Homotypic and heterotypic continuity of symptoms of psychiatric disorders from age 4 to 10 years: a dynamic panel model

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Background: Childhood psychiatric disorders and their symptoms evince both within-disorder (homotypic) and between-disorder (heterotypic) continuities. These continuities may be due to earlier symptoms causing later symptoms or, alternatively, that the same (unknown) causes (e.g., genetics) are operating across time. Applying a novel data analytic approach, we disentangle these two explanations. **Methods:** Participants in a Norwegian community study were assessed biennially from 4 to 10 years of age with clinical interviews ($n = 1,042$). Prospective reciprocal relations between symptoms of disorders were analyzed with a dynamic panel model within a structural equation framework, adjusting for all unmeasured time-invariant confounders and time-varying negative life-events. **Results:** Homotypic continuities in symptoms characterized all disorders; strongest for attention-deficit/hyperactivity disorder (ADHD) ($r = .32$–$.62$), moderate for behavioral disorders ($r = .31$–$.48$) and for anxiety and depression ($r = .15$–$.40$), and stronger between 8 and 10 than between 4 and 6 years. Heterotypic continuity also characterized all disorders. A dynamic panel model showed that most continuities were due to unmeasured time-invariant factors rather than effects of earlier symptoms on later symptoms, although symptoms of behavioral disorders, which evinced two-year homotypic continuity ($B = .14$, 95% CI: .04, .25), did influence later symptoms of ADHD ($B = .13$, CI: .03, .23), and earlier ADHD symptoms influenced later anxiety disorder symptoms ($B = .07$, CI: .01, .12). **Conclusions:** Homotypic and heterotypic continuities of symptoms of childhood psychiatric disorders are mostly due to unobserved time-invariant factors. Nonetheless, symptoms of earlier behavioral disorders may affect later symptoms of such disorders and of ADHD, and ADHD may increase the risk of later anxiety. Thus, even if interventions do not alter basic etiological factors, symptom reduction may itself cause later symptom reduction. **Keywords:** Attention-deficit/hyperactivity disorder; anxiety; conduct disorder; continuity; depression; fixed effects; heterotypic; homotypic; longitudinal; life-events; oppositional defiant disorder; prospective; psychiatric disorder; symptoms.

Introduction

Some children with behavior disorders or who are depressed or anxious at one age look similarly at a later age, a case of homotypic or within-disorder continuity. In contrast, some children who are anxious or depressed at one age develop behavioral disorders at a later age (or vice versa), a case of heterotypic or across-disorder continuity (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Why is this so? Many assume, in the case of homotypic continuity, that some disorders are self-perpetuating (e.g., first depression episode leaves 'scars', increasing vulnerability to later depression; Rohde, Lewinsohn, & Seeley, 1994), or even self-aggravating (e.g., attention-deficit/hyperactivity disorder (ADHD) symptoms evoke peer rejection, exacerbating ADHD; Stenseng, Belsky, Skalicka, & Wichstrøm, 2016). In the case of heterotypic continuity, it is often assumed that the presence of one disorder causes additional problems, including additional disorders [e.g., ADHD contributes to later oppositional defiant disorder (ODD) symptoms by evoking harsh parenting (Harvey, Breaux, & Lugo-Candelas, 2016)]. But

at least two alternative, even competing explanations must be entertained (Angold, Costello, & Erkanli, 1999): First, a range of methodological artifacts may be influential; these include common methods (e.g., parents reporting disorder predictor and outcome on multiple occasions), reporting biases, and the fact that some symptoms characterizing multiple disorders (e.g., attention problems define both ADHD and generalized anxiety disorder) may engender spurious cross-time associations. Second, factor-analytic (Lahey et al., 2015) and genetically informed studies (Roberson-Nay et al., 2015) suggest that disorder comorbidity and continuity are to some degree caused by common etiology.

