Longitudinal relations between impaired executive function and symptoms of psychiatric disorders in childhood

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Background: Malfunctioning of executive functions correlates with psychopathology in children. However, the directionality, the extent to which the relation varies for various disorders, and whether prospective relations afford causal interpretations are not known. Methods: A community sample of Norwegian children (n = 874) was studied biennially from the age of 6 to 14 years. Executive functions were assessed using the Behavior Rating Inventory of Executive Function Teacher-report and symptoms of psychopathology were assessed using the Preschool Age Psychiatric Assessment (age 6; parents) and Child and Adolescent Psychiatric Assessment (ages 8–14; children and parents). Prospective reciprocal relations were examined using a random intercept cross-lagged panel model that adjusts for all unobserved time-invariant confounders. Results: Even when time-invariant confounders were accounted for, reduced executive functions predicted increased symptoms of depressive disorders, anxiety disorders, attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD) 2 years later, even when previous changes in these symptoms were adjusted for. The level of prediction (B = .83, 95% CI [.37, 1.3]) was not different for different disorders or ages. Conversely, reduced executive functions were predicted by increased symptoms of all disorders (B = .01, 95% CI [.01, .02]). Conclusions: Reduced executive functioning may be involved in the etiology of depression, anxiety, ADHD, and ODD/CD to an equal extent. Moreover, increased depression, anxiety, ADHD, and ODD/CD may negatively impact executive functioning. Keywords: Adolescents; BRIEF; CAPA; child development; developmental psychopathology; longitudinal; mental health; self-regulation; PAPA; psychiatric disorder; p factor.

Introduction
Most childhood mental disorders involve problems with self-regulation (Nigg, 2017), whether it be the regulation of emotions, behavior, or attention. Executive functions are the cognitive mechanisms required for such regulation (Diamond, 2013). Cross-sectional studies indicate that poor executive functions are related to psychopathology in children and adolescents (Kavanaugh et al., 2019; Mullin, Perks, Haraden, Snyder, & Hankin, 2020). However, it remains to be adequately understood whether impaired executive functions cause child psychopathology or whether they are a consequence of child psychopathology.

Cause: executive function deficiencies impair mental health
Recent factor analytic studies strongly suggest that different psychopathologies have, to some extent, a common origin – a p factor (Caspi & Moffitt, 2018). Although the content of this p factor remains to be determined, executive functions have been proposed to be such a transdiagnostic risk factor (Snyder, Miyake, & Hankin, 2015). Impaired executive functioning is related to a diversity of mental disorders, although the underlying mechanisms of this relationship may vary between disorders. Because executive functions are central to cognitive self-regulation (Nigg, 2017), one plausible explanation for the relationship between executive functions and emotional disorders is that deficits in executive functions involve poor inhibition (Caspi & Moffitt, 2018), which may manifest differently in different disorder. For example, depression and anxiety, may manifest as an inability to cognitively inhibit negative thoughts and rumination. In oppositional defiant disorder (ODD) and conduct disorder (CD) it may manifest as a deficiency in inhibiting aggressive or antisocial behavior. Finally, attention-deficit/hyperactivity disorder (ADHD) may manifest as difficulty inhibiting behavioral impulses and motor activity. In a similar vein, it is possible that the mechanism linking psychiatric disorders to executive functions is the poor ability to shift attention from ruminative thoughts or excessive worrying, or from one action impulse to more profitable alternatives. According to some longitudinal investigations executive functions affect the development of psychiatric disorders (Kertz, Belden, Tillman, & Luby, 2016; Schaefer et al., 2017; Willoughby, Wyle, & Blair, 2019). However, most research on the relation between executive functioning and psychopathology has been cross-sectional, hence providing limited evidence for the impact of executive functioning on mental health.

Conflict of interest statement: No conflicts declared.
Consequence: mental health problems impair executive functions

As regards the opposite direction of influence, psychiatric disorders may undermine executive functions and leave a “scar” that persists even if the mental health problems remit (Allott, Fisher, Amminger, Goodall, & Hetrick, 2016). Although difficult to identify, preliminary evidence suggests that in adult patients, deficits in executive functions not only persist the following remission from major depression but also worsen with repeated episodes (Semkovska et al., 2019), indicating that depression forecasts an impairment in executive functions. Moreover, individuals with remitted ADHD have the same suboptimal connectivity of brain regions as those with a concurrent ADHD diagnosis (Cortese et al., 2013; Michelini et al., 2019), suggesting permanent damage to, or an alteration of, the brain as a result of the illness. However, such clinical findings do not rule out the possibility that dysfunctions of the executive system predate the disorder and continue after remission. Therefore, long-term prospective community studies are needed to disentangle a “cause” from “consequence,” acknowledging the notion that observational studies cannot provide a strong test of causal relations.

