immediately	.80* (.03)					
Often irritable	.96* (.01)					
Often offended	.92* (.01)					
Prolonged verbally aggressive tantrums	.84* (.02)					
Prolonged physically aggressive tantrums	.69* (.03)					
Chronically irritable mood	.74* (.03)					
Easily annoyed by others	.84* (.02)					
Often loses temper	.93* (.01)					
Stays angry[a]	.74* (.03)					
Is mostly angry	.78* (.03)					
Irritability causes problems	.89* (.02)					
AD-Positive Emotionality						
Is a happy child	-.59* (.04)	.49* (.06)				
Responds positively to adults[b]	-.33* (.06)	.45* (.07)				
Responds positively to peers[b]	-.32* (.06)	.56* (.07)				
Verbalizes emotions	-.52* (.04)	.33* (.07)				

Empathetic/caring	-.45* (.05)	.49* (.07)			
AD-Exuberance					
Can modulate excitement in high-energy situations	.72* (.03)		.31* (.06)		
Prone to exuberant/disruptive outbursts of emotions	.82* (.02)		.30* (.05)		
Overly exuberant when engaging others to play	.50* (.05)		.59* (.05)		
Displays exuberance others find intrusive	.57* (.04)		.77* (.05)		
ADHD-Inattention					
Overlooks details	.57* (.04)			.60* (.04)	
Sustained attention	.63* (.04)			.65* (.03)	
Does not listen	.59* (.04)			.55* (.04)	
Incomplete execution of tasks	.64* (.03)			.65* (.03)	
Difficulties with organization	.56* (.04)			.69* (.03)	
Aversion to cognitive effort	.68* (.03)			.52* (.03)	
Loses things	.47* (.04)			.63* (.04)	
Easily distracted	.68* (.03)			.59* (.03)	
Forgetful	.54* (.04)			.67* (.03)	
ADHD-Hyperactivity/Impulsivity					
Fidgets/squirms	.54* (.04)				.63* (.03)
Gets up	.54* (.04)				.63* (.04)
Walks/climbs	.57* (.04)				.71* (.03)
Inner restlessness	.55* (.04)				.54* (.05)
Difficulties playing quietly	.62* (.04)				.61* (.04)
Extreme agitation	.66* (.03)				.65* (.03)
Frequently on the go	.61* (.04)				.64* (.04)
Blurts	.61* (.04)				.53* (.04)
Difficulties waiting for own turn	.70* (.03)				.45* (.04)
Interrupts/disturbs	.72* (.03)				.49* (.04)
Talks a lot	.52* (.04)				.47* (.05)
ODD-Defiant Dimension					
Argues with adults	.73* (.03)				.45* (.04)

Does not follow rules	.78* (.02)	.51* (.05)
Annoys others	.68* (.03)	.49* (.05)
Accuses others	.75* (.03)	.33* (.05)
Spiteful/vindictive	.60* (.04)	.39* (.07)

Note. S = specific factor, AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension.

* = $p < .001$.

[a,b] Residuals of these items were allowed to correlate.

Table S9

Residual Correlations in the Bifactor S-1 Model (BI S-1) with AD-II as Reference Factor

Variable	1	2	3	4	5	6
1. AD-II	–					
2. AD-PE	n/a	–				
3. AD-EX	n/a	.16	–			
4. ADHD-IN	n/a	-.05	.27*	–		
5. ADHD-HI	n/a	.13	.56*	.58*	–	
6. ODD-D	n/a	-.08	.09	.19*	.25*	–

Note. AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension.

* = $p < .05$.

n/a = not applicable, as correlations between the reference factor and the specific factors were restrained to 0.