Limitations of observational research designs have, to date, precluded determination of whether homo- and/or heterotypic continuities are due to common causes (methodological or substantive) or disorders affecting each other over time. Even widely used approaches that statistically adjust for many potential confounders can never account for all possible ones. And even genetically informed family studies (e.g., twin studies) that account for genetic confounding of detected continuities are often poorly positioned to discount environmental and common-method effects. In both approaches, then, the risk

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remains of mis-specifying any homotypic or heterotypic continuity in disorders or symptoms.

One econometric method, the dynamic panel model (DPM), holds the promise of illuminating longitudinal and reciprocal relationships within and across disorders and symptoms while adjusting for all time-invariant confounders, such as genetics, overlapping symptoms between disorders and stable aspects of parenting (Arellano & Bond, 1991; Blundell & Bonds, 1998), even without measuring them or knowing what they are. Under the assumption of no confounding due to time-varying factors, DPM thus affords the opportunity, in observational studies, to draw valid causal conclusions. To our knowledge, this method has not been applied in psychology or related fields. However, time-varying factors – those that may have varying values over time – may still affect continuity of symptoms. We therefore take into account what is arguably the most important time-varying confounder, negative life-events, as these consistently predict emotional and behavioral problems (Grant, Compas, Thurm, McMahon, & Gipson, 2004). Thus, after chronicling mean level change from 4 to 10 years in DSM-IV defined symptoms of ADHD, ODD and conduct (CD), depressive, and anxiety disorders, we employ DPM to determine whether observed homotypic and/or heterotypic continuity are due to (a) earlier symptoms influencing later symptoms, (b) time-varying negative life-events, or (c) common unmeasured factors.

Methods
Participants and procedure

The Trondheim Early Secure Study (TESS) comprises members of the 2003 and 2004 birth cohorts in Trondheim, Norway ($N = 3,456$) (Wichstrøm et al., 2012). A letter of invitation together with a screen for emotional and behavioral problems, the Strengths and Difficulties Questionnaire (SDQ) 4–16 version (Goodman, Ford, Simmons, Gatward, & Meltzer, 2000), was sent to the parents of all children in the two birth cohorts. Almost all children appeared at the age-4 routine health check-up ($n = 3,358$). Parents were informed about TESS by the health nurse, using procedures approved by the Regional Committee for Medical and Health Research Ethics Mid-Norway, and written consent was obtained. To increase statistical power, children with emotional or behavioral problems were oversampled; participants were allocated to four strata according to their SDQ scores (cut-offs: 0–4, 5–8, 9–11, and 12–40), and the probability of selection increased with increasing SDQ scores (.37, .48, .70, and .89 in the four strata, respectively). Of the 1,250 children randomly drawn into the Study, 997 were successfully enrolled at Time 1 ($M_{age} = 4.7$ years, $SD = .30$). The sample is described in detail elsewhere (Wichstrøm et al., 2012). At enrollment, an equal rate of boys (49.1%) and girls participated. The informant parents were predominantly female (84.8%), of Norwegian (93.0%) or Western (5.8%) origin, married (56.3%) or cohabitating (33.7%); about half held a college or master degree (53.9%). The drop-out rate after consenting to participate did not vary by SDQ score [($t = .17$, $df = 1$, $p = .86$) or gender ($\chi^2 = 1.02$, $df = 1$, $p = .31$]. Retesting occurred biennially at 6, 8, and 10 years ($M_{age} = 6.7$ years, $SD = .25$; 8.8 years, $SD = .24$; 10.5 years, $SD = .16$, see Figure S1). All participants

with information from at least one wave of data collection comprised the analytical sample ($n = 1,042$). At age 8 and 10, attrition was higher among boys (age 8: OR = 1.37, 95% CI: 1.03, 1.83; age 10: OR = 1.44, CI: 1.08, 1.92). Attrition was also higher at age 8 among those with more ADHD symptoms at age 4 (OR = 1.07, CI: 1.00, 1.14).

