Explaining the relationship between temperament and symptoms of psychiatric disorders from preschool to middle childhood: hybrid fixed and random effects models of Norwegian and Spanish children

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Background: Four explanations for the concurrent and prospective associations between temperament and psychopathology in children have been suggested: predisposition, complication/scar, common cause/continuity, and pathoplasty/exacerbation. Because the confounding effects of common causes have not been ruled out in prior work, the support for the various explanations is uncertain. **Methods:** Screen-stratified community samples of 4-year olds in Trondheim, Norway ($n = 1,042$), and 3-year olds in Barcelona, Spain ($n = 622$), were assessed biennially for symptoms of attention-deficit/hyperactivity (ADHD), oppositional defiant (ODD), conduct (CD), anxiety, and depressive disorders through interviewer-based psychiatric interviews across four waves of data collection. The parents completed child temperament ratings. The data were analyzed with random and fixed effects regression adjusted for all time-invariant unmeasured confounders (e.g., genetics, common methods bias, item overlap). **Results:** In both Norway and Spain and across ages, negative affect predisposed children to symptoms of all disorders except CD, low effortful control predisposed children to ADHD and ODD-symptoms, and surgency predisposed children to increased ADHD-symptoms. Complication effects were observed in the Spanish children for ADHD-symptoms, which increased surgency and diminished effortful control, and for ODD-symptoms, which decreased surgency. The common cause and pathoplasty/exacerbation explanations were not supported. **Conclusions:** The present results are consistent with the view that temperament plays a causal role in the development of symptoms of psychiatric disorders in children. Because temperament is malleable, interventions targeting the affective, attentional, and behavioral regulatory components of temperament may reduce psychopathology in children. **Keywords:** ADHD; anxiety; conduct disorder; complication; continuity; depression; effortful control; exacerbation; fixed effects; longitudinal; negative affectivity; oppositional defiant disorder; pathoplasty; predisposition; prospective; psychiatric symptoms; random effects; scar; surgency; temperament.

Introduction

Temperament and psychopathology correlate consistently across childhood (De Pauw & Mervielde, 2010; Nigg, 2006). Four etiological explanations for these associations have been proposed (De Bolle, Beyers, De Clercq, & De Fruyt, 2012; Nigg, 2006; Tackett, 2006): (a) *predisposition* (or vulnerability), that negative extremes in certain temperamental traits cause specific types of pathology; (b) *complication* (or 'scar effects'), that psychopathology influences temperament; (c) *common cause,* that third variables are causing both temperament and pathology, thereby producing spurious concurrent and prospective correlations. One variety of the common cause explanation is the *continuity explanation*, i.e., that psychopathologies are extreme versions of common temperamental traits; (d) *pathoplasty/exacerbation*, that temperamental traits alter the course of psychopathology.

Longitudinal studies among children, some of which address personality traits (De Bolle et al., 2012) generally support the predisposition hypothesis, as certain temperamental traits prospectively predict specific mental health problems beyond the contribution of initial mental health problems (De Bolle et al., 2012; Laceulle, Ormel, Vollebergh, van Aken, & Nederhof, 2014). Although the continuity hypothesis is widely supported by studies showing correlational patterns between specific temperamental/personality traits and specific psychopathology (De Bolle et al., 2012; De Pauw & Mervielde, 2010; Nigg, 2006), the common cause explanation has seldom been addressed directly. However, genetically informed studies report that some concurrent (Tackett et al., 2013) and prospective (Gjone & Stevenson, 1997; Goodnight et al., 2016) correlations between temperament and psychopathology are fully or partly explained by common genetics. More scattered and/or less consistent support has been obtained for the complication hypothesis (De Bolle et al., 2012; Shiner, Masten, & Tellegen, 2002).

The pathoplasty/exacerbation explanation has at times been conceptualized as change in temperament predicting change in psychopathology (i.e., a main effect; De Bolle et al., 2012)—a conceptualization that may be seen as an extension of the predisposition hypothesis and has received some empirical support (De Bolle et al., 2012; Laceulle et al., 2014). Nigg (2006) and Tackett (2006), however, portrayed pathoplasty/exacerbation as temperament altering the course or expression of psychopathology *once it occurs* (i.e., an interaction effect). To maintain a clear distinction between predisposition and pathoplasty/exacerbation, we therefore conceptualize pathoplasty/exacerbation as an interaction between temperament and psychopathology that influences later psychopathology.