Table S10

(Residual) Correlations of the Bifactor S-1 Model (BI S-1) Factors with AD-II as Reference Factor with External Correlates

Factor BI S-1 model	PROMIS	FRUST-A	FRUST-M
AD-II (Reference factor)	.90*	-.71*	.82*
AD-PE	.08*	.46*	-.11*
AD-EX	-.09*	.10	-.18*
ADHD-IN	-.11*	-.05	-.08*
ADHD-HI	-.14*	.02	-.10*
ODD-D	-.04	.00	.09*
PROMIS		-.59*	.74*
FRUST-A			-.77*

Note. AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension. * = $p < .05$. PROMIS = Patient-Reported Outcomes Measurement Information System – Parent Proxy Anger Scale, A = adaptive, M = maladaptive

Table S11

Standardized Factor Loadings (Standard Error) for the Bifactor S-1 Model (BI S-1; ODD-D) with ODD-D as Reference Factor

Item	Reference ODD-D	S AD-II	S AD-PE	S AD-EX	S ADHD-IN	S ADHD-HI
AD-Irritability/Impulsivity						
Exhibits mood swings	.82* (.03)	.36* (.05)				
Switches easily between activities	.59* (.04)	.22* (.06)				
Calms down after being angry[a]	.59* (.04)	.36* (.06)				
Easily frustrated	.79* (.03)	.19* (.06)				
Can wait for good things	.76* (.03)	-.05 (07)				
Angry about limit-setting	.81* (.03)	.12 (.05)				
Impulsive	.83* (.02)	-.01 (.06)				
Demands must be met immediately	.79* (.03)	.10 (.06)				
Often irritable	.90* (.02)	.33* (.04)				
Often offended	.86* (.02)	.36* (.04)				
Prolonged verbally aggressive tantrums	.76* (.03)	.44* (.05)				
Prolonged physically aggressive tantrums	.62* (.04)	.39* (.07)				
Chronically irritable mood	.65* (.04)	.55* (.05)				
Easily annoyed by others	.81* (.03)	.17* (.06)				
Often loses temper	.89* (.02)	.26* (.05)				
Stays angry[a]	.64* (.04)	.53* (.05)				
Is mostly angry	.66* (.04)	.57* (.06)				
Irritability causes problems	.88* (.02)	.07 (.05)				
AD-Positive Emotionality						
Is a happy child	-.55* (.04)		.74* (.08)			
Responds positively to adults[b]	-.31* (.06)		.39* (.07)			
Responds positively to peers[b]	-.30* (.06)		.49* (.08)			
Verbalizes emotions	-.51* (.04)		.27* (.07)			

Empathetic/caring	-.44* (.05)	.34* (.07)			
AD-Exuberance					
Can modulate excitement in high-energy situations	.74* (.03)		.20* (.07)		
Prone to exuberant/disruptive outbursts of emotions	.84* (.02)		.19* (.05)		
Overly exuberant when engaging others to play	.53* (.05)		.56* (.06)		
Displays exuberance others find intrusive	.62* (.04)		.79* (.08)		
ADHD-Inattention					
Overlooks details	.60* (.04)			.56* (.04)	
Sustained attention	.67* (.03)			.60* (.04)	
Does not listen	.63* (.04)			.49* (.04)	
Incomplete execution of tasks	.68* (.03)			.60* (.03)	
Difficulties with organization	.61* (.04)			.66* (.03)	
Aversion to cognitive effort	.72* (.03)			.47* (.04)	
Loses things	.51* (.04)			.61* (.04)	
Easily distracted	.72* (.03)			.54* (.03)	
Forgetful	.58* (.04)			.63* (.04)	
ADHD-Hyperactivity/Impulsivity					
Fidgets/squirms	.61* (.04)				.57* (.04)
Gets up	.60* (.04)				.57* (.04)
Walks/climbs	.64* (.04)				.64* (.04)
Inner restlessness	.61* (.04)				.47* (.05)
Difficulties playing quietly	.69* (.04)				.53* (.04)
Extreme agitation	.73* (.03)				.57* (.04)
Frequently on the go	.68* (.04)				.57* (.04)
Blurts	.67* (.04)				.46* (.04)
Difficulties waiting for own turn	.75* (.03)				.36* (.04)
Interrupts/disturbs	.78* (.03)				.39* (.04)
Talks a lot	.57* (.04)				.40* (.05)
ODD-Defiant Dimension					

Argues with adults	.80* (.02)
Does not follow rules	.85* (.02)
Annoys others	.75* (.03)
Accuses others	.79* (.02)
Spiteful/vindictive	.65* (.04)

Note. S = specific factor, ODD-D = oppositional defiant disorder – only defiant dimension, AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, HI = hyperactivity/impulsivity.

* = $p < .001$.

[a,b] Residuals of these items were allowed to correlate.