Measures

Symptoms of psychiatric disorders were assessed using the Preschool Age Psychiatric Assessment (PAPA) and the Child and Adolescent Psychiatric Assessment (CAPA). The PAPA, administered at ages 4 and 6, is a semistructured psychiatric interview with parents about their children (Egger et al., 2006) to determine diagnoses according to DSM-IV (American Psychiatric Association, 1994). Decisions on all ODD and some CD symptoms rely on normative evaluations of frequency (e.g., 'Often loses temper'). 'Often' was defined post hoc as the highest 10% of the population as determined by frequency counts in the present sample at the specified age (Egger & Angold, 2006). Six CD symptoms deemed age inappropriate for preschoolers were by default not included in the PAPA (e.g., 'Forced someone into sexual activity'). Depressive disorders included major depression and dysthymia, and anxiety disorders included specific phobias, generalized anxiety, separation anxiety, and social anxiety disorders. Symptoms counts of each disorder group were used in all analyses. Due to low prevalence of CD, ODD and CD symptoms were combined to form a behavioral disorder (ODD/CD) score. Blinded raters recoded 9% of the audio-recorded interviews. Inter-rater reliabilities using intraclass correlations ranged from .90 to .97 across disorders. At ages 8 and 10 years, we applied the child and adolescent version of the PAPA, the CAPA (Angold & Costello, 2000), this time also interviewing the children. A symptom was considered present if reported by either respondent, except for ADHD, as only parents were questioned. Inter-rater reliabilities from blinded recodings of 15% of the CAPA interviews ranged from .86 to .90 across disorders.

Negative life-events were assessed by the PAPA at ages 4 and 6. The life-time occurrence of 25 traumatic events (e.g., assault, near-drowning, abuse) and the presence of 14 stressful life-events (e.g., parental divorce, victimization) during the previous 3 months were recorded and summed. At age 8, parents completed a questionnaire covering the same trauma and events; this time we asked whether these had occurred during the previous 2 years.

Statistical analysis

Changes in mean levels of symptoms over time were estimated with linear and quadratic latent growth curves portraying yearly change in the number of symptoms. To examine change in stability over time a Wald test was applied to compare a model in which correlations between symptoms at different time points were fixed to be identical with another in which they were freely estimated.

To adjust for all time-invariant unmeasured confounders, a DPM analysis was conducted. DPM combines autoregressive cross-lagged and fixed or random effects models. The advantage of fixed effects models is that time-varying predictors are adjusted for all unmeasured time-invariant causes of the dependent variable, whereas in random effects models they are not. However, random effects models have the advantage of using both within and between-person information, whereas fixed effects only utilize within-person information. Random effects models thus have more statistical power. Traditionally, DPM is estimated using a first difference method and a generalized methods of moments estimator (GMM; Blundell & Bonds, 1998). However, maximum likelihood estimation does equally well or outperforms GMM in several situations (Moral-Benito, Allison, & Williams, 2017), handles unbalanced

designs well (i.e., with missingness), and offers flexibility in specifying the relationship between model parameters to arrive at a best-fitting model, while effectively handling missing data. Perhaps especially important in the present context is that GMM presupposes that the variables have reached a steady state distribution at t1, whereas a maximum likelihood approach does not, treating the initial values as exogenous. As there are no data to support the claim that *emerging* psychiatric symptoms of preschool children have reached such a steady state, this is arguably not a realistic assumption. For these reasons, we used structural equation modeling (*SEM*; Bollen & Brand, 2010; Moral-Benito et al., 2017). A program for DPM in Stata has recently been written (Williams, Allison, & Moral-Benito, 2016), but here we model this in Mplus 7.31 due to its greater flexibility in certain situations. Our DPM consisted of a traditional autoregressive cross-lagged model in which symptoms of ADHD, ODD/CD, anxiety, and depression at one time point (t) were regressed on these symptoms and negative life-events at t_{-1}, while the error terms of ADHD, ODD/CD, anxiety, depression, and life-events at each time point were allowed to correlate. A time-invariant component was added to the autoregressive cross-lagged model by constructing a latent variable loading on all symptoms at ages 6, 8, and 10, and this latent variable was allowed to correlate with the initial (age-4) values and negative life-events at ages 4, 6, and 8. A more detailed description of random, fixed, and hybrid models, and the model fitting to the present data can be found in the comment to Table S1, and a path diagram is depicted in Figure S2.