Is the relation due to a common cause?

Besides the possibility that one causes the other, there is also a possibility that executive functions and psychiatric disorders have common origins, producing a spurious relation between them that is difficult to detect in observational research. For example, harsh parenting hampers the development of executive functions (Halse, Steinsbekk, Hammar, Belsky, & Wichstrom, 2019) and is also detrimental to childhood mental health (Scott, 2012). Other likely candidates include common genes (Friedman, du Pont, Corley, & Hewitt, 2018) and chronic stress (Evans & Kim, 2013). Also, recent investigations have indicated that inflammation might be a common source of depression and executive dysfunction (Peters et al., 2019). Hence, there is a need for observational methods that are better suited to adjust for confounding factors.

State-of-the-art

To contrast the above hypotheses within an observational framework, several methodological requirements must be met. First, prospective studies adjusting for prior levels of outcome(s) are needed. Second, only a minority of children with mental health problems receive treatment (Wichstrom, 2004; Wang & Liu, 2020). However, one study reported that executive functions in adolescents do not predict later depression (Schaefer et al., 2017). Since the comorbidity and heterotypic continuity of disorders are substantial (Caspi & Moffitt, 2018), interpreting the above findings is difficult when the associations are not adjusted for comorbidity.

Ideally, to test whether the prospective relations between executive functions and mental health are due to confounding, experimental designs should be employed. However, although studies have shown that training aimed at improving executive functions may also lead to a decline in psychiatric symptoms (Koster, Hoorelbeke, Onraedt, Owens, & Derakshan, 2017; Shuai et al., 2020), it is still unknown whether the decline in symptoms is attributable to improved executive functions or a direct effect of the treatment itself, or whether the findings are generalizable to the population. Therefore, observational studies that control for potential confounding factors might be preferred. Within-person analyses adjust for one class of such confounders, namely, those that do not change over the observational period (e.g., the stable impact of genetics, parental socioeconomic status, and parenting styles; Hamaker, Kuiper, & Grasman, 2015). Moreover, causality operates only on the within-person level: What happens to other individuals in the population (i.e., at the between-person level) cannot be involved in the development of psychiatric symptoms or executive functions in a specific child; only changes that involve that specific child can (i.e., changes at the within-person level). However, estimates from traditional methods (e.g., regressions and cross-lagged panel models) conflate within- and between-person information, thereby preventing causal implications from being drawn. As recently shown by Willoughby et al. (2019), what applies to a group and to each individual child may differ considerably. Their results show that most of the moderately sized bivariate association between

health, socioeconomic status; Gander, Campbell, Flood, & Crowley, 2019; Liddle, Askew, Betts, Hayman, & Alati, 2014) which are also associated with poor executive functions (Evans & Schamberg, 2009; Hughes, Roman, Hart, & Ensor, 2013). Hence, the association between executive functions and mental health may be different in clinical samples compared to the population; thus, community studies are needed. To the best of our knowledge, only two community studies have addressed the possible bidirectionality: Connolly et al. (2014) found that neither executive functions nor depression predicted the other in adolescents, whereas Willoughby et al. (2019) reported bidirectional associations between executive functions and parent-ratings of ADHD symptoms in preschoolers. A few have examined the relation longitudinally but unidirectionally and found that executive functions predict both anxiety and depression (Kertz et al., 2016), as well as externalizing problems (Riggs, Blair, & Greenberg, 2004; Wang & Liu, 2020). However, one study reported that executive functions in adolescents do not predict later depression (Schaefer et al., 2017). Since the comorbidity and heterotypic continuity of disorders are substantial (Caspi & Moffitt, 2018), interpreting the above findings is difficult when the associations are not adjusted for comorbidity.

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executive functions and ADHD in preschoolers is due to time-invariant between-person variation, rendering the within-person association almost absent. Thus, the above considerations and findings highlight the need for investigating the extent to which the relation between executive functions and a wider range of mental health problems persists when all unmeasured time-invariant factors and time-varying comorbid disorders are adjusted for throughout childhood and adolescence in the community.