Importantly, if common causes do exist and are not adjusted for, models examining the predisposition, complication, and pathoplasty/exacerbation explanations will be misspecified, leading to inflated support for these other explanations. The empirical support for the various explanations for temperament-psychopathology associations are therefore less certain than perhaps believed. Although family designs can adjust for the conceivably most important common cause – genetics, genes are obviously not the only possible reason why temperament and psychopathology correlate. Such confounders can be divided into time-*invariant* and time-*varying* factors. Although time-invariant factors, for example, gender, genetics, common methods effects, or item overlap between temperament and psychopathology, do not change over the observational period (although their impact may), time-varying factors may change over time, e.g., parenting or peer-relations. However, seemingly time-varying variables typically involves a time-invariant component as well, e.g., sustained poverty or consistent parenting styles. Notably, fixed effects regression models, which have been developed within econometrics, enable adjustment for all *time-invariant* common causes, whether they are observed or not (Allison, 2009; Bollen & Brand, 2010; Firebaugh, Warner, & Massoglia, 2013). Because the suggested common causes (e.g., genetics, item overlap) are mostly time-invariant we are able to directly test the common cause explanation.

Acknowledging that most results in psychology may not replicate (Aarts et al., 2015), we further employ two large community samples of children in Norway and Spain. By applying clinical diagnostic interviews through four waves of data collection, and adjusting for all time-invariant factors, here we examine four explanations for the prospective relationships between temperament and DSM-IV-defined symptoms of common psychiatric disorders: (a) *predisposition*: temperament→symptoms; (b) *complication:* symptoms→temperament; (c) *common cause*: difference between temperament→symptoms with and without adjustment for all time-invariant common causes; (d) *pathoplasty/exacerbation:* psychopathologyXtemperament→psychopathology.

General methodology
Measures

Temperament: the children's behavior questionnaire-short form (CBQ-SF). The CBQ-SF measures reactive and self-regulative temperament as observed by parents (Putnam, Helbig, Gartstein, Rothbart, & Leerkes, 2014). The CBQ-SF contains 15 scales clustered in three broad dimensions of temperament: negative affectivity (anger-frustration, discomfort, fear, sadness, low soothability), effortful control (attention focusing, inhibitory control, low-intensity pleasure, perceptual sensitivity), and surgency (activity level, high-intensity pleasure, impulsivity, low shyness). Here, we focus on these broad bands.

Symptoms of psychiatric disorders. Interviewer-based clinical interviews were used, implying that mandatory and optional questions were presented until the interviewer had enough information to decide whether a symptom was present. Depressive disorders included major depression and dysthymia, and anxiety disorders comprised specific phobias and generalized, separation, and social anxiety disorders. Symptom counts for each disorder group were used.

Analysis plan

Analyses were conducted with Mplus 7.41 (Norway) and 7.11 (Spain), with $\alpha < .05$ considered significant.

The fixed, random, and hybrid effects models. The four explanations were tested using a fixed/random effects regression approach within a structural equation framework. The analytical model consisted of an autoregressive cross-lagged part and a time-invariant factor part (see Figures S1A–C, available online). In the autoregressive cross-lagged part, attention deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), conduct disorder (CD), anxiety, and depression measured during the last three waves of data collection were regressed on these symptoms 2 years earlier. In addition, symptoms at T3 and T4 were regressed on temperament at T2, symptoms at T2 were regressed on temperament at T1, and temperament at T2 was also regressed on temperament and all symptoms at T1. The error terms of all predictors were allowed to correlate at each time point. The time-invariant factor part consisted of a latent factor loading on symptoms measured at the last three time-points, while being correlated with all time-varying predictors, i.e., symptoms at T1 and temperament at T1 and T2.

To arrive at the best-fitting model, we examined (a) whether random or fixed effects fit the data best. The

Satorra-Bentler scaled $\Delta\chi^2$ test (Satorra, 2000) is a functional equivalent to the Hausmann test (Hausman, 1978) to make such a decision; (b) The number of time-invariant latent factors. Previous factor analytic work indicates that there may be two overarching psychopathology factors (i.e., internalizing and externalizing) or an additional general psychopathology factor loading on all symptoms of disorders in childhood (e.g., Lahey et al., 2015); (c) whether the correlations between residuals of symptoms within each time point should be allowed to vary over time; (d) because the importance of time-invariant factors such as genetics may well change as the child grows, we tested whether a model allowing the effects of time-invariant factors to vary over time fit the data better than a more parsimonious model in which these effects were equivalent at each time point. In all analyses, missing values were handled using full information maximum likelihood estimation (FIML). 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. A robust maximum likelihood estimator was used, which also produces the robust standard errors needed because of the stratified sampling.