In all analyses, missing values were handled using full information maximum likelihood estimation (FIML) under the assumption that data were missing at random, as indicated by the attrition analyses. Due to oversampling, the results were weighted back with a factor corresponding to the number of children in the population in a particular stratum divided by the number of participants in that stratum. As symptoms were expected to be right-skewed, a robust maximum likelihood estimator was used, which also yields robust standard errors.

Results
Continuities and change in symptom levels

Growth curves indicated that mean symptom levels of anxiety disorders increased over time (M_{growth} = .058, 95% CI: .039–.076; see Table 1). Adding a quadratic component to capture the disproportionate increase from 8 to 10 years confirmed this nonlinear development ($M_{quadratic}$ = .022, 95% CI: .012, .033). Symptoms of depression increased linearly over time (M_{growth} = .041, CI: .016, .065). The developments of ADHD and ODD/CD symptoms were curvilinear, increasing from ages 4 to 8, followed by a decline at age 10 (ADHD: $M_{quadratic}$ = −.022, CI: −.036, −.008; ODD/CD: $M_{quadratic}$ = −.034, CI: −.045, −.008). No other change was significant.

Turning to the stability of individual differences, two-year homotypic correlations were high in ADHD, slightly lower for ODD/CD, and modest for anxiety and depression (see diagonal in Table 1). The two-year homotypic correlations for all symptom groups were higher between ages 8 and 10 years than ages 4 and 6 years: ADHD (Wald = 8.47, df = 1, p = .004), ODD/CD (Wald = 5.91, df = 1, p = .02), anxiety disorders (Wald = 9.49, df = 1, p = .002), and

depressive disorders (Wald = 5.09, df = 1, p = .02). All concurrent heterotypic and prospective homotypic correlations were significant, as were practically all prospective heterotypic correlations. Associations between negative life-events and symptoms were small, if not insignificant.

Heterotypic and homotypic continuity: effects of prior symptoms

Details on DPM fitting results are presented in online material (Table S1). The best-fitting and most statistically powerful model was preferred. This had three time-invariant factors [(a) ADHD, (b) ODD/CD, and (c) emotional disorders] and the impacts of these time-invariant factors were allowed to vary over time as were the error terms of all predictors and 10-year outcomes at each time point. The two-year continuities in this model were set to be similar for each lag, and the correlations between the time-invariant factors and negative life-events at ages 4 and 6 were insignificant and therefore set to 0 (i.e., a hybrid model). An Mplus code for this is presented as supplementary text, Appendix S1, available online only.

The coefficients of this model indicate that ODD/CD evinced modest homotypic continuity, with homotypic continuity of ADHD, anxiety, and depression proving to be insignificant (see Table 2). As regards heterotypic continuity, ADHD symptoms increased later anxiety symptoms, whereas ODD/CD increased later ADHD symptoms.

Heterotypic and homotypic continuity: confounding effects of common time-invariant factors

The effects of unmeasured time-invariant factors on homotypic and heterotypic continuity can be seen by comparing the results from the above DPM with those from the autoregressive cross-lagged part of the model. Without adjustment for time-invariant factors, homotypic continuity proved moderately strong for ADHD and ODD/CD (β = .34 and β = .32, respectively, see Table S3 for details), and moderate for anxiety and depressive disorders (both β = .22 and β = .16, respectively). ADHD and ODD/CD seemingly evinced heterotypic continuity with all other disorders (β = .06–.23), except for the ODD/CD → anxiety relationship, whereas anxiety and depression did not, except for depression predicting ODD/CD. The effects of negative life-events were small and inconsistent.