Furthermore, although executive functions begin to emerge early in life, they continue to develop during the school years and adolescence (Diamond, 2013). The prevalence of different types of psychiatric disorders and symptoms also change from preschool to adolescence (Morken, Viddal, Ranum, & Wichstrom, 2021; Steinsbekk, Ranum, & Wichstrom, 2021). The relation between executive functions and the various mental health problems might thus also change during this period. However, with few exceptions, the relationship has been studied in either young children (e.g. Willoughby et al., 2019) or adolescents (e.g. Han et al., 2016; but see Kertz et al., 2016). Hence, there is a need for studies that examine changes in the relationship over an extended period of time. Executive functions consist of different subcomponents, and working memory, inhibition, and cognitive flexibility or shifting are often highlighted as core executive functions (Diamond, 2013). Most of the current research has focused on the relationship between one or two aspects of executive functions and disorders (e.g. Han et al., 2016; Schaefer et al., 2017) or used a general score of executive functions (Hawkey, Tillman, Lucy, & Barch, 2018). Moreover, studies have typically included one or two types of disorders. Therefore, it remains unknown whether the relation between different aspects of executive functions is different for different disorders – an idea that will be addressed herein.

In summary, although poor executive functioning seems to correlate with psychiatric disorders in children, the nature of this relationship is ambiguous. Hence, two explanations are proposed: (a) reduced executive functioning predicts increased mental health problems; and (b) increased mental health problems forecast reduced executive functioning. Due to a dearth of prospective research, the support for the two hypotheses is equivocal. Moreover, it is unknown whether any of these effects is stronger for some types of disorders than for others and whether such relations have different levels of significance at different ages. To contrast these explanations and assess the effects specific to each disorder, type of executive function, and age, the prospective relations between everyday executive functions and the number of DSM-5-defined symptoms of depressive disorders, anxiety disorders, ADHD, ODD, and CD were examined in a representative community sample of Norwegian children, aged 6–14, who were studied biennially.

**Methods**

**Participants and procedure**

Data from the Trondheim Early Secure Study (TESS; Steinsbekk & Wichstrom, 2018) were utilized. All children born between 2003 and 2004 whose parents lived in Trondheim, Norway were invited to participate. A letter of invitation including the Strength and Difficulties Questionnaire (SDQ) 4–16 version (Goodman, 1997) was sent to their homes, requesting that their parents bring completed SDQs to a scheduled routine health checkup for 4-year-olds. Of the 3,456 parents invited, 97% (n = 3,358) showed up at the clinic. Parents with insufficient proficiency in Norwegian to complete the SDQ were excluded (n = 167). Following procedures prescribed by the Regional Authority for Medical and Health Research Ethics, Mid-Norway, the parents were informed of the study by the clinic’s nurse, who also obtained their written consent to participate. A total of 2,475 parents (82.1%) consented to participate (see flow chart in Figure S1).

To increase statistical power, we oversampled for mental health problems by dividing the sample into four strata according to their SDQ scores (cut-offs: 0, 5–8, 9–11, and 12–40). Then, using a random number generator, defined proportions of children were chosen to participate in the further study. The drawing probabilities increased with increasing SDQ scores, being .37, .48, .70, and .89 in the four strata, respectively. This oversampling was adjusted for in the analyses.

Of the 1,250 parents who were invited to participate, 1,007 (80.2%) were interviewed at the first assessment. After consent was obtained at the well-child clinic the drop-out rate did not differ across the four SDQ strata (χ² = 5.70, df = 3, p = .13).

Measures of executive functions were included from the second assessment (age 6) onwards. Thus, data were used from the second (n = 788, Mage = 6.7 years, SD = .17), third (n = 697, Mage = 8.8 years, SD = .24), fourth (n = 701, Mage = 10.5 years, SD = .15), fifth (n = 661, Mage = 12.5 years, SD = .15), and sixth (n = 625, Mage = 14.35 years, SD = .16) follow-up. The analytical sample comprised children with at least one wave of complete data (n = 874). Descriptive information is presented in Table S1.