Table S12

Standardized Factor Loadings (Standard Error) for the Bifactor S-1 Model (BI S-1; ADHD-IN) with ADHD-IN as Reference Factor

Item	Reference ADHD-IN	S AD-II	S AD-PE	S AD-EX	S ADHD-HI	S ODD-D
AD-Irritability/Impulsivity						
Exhibits mood swings	.60* (.04)	.66* (.03)				
Switches easily between activities	.47* (.04)	.41* (.05)				
Calms down after being angry[a]	.41* (.05)	.54* (.04)				
Easily frustrated	.63* (.04)	.51* (.04)				
Can wait for good things	.64* (.04)	.36* (.04)				
Angry about limit-setting	.63* (.04)	.54* (.04)				
Impulsive	.67* (.04)	.46* (.04)				
Demands must be met immediately	.59* (.04)	.53* (.04)				
Often irritable	.68* (.03)	.68* (.03)				
Often offended	.63* (.04)	.68* (.03)				
Prolonged verbally aggressive tantrums	.50* (.04)	.71* (.03)				
Prolonged physically aggressive tantrums	.41* (.05)	.60* (.05)				
Chronically irritable mood	.39* (.05)	.71* (.04)				
Easily annoyed by others	.64* (.04)	.53* (.04)				
Often loses temper	.67* (.03)	.65* (.03)				
Stays angry[a]	.37* (.05)	.72* (.03)				
Is mostly angry	.38* (.06)	78* (.04)				
Irritability causes problems	.72* (.03)	.50* (.04)				
AD-Positive Emotionality						
Is a happy child	-.39* (.05)		.83* (.06)			
Responds positively to adults[b]	-.24* (.06)		.43* (.06)			
Responds positively to peers[b]	-.20* (.06)		.50* (.07)			
Verbalizes emotions	-.46* (.05)		.48* (.06)			

Empathetic/caring	-.39* (.05)	.51* (.06)		
AD-Exuberance				
Can modulate excitement in high-energy situations	.66* (.04)		.44* (.05)	
Prone to exuberant/disruptive outbursts of emotions	.61* (.04)		.81* (.05)	
Overly exuberant when engaging others to play	.46* (.05)		.48* (.06)	
Displays exuberance others find intrusive	.57* (.05)		.51* (.05)	
ADHD-Inattention				
Overlooks details	.81* (.02)			
Sustained attention	.89* (.02)			
Does not listen	.81* (.03)			
Incomplete execution of tasks	.90* (.02)			
Difficulties with organization	.85* (.02)			
Aversion to cognitive effort	.89* (.02)			
Loses things	.74* (.03)			
Easily distracted	.92* (.02)			
Forgetful	.82* (.02)			
ADHD-Hyperactivity/Impulsivity				
Fidgets/squirms	.64* (.03)			.48* (.04)
Gets up	.66* (.04)			.42* (.05)
Walks/climbs	.69* (.04)			.53* (.04)
Inner restlessness	.58* (.05)			.53* (.05)
Difficulties playing quietly	.71* (.04)			.48* (.04)
Extreme agitation	.70* (.04)			.64* (.04)
Frequently on the go	.67* (.04)			.58* (.04)
Blurts	.65* (.04)			.50* (.04)
Difficulties waiting for own turn	.74* (.03)			.44* (.04)
Interrupts/disturbs	.78* (.03)			.42* (.04)
Talks a lot	.53* (.04)			.50* (.04)
ODD-Defiant Dimension				

Argues with adults	.56* (.04)	.67* (.04)
Does not follow rules	.63* (.04)	.66* (.04)
Annoys others	.54* (.05)	.62* (.05)
Accuses others	.64* (.04)	.54* (.04)
Spiteful/vindictive	.42* (.06)	.62* (.07)

Note. S = specific factor, ADHD = attention-deficit/hyperactivity disorder, IN = inattention, AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, HI = hyperactivity/impulsivity, ODD-D = oppositional defiant disorder – only defiant dimension.

* = $p < .001$.

[a,b] Residuals of these items were allowed to correlate.