With time-invariant factors taken into consideration, however, the DPM analysis revealed only weak homotypic continuity of ODD/CD. This clearly indicated that the homotypic continuities evident in the autoregressive cross-lagged part of the model could be attributed to time-invariant causes. Such causes could also explain part of the heterotypic continuity

Table 1 Mean number of symptoms and correlations between symptoms of ADHD, behavioral disorders, anxiety disorders, and depressive disorders in the sample at ages 4, 6, 8, and 10 years (n = 1,042)

	Mean	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. ADHD 4 years	1.05	1.84																		
2. ADHD 6 years	1.30	2.24	.41																	
3. ADHD 8 years	1.20	2.40	.38	.59																
4. ADHD 10 years	1.10	2.27	.32	.55	.62															
5. ODD/CD 4 years	0.98	1.43	.36	.23	.20	.19														
6. ODD/CD 6 years	1.18	1.46	.25	.38	.30	.24	.36													
7. ODD/CD 8 years	1.37	1.71	.25	.36	.39	.40	.32	.39												
8. ODD/CD 10 years	0.99	1.45	.20	.24	.30	.39	.31	.32	.48											
9. Anxiety 4 years	0.79	1.11	.33	.16	.16	.17	.27	.19	.13	.14										
10. Anxiety 6 years	0.79	1.24	.23	.34	.27	.20	.10	.34	.17	.16	.20									
11. Anxiety 8 years	0.88	1.24	.31	.36	.47	.33	.19	.26	.37	.25	.28	.33								
12. Anxiety 10 years	1.24	1.49	.19	.29	.39	.43	.21	.19	.25	.33	.21	.24	.40							
13. Depression 4 years	0.80	1.36	.37	.17	.20	.18	.37	.27	.20	.19	.41	.15	.26	.25						
14. Depression 6 years	0.94	1.55	.17	.37	.25	.23	.12	.44	.24	.21	.15	.50	.23	.20	.21					
15. Depression 8 years	0.95	1.57	.17	.22	.30	.28	.16	.23	.28	.18	.17	.20	.50	.28	.25	.28				
16. Depression 10 years	1.07	1.77	.10*	.14	.19	.19	.20	.17	.19	.29	.07*	.13	.22	.45	.15	.19	.29			
17. Life-events 4 years	2.22	1.31	.06*	.07*	.09	.11	.09*	.03*	.10*	.05*	.09	.10	.07*	.03*	.11	.05*	.07*	.00		
18. Life-events 6 years	2.56	1.43	.08	.03*	.07*	.04*	.08	.04*	-.02*	.04*	.04*	.03*	.01*	.00*	.06*	.02*	.00*	.03*	.41	
19. Life-events 8 years	1.01	1.12	.11	.07*	.08	.13	.11	.06*	.08	.09	.08	.11	.09	.09	.10	.18	.08*	.12	.12	.11

ADHD, Attention-deficit/hyperactivity disorder symptoms; ODD/CD, Oppositional defiant disorder symptoms + conduct disorder symptoms.

*p-value >.05.

Table 2 Dynamic panel model: symptoms of disorders and negative life-events predicting symptoms of disorders 2 years later while adjusting for all unmeasured time-invariant factors