Attrition at the first follow-up was greater for those with higher scores on problems with executive functions (OR = 1.01, 95% CI[1.003, 1.02]), but the effect was small (Nagelkerke proxy R² = .016). Attrition at the second follow-up was predicted by more problems with executive function and symptoms of psychiatric disorders, but their combined effect was small (t3. Nagelkerke proxy R² = .080, t4: Nagelkerke proxy R² = .109, t5: Nagelkerke proxy R² = .077).

**Measures**

**Symptoms of psychiatric disorders.** Symptoms of psychiatric disorders at the age of 6 were assessed by interviewing the parents using the Preschool Age Psychiatric Assessment (PAPA; Egger, Ascher, & Angold, 2003) with a structured protocol involving both mandatory and optional follow-up questions. In this context, the interviewer keeps asking questions until they can decide whether a symptom is present or not (see Wichstrom et al., 2012 for an elaborate description of how the instrument was applied). The interrater reliability (ICC) of the symptom counts was assessed through blinded recoding of 9% of the interviews (depression = .90, anxiety = .91, ADHD = .97, ODD/CD = .96). At the ages of 8,
10, 12, and 14, the Child and Adolescent Psychiatric Assessment (CAPA) was used (Angold & Costello, 2000), which is equivalent to the PAPA but is administered separately to both the parent and the child. The ICC of the CAPA was assessed equivalent to the PAPA but is administered separately to both PAPA and CAPA were removed (see Table S2). The correlation which nine items with similar wording to questions used in theutive functions and symptom counts of disorders. To investi-

gate the overlap could produce a spurious relationship between exec-

utive functions was equal across symptom types. As a

test whether executive functions predicted the disorders differently we created a model (Model 3) built upon Model 2 which included the restriction that paths between executive functions and the various symp-

toms were equal across all symptoms. Model 3 proved to be as good a fit as Model 2 ($\chi^2 = 348.74$, $df = 205$, $p < .001$, CFI = .965, TLI = .948, RMSEA = .028, 90% CI[.023, .033], $\Delta \chi^2 = 24.51$, $df = 27$, $p = .60$), indicating that the effect of executive functions was equal across symptom types. As a further test of the equality of the potential impact, we tested whether different disorders had significantly different effects on executive functions. To this end, we compared Model 3 to a fourth model (Model 4) where the paths from the different symptoms to executive functions were set to be equal. The fit of Model 4 was just as good that of Model 3 ($\chi^2 = 354.39$, $df = 208$, $p < .001$, CFI = .964, TLI = .948, RMSEA = .028, 90% CI[.023, .033], $\Delta \chi^2 = 3.15$, $df = 3$, $p = .37$). For parsimonious rea-

sons, a more restricted model is favored over a less restricted one; hence, Model 4 was preferred (see Figure 1). The results revealed that worsening of executive functions predicted increased symptoms across all disorders, ($B = .83$, 95% CI[.37, 1.3]), with average standardized estimates across disorders and time ($\beta = .14$, 95% CI[.04, .19]), whereas an increased number of symptoms for any disorder predicted worsening of executive functions ($B = .01$, 95% CI[.01, .02]) and average standardized esti-
mates across disorders and time for each disorder ($\beta = .06$, 95% CI[.02, .09]). See Table S5 for autoregressive paths of the symptoms.

Because of sex-related differences in the preva-

lence of disorders, it is plausible that results may

Executive functions. Executive functions can be mea-

ured using either performance-based tests or rating measures of everyday behavior. Notably, there is little overlap between the two types of measures (Toplak, West, & Stanovich, 2013), probably because they assess executive functions engaged in very different ways in which different motivations, knowledge, and values, among other factors, are activated (Doebel, 2020). Questionnaire-based measures of executive functions seem to better capture differences between individuals than behavioral tasks, and may therefore be more suitable for studies of individual differences (Dang, King, & Inzlitch, 2020). Thus, we employed the Behavior Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000) in our study. To reduce common method variance, we used the teacher version (BRIEF-T; Gioia, Kenworthy, & Isquith, 2010), com-
pleted by the child’s primary teacher. BRIEF-T contains 86 items tapping inhibition, shifting, initiation, planning, organizing, monitoring, emotional control, and working memory. Questions are rated on a 3-point scale ranging from never (0), sometimes (1), to often (2). BRIEF-T provides a score of general executive functioning and several subscales (Gioia et al., 2010). In the present inquiry, we used the general score and the subscales inhibition, working memory, and shifting, as these represent core executive functions (Diamond, 2013). Favorable ecological validity (Dekker, Ziermans, Spruitj, & Swaab, 2017), satisfactory internal consistency, and convergent validity (Ezepeleta, Gran-

ero, Penelo, de la Osa, & Domenech, 2015) have been reported for BRIEF-T.