Study 1
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). The TESS was approved by the Regional Committee for Medical and Health Research Ethics Mid-Norway, and written consent was obtained. Children were screened with the Strengths and Difficulties Questionnaire (SDQ[4–16]) (Crone, Vogels, Hoekstra, Treffers, & Reijneveld, 2008) at routine health check-ups at age 4 years (n = 3,358), and those with emotional or behavioral problems were oversampled. The 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 for the four strata respectively). Of the 1,250 children randomly drawn into the study, 995 were successfully enrolled at T1 (M_{age} = 4.7 years, SD = .30). The drop-out rate after consent at the well-child clinic did not differ across SDQ score (p = .86) or gender (p = .31). Retesting occurred at 6 years (T2): M_{age} = 6.7 years, SD = .25; 8 years (T3): M_{age} = 8.8, SD = .24; and 10 years (T4): M_{age} = 10.5 years, SD = .16. Overall, 1,042 participants had information from at least one wave of data collection and comprised the analytical sample. At T3 and T4, attrition was higher among boys (T3: OR = 1.37, 95% CI: 1.03–1.83); T4: OR = 1.44, CI: 1.08–1.92). Attrition was also higher at T3 among those who had more ADHD-symptoms at T1 (OR = 1.07, CI: 1.00–1.14). The characteristics of the TESS participants are presented in Table S1.

Measures

The preschool age psychiatric assessment (PAPA) and the child and adolescent psychiatric assessment (CAPA). The PAPA is a semistructured psychiatric interview with parents about their children (Egger et al., 2006) to determine diagnoses according to the Diagnostic and Statistical Manual, 4th Edition (DSM-IV) (American Psychiatric Association, 1994). The PAPA was administered at T1 and T2. At T3 and T4, we applied the child and adolescent version of the PAPA, the CAPA (Angold & Costello, 2000), this time also interviewing the children. A symptom was regarded as present if at least the child or the parent reported it. The exception was ADHD, for which only the parents were questioned. The interrater reliabilities (ICCs) from blinded recodings of 9% and 15% of the PAPA and CAPA interviews, respectively, were as follows: ODD/CD = .96/.88, ADHD = .97/.90, depressive disorders = .90/.87, anxiety disorders = .91/.86.

CBQ-SF. CBQ-SF was applied at ages 4 (T1) and 6 (T2) years. The internal consistencies (α) for T1 and T2, respectively, were: negative affectivity .77/.81, surgency .77/.83, and effortful control .77/.75.

Results

Descriptives of the study variables can be found in Table 1. The model-fitting procedure revealed that a hybrid random and fixed effects model fit the data best while maintaining maximum statistical power. For details, see Table S3. This model consisted of three time-invariant latent factors (i.e., (a) ADHD; (b) ODD and CD; (c) anxiety and depression). The effects of these latent factors on symptoms were equivalent at all ages and proved uncorrelated with temperament but correlated with initial symptoms. The impacts of temperament on later symptoms were equivalent across ages.

Predisposition

The path coefficients from temperament to later symptoms are shown in Table 2. Note that the effects of temperament at T2 on symptoms at T4 are presented as the sum of direct and indirect effects mediated through homotypic and heterotypic continuity of symptoms from T3 to T4. With two exceptions, negative affectivity predisposed children to increased symptom levels at all ages for all disorders except CD. Surgency predicted more symptoms of ADHD and ODD at all ages and more CD-symptoms at T3 and T4. High effortful control forecasted fewer symptoms of ADHD in particular, and marginally

Table 1 Weighted descriptives of study variables

	Study 1: Norway		Study 2: Spain	
	M	SD	M	SD
Negative affectivity – T1	3.98	.57	3.79	.74
Surgency – T1	4.37	.67	4.34	.79
Effortful control – T1	5.02	.42	5.26	.65
Negative affectivity – T2	3.73	.55	3.68	.79
Surgency– T2	4.31	.62	4.25	.82
Effortful control – T2	5.18	.49	5.31	.59
ADHD – T1	1.05	1.84	2.05	2.83
ODD – T1	.67	1.09	1.10	1.49
CD – T1	.30	.62	.22	.60
Anxiety Disorders – T1	.61	1.13	.90	1.23
Depressive Disorders – T1	.80	1.36	.29	.82
ADHD – T2	1.30	2.24	1.75	2.77
ODD – T2	.96	1.21	.94	1.41
CD – T2	.22	.49	.12	.40
Anxiety Disorders – T2	.87	1.51	.73	1.00
Depressive Disorders – T2	.94	1.55	.11	.51
ADHD – T3	1.20	2.40	2.35	3.45
ODD – T3	1.07	1.39	.91	1.41
CD – T3	.30	.60	.11	.37
Anxiety Disorders – T3	.88	1.25	.84	1.32
Depressive Disorders – T3	.95	1.57	.12	.57
ADHD – T4	1.09	2.24	2.50	3.74
ODD – T4	.77	1.15	1.10	1.55
CD – T4	.16	.44	.07	.32
Anxiety Disorders – T4	1.10	1.47	1.16	1.65
Depressive Disorders – T4	1.07	1.77	.29	1.07