Predictors: Symptoms 2 years earlier	ADHD			ODD/CD			Anxiety disorders			Depressive disorders		
	B (95% CI)	β	P-value	B (95% CI)	β	P-value	B (95% CI)	β	P-value	B (95% CI)	β	P-value
ADHD	.09 (−.04,.23)	.08	.17	.03 (−.04,.09)	.05	.34	.07 (.01,.12)	.11	.022	−.01 (−.07,.05)	−.01	.78
ODD/CD	.13 (.03,.23)	.08	.014	.14 (.04,.25)	.17	.008	.00 (−.08,.08)	.00	.94	.02 (−.06,.11)	.02	.58
Anxiety disorders	−.01 (−.14,.11)	−.01	.84	−.02 (−.12,.08)	−.01	.72	.09 (−.04,.24)	.08	.17	−.07 (−.17,.03)	−.04	.16
Depressive disorders	.01 (−.10,.12)	.00	.91	.06 (−.06,.11)	.02	.60	−.06 (−.14,.02)	−.06	.14	.10 (−.01,.21)	.09	.086
Negative life-events	−.02 (−.08,.05)	−.01	.65	.03 (−.03,.08)	.02	.34	−.05 (−.10,.00)	−.05	.038	−.02 (−.07,.04)	−.01	.49

ADHD, Attention-deficit/hyperactivity disorder symptoms; ODD/CD, Oppositional defiant disorder symptoms + conduct disorder symptoms.

from ADHD and ODD/CD to other disorders. However, the heterotypic prospective correlations involving anxiety and depression (see Table 1) should be attributed to them being comorbid with ADHD and ODD/CD; this is because they were for the most part not predictive of other symptoms even in an autoregressive cross-lagged model.

Discussion

It is well established that psychiatric disorders and their symptoms persist during childhood and that the presence of one disorder or its symptoms increase the risk for other future disorders. But exactly why such homotypic and heterotypic continuity characterizes development remains poorly understood due to the fact that observational designs have been unable to disentangle (a) the effect of disorders and symptoms on later disorders and symptoms from (b) effects of common causes for various disorders and symptoms over time. Such time-invariant causes could include genetics, stable parenting and peer relations, response biases, and/or overlapping symptoms across disorders, to name but a few possibilities. Here, we employed a novel analytical approach, the DPM, capable of disentangling the two sources of influence.

Results revealed that most of the observed homotypic continuities in symptoms of psychiatric disorders in childhood are not the result of effects of earlier symptoms on later symptoms, but rather due to unmeasured time-invariant factors, with the same being true of heterotypic continuity of symptoms of ADHD and ODD/CD. Even so, the evidence also indicated that earlier symptoms of behavioral disorders (ODD and CD) did influence later (homotypic) symptoms; that heterotypic continuity of symptoms of anxiety and depression were due to their comorbidity with ADHD and ODD/CD; and that while behavioral disorders predicted future ADHD symptoms, ADHD symptoms forecasted more anxiety disorder symptoms. In other words, even if most homo- and heterotypic continuity resulted from common, time-invariant causes, this was not entirely the case.

Homotypic continuity

Studies using questionnaires or rating scales chronicle moderate to strong homotypic continuity of symptoms of most disorders within the preschool period (Klein, Otto, Fuchs, Reibiger, & von Klitzing, 2015) and from preschool through middle childhood (Kim-Cohen et al., 2005). Such rating scales are not without their limits, given that correspondence with clinical interviews is moderate (Sveen, Berg-Nielsen, Lydersen, & Wichstrøm, 2013), and that they may overestimate continuities due to rater bias. One may wonder, then, whether observed continuity is also evident when structured clinical interviews are used.

Some studies provide preliminary evidence to this effect: When applying a diagnostic interview, continuity in common disorders, except depression, prove evident from age 3 to 6 (Bufferd, Dougherty, Carlson, Rose, & Klein, 2012). This finding is complemented by other work chronicling continuity in depression from preschool through middle childhood (Luby, Gaffrey, Tillman, April, & Belden, 2014). Follow-up studies of clinical or high-risk children assessed during the preschool years also document continuity of ADHD (Lahey et al., 2016) and behavioral disorders (Keenan et al., 2011). Here, we extended this research by showing that homotypic continuity exists among community children, and for all major symptom groups from preschool to middle childhood.