Notably, the items in BRIEF-T somewhat overlap with questions used in the PAPA and CAPA to assess ADHD. This overlap could produce a spurious relationship between exec-

utive functions and symptom counts of disorders. To investi-

gate this threat to validity, we created a version of BRIEF-T in which nine items with similar wording to questions used in the PAPA and CAPA were removed (see Table S2). The correlation between this revised version and the original BRIEF-T ranged between .97 and .98 at the various measurement points, and hence the original BRIEF-T was applied.

Analysis plan

All analyses were performed in Mplus 8.5 (Muthén & Muthén, 2017), applying a robust maximum likelihood estimator that does not presuppose multivariate normality. Given the screen stratification, to arrive at corrected population estimates, all analyses were performed using probability weights corre-

sponding to the number of children in the population in a specific stratum divided by the number of participating chil-

dren in that stratum. A full-information maximum likelihood procedure was used to address missing data. To test the hypotheses, a random intercept cross-lagged panel model (RI-CLPM) was used. In this model, within-person variance is separated from between-person variance (Orth, Clark, Donnellan, & Robins, 2020) using children as their own controls and, hence, all time-invariant confounders are adjusted for. The within-person results then reveal whether changes in executive functions from one’s own mean at time-point t predict deviations from one’s own mean in symptoms of disorders at $t + 1$ with changes at t in such symptoms adjusted for. The opposite direction of prediction, that is, executive functioning predicting symptoms of disorders, is simultane-

ously considered.

Results

Table S3 shows the means and standard deviations of the study variables. With few exceptions, symp-

toms of psychiatric disorders were correlated with deficits in executive functions for all disorders and across all time-points (Table S4).

To test whether executive functions predict symp-

toms and vice versa a RI-CLPM was developed. All disorders were included in the same model and the residuals of the psychiatric disorders at each time point were allowed to correlate. In addition, the cross-lagged correlations and paths between symp-

toms and executive functions were allowed to vary over time (Model 1). Model 1 fitted the data well ($\chi^2 = 190.21$, $df = 135$, $p = .001$, CFI = .987, TLI = .970, RMSEA = .022, 90% CI[.014, .028]). To test for age-specific effects of executive function, we tested a model in which both concurrent correlations and paths from executive functions to symptoms were fixed to be identical over time (Model 2). This model did not prove a poorer fit to the data ($\chi^2 = 346.33$, $df = 202$, $p < .001$, CFI = .965, TLI = .948, RMSEA = .029, 90% CI[.023, .034], $\Delta \chi^2 = 4.22$, $df = 40$, $p = .73$). Furthermore, to test whether executive functions predicted the disorders differently we created a model (Model 3) built upon Model 2 which included the restriction that paths between executive functions and the various symp-

toms were equal across all symptoms. Model 3 proved to be as good a fit as Model 2 ($\chi^2 = 348.74$, $df = 205$, $p < .001$, CFI = .965, TLI = .949, RMSEA = .028, 90% CI[.023, .033], $\Delta \chi^2 = 24.51$, $df = 27$, $p = .60$), indicating that the effect of executive functions was equal across symptom types. As a further test of the equality of the potential impact, we tested whether different disorders had significantly different effects on executive functions. To this end, we compared Model 3 to a fourth model (Model 4) where the paths from the different symptoms to executive functions were set to be equal. The fit of Model 4 was just as good that of Model 3 ($\chi^2 = 354.39$, $df = 208$, $p < .001$, CFI = .964, TLI = .948, RMSEA = .028, 90% CI[.023, .033], $\Delta \chi^2 = 3.15$, $df = 3$, $p = .37$). For parsimonious rea-

sons, a more restricted model is favored over a less restricted one; hence, Model 4 was preferred (see Figure 1). The results revealed that worsening of executive functions predicted increased symptoms across all disorders, ($B = .83$, 95% CI[.37, 1.3]), with average standardized estimates across disorders and time ($\beta = .14$, 95% CI[.04, .19]), whereas an increased number of symptoms for any disorder predicted worsening of executive functions ($B = .01$, 95% CI[.01, .02]) and average standardized esti-
mates across disorders and time for each disorder ($\beta = .06$, 95% CI[.02, .09]). See Table S5 for autoregressive paths of the symptoms.