Table S13

Standardized Factor Loadings (Standard Error) for the Bifactor S-1 Model (BI S-1; ADHD-HI) with ADHD-HI as Reference Factor

Item	Reference ADHD-HI	S AD-II	S AD-PE	S AD-EX	S ADHD-IN	S ODD-D
AD-Irritability/Impulsivity						
Exhibits mood swings	.61* (.04)	.66* (.03)				
Switches easily between activities	.44* (.04)	.45* (.05)				
Calms down after being angry[a]	.40* (.05)	.56* (.04)				
Easily frustrated	.63* (.04)	.51* (.04)				
Can wait for good things	.66* (.03)	.32* (.04)				
Angry about limit-setting	.64* (.04)	.52* (.04)				
Impulsive	.71* (.03)	.39* (.04)				
Demands must be met immediately	.63* (.04)	.48* (.04)				
Often irritable	.69* (.03)	.67* (.03)				
Often offended	.64* (.04)	.67* (.03)				
Prolonged verbally aggressive tantrums	.53* (.05)	.70* (.04)				
Prolonged physically aggressive tantrums	.44* (.05)	.58* (.05)				
Chronically irritable mood	.40* (.05)	.73* (.04)				
Easily annoyed by others	.65* (.03)	.51* (.03)				
Often loses temper	.70* (.03)	.62* (.03)				
Stays angry[a]	.40* (.05)	.71* (.03)				
Is mostly angry	.42* (.06)	.74* (.04)				
Irritability causes problems	.74* (.03)	.46* (.04)				
AD-Positive Emotionality						
Is a happy child	-.32* (.05)		.86* (.06)			
Responds positively to adults[b]	-.19* (.06)		.46* (.06)			
Responds positively to peers[b]	-.15* (.06)		.54* (.07)			

Verbalizes emotions	-.41* (.05)	.45* (.05)		
Empathetic/caring	-.34* (.05)	.44* (.05)		
AD-Exuberance				
Can modulate excitement in high-energy situations	.70* (.04)		.39* (.06)	
Prone to exuberant/disruptive outbursts of emotions	.72* (.03)		.65* (.06)	
Overly exuberant when engaging others to play	.60* (.04)		.17* (.07)	
Displays exuberance others find intrusive	.71* (.04)		.18* (.06)	
ADHD-Inattention				
Overlooks details	.63* (.04)			.56* (.03)
Sustained attention	.74* (.03)			.48* (.04)
Does not listen	.68* (.04)			.43* (.04)
Incomplete execution of tasks	.73* (.03)			.54* (.04)
Difficulties with organization	.65* (.04)			.61* (.03)
Aversion to cognitive effort	.74* (.03)			.46* (.04)
Loses things	.52* (.05)			.62* (.04)
Easily distracted	.77* (.03)			.49* (.04)
Forgetful	.62* (.04)			.59* (.04)
ADHD-Hyperactivity/Impulsivity				
Fidgets/squirms	.80* (.02)			
Gets up	.79* (.03)			
Walks/climbs	.86* (.02)			
Inner restlessness	.76* (.04)			
Difficulties playing quietly	.86* (.02)			
Extreme agitation	.92* (.02)			
Frequently on the go	.87* (.02)			
Blurts	.81* (.03)			
Difficulties waiting for own turn	.87* (.02)			
Interrupts/disturbs	.91* (.02)			
Talks a lot	.70* (.03)			

ODD-Defiant Dimension		
Argues with adults	.61* (.04)	.62* (.04)
Does not follow rules	.65* (.04)	.65* (.04)
Annoys others	.57* (.05)	.58* (.05)
Accuses others	.65* (.03)	.54* (.04)
Spiteful/vindictive	.49* (.06)	.53* (.07)

Note. S = specific factor, ADHD = attention-deficit/hyperactivity disorder, HI = hyperactivity/impulsivity, AD = affective dysregulation, II = irritability/impulsivity, PE = positive emotionality, EX = exuberance, ODD-D = oppositional defiant disorder – only defiant dimension.

* = $p < .001$.

[a,b] Residuals of these items were allowed to correlate

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<u>Übersicht der Publikationen:</u>

Junghänel, M., Rodenacker, K., Dose, C., & Döpfner, M. (2020). Applying the bifactor S-1 model to ratings of ADHD/ODD symptoms: A commentary on Burns et al. (2019) and a re-analysis. *Journal of Abnormal Child Psychology*, *48*(7), 905–910. https://doi.org/10.1007/s10802-020-00637-4

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