As regards why there is such continuity, our findings indicate that most of the homotypic continuity discerned here – and conceivably in other research – is due to time-invariant factors, producing the commonly chronicled high to modest continuity. Nonetheless, elevated symptom levels of behavioral disorders did seem to cause later symptoms of behavioral disorders, as this cross-time association could not be accounted for by time-invariant factors. Thus, the reasons for the seeming causal effect of earlier symptoms of behavioral disorders on later symptoms of such disorders should be sought among unmeasured, time-varying factors. Negative life-events are one prominent example, which we took into account in our analysis. Conceivably, symptoms of behavioral disorders produce environmental effects that are fed back to the child, thereby increasing the likelihood of later behavioral symptoms. These evoked and even amplifying environmental effects could involve harsh parenting (Patterson, Debaryshe, & Ramsey, 1989), peer deviancy training (Snyder et al., 2005) and/or deteriorating relationships with the teachers (Skalicka, Stenseng, & Wichstrøm, 2015). Future DPM research should thus include possible time-varying factors to illuminate mechanisms responsible for the self-perpetuation or aggravation of behavioral symptoms.

Heterotypic continuity

Prospective studies (Burke, Hipwell, & Loeber, 2010) provide considerable support for theories detailing causal effects linking different disorders and symptoms, such as the 'failure model' stipulating that behavioral disorder symptoms engender conflict and problems with others (e.g., peer rejection, problematic teacher–child relations), which in turn contribute to depression. Indeed, many investigations, including the present inquiry, reveal heterotypic continuities between many, if not most, disorders and their symptoms (Costello et al., 2003). Notably, however, prior research has failed to account for the confounding effects of common etiology between behavioral and emotional disorders.

Results from the DPM analyses presented herein support the view that alleged causal effects from symptoms of behavioral disorders to symptoms of depression are spurious, but that elevated levels of ADHD symptoms may indeed lead to small increases in risk of developing anxiety. This latter finding accords well with results of a twin study of adolescents which revealed that about half of the correlation between attention problems in ADHD and anxiety disorders was attributable to nonshared environmental factors (Michelini, Eley, Gregory, & McAdams, 2015). We extend this observation by showing that it is ADHD that likely influences anxiety, not the other way around. Problems in executive functioning, specifically in shifting and the disregard of task irrelevant information, may be common to both ADHD and anxiety, and has been thought to explain at least part of the association between them (Mogg et al., 2015). However, because such trait-like characteristics were adjusted for in our model, stable executive functioning cannot explain the ADHD effect on anxiety. Thus, potential explanations should be sought (a) among time-varying confounding variables, such as the possibility that peer problems and social exclusion increase ADHD symptomatology (Stenseng et al., 2016) as well as anxiety (Wichstrøm, Belsky, & Berg-Nielsen, 2013), or (b) among time-varying mediating mechanisms, such as ADHD impairing social competence, which in turn may increase the probability of later anxiety (Wichstrøm et al., 2013). Consideration of time-varying factors could also help illuminate mechanisms responsible for the ODD/CD effect on later ADHD, including peer rejection resulting from ODD/CD (Stenseng et al., 2016).

Contrary to research on older children and adolescents, and some studies on young children (e.g., Luby, Belden, & Spitznagel, 2006), negative life-events did not predict psychopathology, except for a slight protective effect on anxiety. Because the latter result was based on a p-value just bordering on significance, it may be a chance finding, especially given the small positive to zero bivariate correlations between negative life-events and anxiety. Perhaps the impact of such events is smaller in the case of young children than later in middle childhood and in adolescence, as indicated by one study (Timmermans, van Lier, & Koot, 2010).

Changing levels of symptoms over time

The increase in rates of depression (particularly among girls) has been found to start in early adolescence, typically around age 13 (Costello et al., 2003). To our knowledge, no study has followed community children with respect to their DSM-defined depression symptoms from preschool to middle childhood. Our results indicate that symptoms of depression may increase slightly before this sharp, adolescent-related increase sets in, at least from age 4 to 10.