Because of sex-related differences in the preva-

lence of disorders, it is plausible that results may

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differ between the sexes. However, a model where associations could differ between the genders did not prove superior to a model where prospective cross-lagged paths were set to be identical for both sexes ($\Delta \chi^2 = 15.64, df = 13, p = .269$).

To examine whether the components of executive functions differently impacted the symptoms, we fitted a RI-CLPM that included working memory, shifting, inhibition, and symptoms of the four disorders. A model where the impact of these components on prospective symptoms was set to be different did not have a better fit than a model where the effects were set to be similar ($\Delta \chi^2 = 40.41, df = 32, p = .146$). However, in contrast, the effects of symptoms were different on different components of executive functions ($\Delta \chi^2 = 87.76, df = 32, p < .001$). This differential effect was observed at the ages of 12 ($\Delta \chi^2 = 27.94, df = 11, p = .003$) and 14 ($\Delta \chi^2 = 37.44, df = 11, p < .001$), but not at age of 8 or 10. Closer inspection revealed that problems in working memory (age 12: $B = .13, p = .001$, age 14: $B = .13, p = .001$) were more strongly predicted by increased symptoms than problems with inhibition (age 12: $B = .07, p = .077$, age 14: $B = -.01, p = .742$) and shifting (age 14: $B = .06, p = .039$).

**Discussion**

Several studies have shown that poor executive functioning correlates with a range of mental health problems in children. However, whether this is due to poor executive functioning causing impaired mental health or the other way around remains to be explored. In this study, we identified a bidirectional prospective relation between everyday executive functions and symptoms of depression, anxiety, ODD/CD, and ADHD, even when unobserved time-invariant confounders and time-varying comorbidity were adjusted for. Although the effect was similar across disorders and ages, problems with working memory were more strongly predicted by psychiatric symptoms in early adolescence than inhibition and shifting.

**Poor executive functioning as a risk factor for symptoms of psychiatric disorders**

Deficiencies in everyday functions emerged as a risk factor for increased symptoms of the most common forms of psychopathology in children. Our results are in line with previous studies that have used questionnaire-based measures of executive function, reporting impaired executive functions to predict depression, anxiety, and ADHD (Hawkey et al., 2018; Kertz et al., 2016). Studies using performance-based tests, on the other hand, have yielded conflicting results. For instance, some report a prospective relation (Lin & Gau, 2019; Riggs et al., 2004) whereas others do not (Connolly et al., 2014; Schaefer et al., 2017). These discrepancies may be due to methodological differences: different samples, ages, disorders, and types of measurement. These inconsistencies notwithstanding, the abovementioned studies have used methods conflating between- and within-person variance. To the best of our knowledge, the only study disaggregating within- and between-person variation was that by Willoughby et al. (2019), who reported only a minuscule relation between performance-assessed executive functioning and ADHD symptoms in preschoolers. We, however, found a stronger, albeit still modest, relationship with not only ADHD symptoms but also symptoms of all other disorders examined.

Previous cross-sectional studies (Shields, Reardon, Brandes, & Tackett, 2019; Snyder, Friedman, & Hankin, 2019) suggest that executive functions are a general risk factor for developing psychopathology – a $p$ factor. Although the $p$ factor has not been studied directly, our research still contributes to the understanding of the content of the $p$ factor by demonstrating that executive functions predict symptoms of all common childhood disorders examined when all unmeasured time-invariant confounders are controlled for.
Symptoms of psychiatric disorders impairing executive functions

To the best of our knowledge, this is the first study investigating the longitudinal effect of depression, anxiety, ODD/CD, and ADHD on executive functions in children. We found that increased symptoms of all four types of psychiatric disorders forecasted worsening of executive functions over and above the effect of the symptoms of the other disorders. Hence, although the effect was small for each disorder, the combined effect was stronger.