Although the prevalence of disorders and mean levels of symptoms of different anxiety disorders may change during childhood [e.g., separation anxiety and specific phobias diminishing, generalized anxiety increasing (Beesdo, Knappe, & Pine, 2009)], it is not yet established whether the net amount of anxiety disorders changes during childhood. Our results indicate that there is an increase in the number of symptoms of anxiety disorders during middle childhood, at least from 8 to 10 years.

Limitations

The present findings should be interpreted in the context of several limitations. First, we analyzed symptom counts, not disorders. Hence, although there is no convincing evidence for most psychopathology being categorical in nature (Haslam, Holland, & Kuppens, 2012), we do not know whether the present findings would generalize to diagnosed disorders. Second, even though we adjusted for all unmeasured time-invariant confounders and time-varying life-events, we cannot rule out the possibility that other time-varying factors, such as those involving parenting or peer relations, caused concurrent symptoms and additional increases in prospective symptoms. The present study was conducted in Norway, a country with particular low rates of childhood psychiatric disorders (Wichstrøm et al., 2012), and generalization to other locales should be performed with this in mind.

Conclusion

Prior research has established that childhood psychiatric disorders and their symptoms persist and are associated with increased risk of other disorders, but the reasons for these continuities have been poorly understood. Thus, we used a novel statistical tool, the DPM, which is able to adjust for all unmeasured time-invariant confounders while examining reciprocal relations. Results indicate that most of the observed continuities are due to unmeasured, time-invariant causes – like genetics or stable parenting practices – exerting their influence throughout childhood rather than earlier symptoms of a disorder contributing to similar symptoms of the same disorder or to symptoms of

a different disorder later on. Nonetheless, early symptoms of ODD/CD do seem to influence later ODD/CD symptoms. Moreover, ODD/CD increased the risk of ADHD symptoms, and ADHD symptoms increase the risk of later anxiety disorder symptoms. Hence, early intervention to reduce ODD/CD symptoms is likely to benefit later ADHD and ODD/CD symptoms, and reduced ADHD symptomatology might reduce later anxiety even if etiological factors are not addressed. Finally, in the methodological approach, we applied (i.e., DPM), both reversed causation between predictors and outcome and time-invariant confounders are adjusted for, thus narrowing the gap between prediction and causation in observational research in psychology and related fields.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Table S1. Results of model fitting procedure.

Table S2. Dynamic panel model: Correlations between time-varying predictors and time-invariant factors. Standardized values.

Table S3. Autoregressive cross-lagged model: symptoms of disorders and negative life-events predicting symptoms of disorders 2 years later.

Figure S1. Flowchart of the recruitment and follow-up.

Figure S2. Path diagram of a fixed effects dynamic panel model with two symptom categories, and life-events as time-varying predictors.

Appendix S1. Mplus Code for a hybrid fixed and random effects dynamic panel model of symptoms of psychiatric disorders with negative life-events as time-varying predictors.

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Key points

- Prior research has consistently demonstrated homotypic (within-disorder) and heterotypic (between-disorder) continuities in children's psychopathology, but the reasons for these continuities are poorly understood.
- Applying a novel data analytic method, we demonstrate that homotypic continuities in symptoms are mostly due to common time-invariant factors, such as genetics or stable parenting practices. Even so, behavioral disorders do increase the risk of later behavioral disorders.

- Heterotypic continuities from ADHD and behavioral disorders were mostly due to common time-invariant factors, but symptoms of behavioral disorders increased later ADHD symptoms, and ADHD predicted more symptoms of anxiety disorders.
- Early intervention to reduce ODD/CD is likely to benefit ADHD symptoms, and reduced ADHD symptomatology may decrease the risk of anxiety. Symptom reduction in behavioral disorders may cause later, and additional, improvements in behavioral disorder symptoms.

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