There are several mechanisms that could potentially be responsible for the effect of disorders on executive function. We draw attention to some behavioral, emotional, cognitive, and biological processes that may be operational. First, executive functions seem to thrive when the individual is exposed to circumstances demanding the use of these functions, such as social or learning situations (Diamond, 2013). Symptoms of psychiatric disorders may imply altered behavior that reduces such exposure. For example, depressed children and children with several types of anxiety are likely to withdraw from social contexts, whereas children with symptoms of ODD/CD are more likely to conflict with other children and also with their teachers. Both withdrawal and conflict may result in fewer opportunities to train and develop executive functions. Second, the resource allocation hypothesis (Gotlib & Joormann, 2010) implies that engaging in emotional thoughts depletes cognitive resources so that the executive functions operate suboptimally. If an individual has fewer cognitive resources available for their executive functions to be engaged, then their development is probably delayed, and they will experience an attenuated growth in their executive functions. Third, evidence indicates that psychiatric problems may cause structural and functional changes in the nervous system of a child, which in turn increase the risk of a variety of psychopathologies, a hypothesis known as the scarring hypothesis (Allott et al., 2016). To date, studies in this field have been limited by being cross-sectional and/or being conducted after the onset of the disorder. Therefore, to better test the scarring hypothesis, large-scale studies from an early age that include brain imaging techniques are needed.

Overall, an increased number of psychiatric symptoms did predict reduced working memory at the ages of 12 and 14 to a stronger degree compared to inhibition and shifting. Although the present study was not positioned to examine the reason, we would like to point out that working memory refers to a system with limited capacity operating on information that is no longer perceptually present. To have an optimal working memory, irrelevant information needs to be cleared from the mind (Diamond, 2013). The symptoms themselves (e.g., worry and intrusive thoughts) or their consequences (e.g., hostile thoughts due to conflicts with peers and teachers) may not be easily cleared out and may limit the working memory capacity. More granular and intensive studies may shed light on these mechanisms. There is no ready explanation as to why the effect appears during adolescence and not before. However, at least for emotional disorders, cognitively related symptoms (e.g., low self-esteem feelings of guilt and hopelessness) and disorders (e.g., generalized anxiety) tend to increase during late middle childhood and adolescence (Morken et al., 2021; Steinsbekk et al., 2021), thus possibly taxing working memory to a larger extent than before. Increasing demands are likely placed on working memory in the late middle school and high school, and the above working memory problems may additionally become more evident to teachers (which we used as raters) at these ages than earlier.

Limitations

Although this study possessed several strengths, including the assessment of psychopathology with interviewer-based clinical interviews, long-term follow-up with repeated measures of a large representative community sample, and the use of strong statistical methods to disentangle within- and between-person effects, some limitations do exist. The overlap between performance-based tests and report forms (here: teacher report) assessing executive functions is small or nonexistent (Toplak et al., 2013). Therefore, it remains to be examined whether this study's findings can be replicated when performance-based tests of executive function are used.

Furthermore, this study included only a small share of ethnic minorities (92% of Norwegian origin, see Table S1). Although there is no reason to believe that the results will be fundamentally different in other populations, this remains to be investigated. Finally, although we adjusted for all time-invariant confounding, the relation between executive functioning and psychopathology may still be confounded by time-variant factors such as stressful life events (Pechtel & Pizzagalli, 2011) and sleep duration (Holley, Hill, & Stevenson, 2008; Ranum et al., 2019), to name a few. Hence, strong causal interpretations are precluded.

Conclusions

Cross-sectional studies have repeatedly reported that deficiencies in executive functions occur in a range of psychiatric disorders. In this study, we investigated the longitudinal relations between the two, revealing a bidirectional relationship. Our findings indicate that childhood depression, anxiety, ODD/CD, and ADHD, may to an equal extent (even at subclinical levels) impair executive functioning.
Further, executive function deficiencies may be involved in the etiology of all common forms of psychopathology in school-aged children. Hence, such deficiencies may be one of the specific factors comprising the statistically identified p factor of psychopathology. The prospective relations revealed could not be fully attributed to common time-invariant confounding, supporting the assumption that the two phenomena are both a cause and a consequence of the other.

Supporting information
Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1. Sample descriptives.
Table S2. Items removed from BRIEF-T and their corresponding items in PAPA.

Key points
- Executive functions and psychiatric disorders are correlated in youth.
- Until now it has not been clear whether either one of the constructs predicts the other, or if the correlation is due to confounding.
- The current study shows that both constructs have a reciprocal influence on each other.
- These results highlight the probability that executive functions might be a transdiagnostic risk factor for psychopathology.

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