ADHD, attention-deficit/hyperactivity disorder; ODD, oppositional defiant disorder; CD, conduct disorder. T1: age 4 in Norway, age 3 in Spain; T2: age 6 in Norway, age 5 in Spain; T3: age 8 in Norway, age 7 in Spain; T1: age 10 in Norway, age 9 in Spain.

and somewhat inconsistently fewer ODD and CD-symptoms.

Complication

Temperamental traits at T2 were not predicted by symptoms at T1 (see Table S4).

Common cause

Because temperamental traits were uncorrelated with the three time-invariant symptom factors in our best-fitting hybrid model, common causes could not explain the observed prospective associations between temperament and symptoms.

Pathoplasty/exacerbation

Multiplicative terms between the mean centered values of (a) temperament at age 4 and symptoms at 4 years predicting symptoms at 6 years, (b) temperament at 6 years and symptoms at 6 years predicting symptoms at 8 years, and (c) temperament at age 6 years and symptoms at 6 years predicting symptoms at 10 years directly and indirectly through heterotypic continuity of symptoms from

age 8 to 10 years were added to the overall model one at a time. No interaction was significant at $p < .028$, and none proved significant when adjusting for the false discovery rate (Benjamini & Hochberg, 1995).

Brief discussion

Of the four explanations that were examined, only predisposition was supported. ADHD was forecasted by high negative affectivity and surgency and low effortful control. A similar pattern was observed for ODD, but the effect of effortful control was somewhat weaker. Both anxiety and depression were predicted by negative affectivity. Notably, as the covariation between symptoms was adjusted for, the effect of temperament was unique for each disorder. Even so, and ruling out the influence of time-invariant common causes between disorders, negative affectivity emerges as a potential shared cause of symptoms of all common psychiatric disorders in Norwegian children.

Study 2
Participants and procedure

The Spanish investigation began in 2010 (T1) with the selection of a random sample of 2,283 children from the census of preschoolers in grade P3 (3-year olds) in Barcelona (Ezpeleta, de la Osa, & Doménech, 2014). A total of 1,341 families agreed to participate, and the parents completed the SDQ enriched with items addressing four additional symptoms of ODD. All children scoring above the 90th percentile on the conduct problems scale of SDQ[3–4] or with a score of 2 – *certainly true* – on any of the eight DSM-IV parent-reported oppositional defiant symptoms and a random sample of 30% of the children with negative screening results were invited to continue ($n = 622$). Retesting took place annually, but for the present inquiry, T1 ($n = 622$; $M_{age} = 3.8$ years, $SD = .33$), T2 ($n = 573$; $M_{age} = 5.7$ years, $SD = .36$), T3 ($n = 497$; $M_{age} = 7.7$ years, $SD = .35$) and T4 ($n = 441$; $M_{age} = 9.7$ years, $SD = .35$) were used. Attrition was not selective according to gender ($p \geq .29$) or the number of symptoms ($p \geq .12$). However, a higher proportion of children from low socioeconomic levels abandoned the study at T2 ($p = .031$) and T4 ($p = .007$). Characteristics of the participants are presented in Table S1. The project was approved by the ethics review committee of the authors' institution (Comissió d'Etica en l'Experimentació Animal i Humana).

Measures

The diagnostic interview for children and adolescents for parents of preschool and young children. The Diagnostic Interview for Children and Adolescents for Parents of Preschool and Young Children (DICA-PPYC; Ezpeleta, de la Osa, Granero,

Table 2 Hybrid fixed and random effects models: symptoms of psychiatric disorders at follow-ups 2, 3, and 4 in Norwegian (left) and Spanish (right) children predicted from temperamental traits at follow-ups 1 and 2

	ADHD	ODD	CD	Anxiety Disorders	Depressive Disorders
T2					
NA – T1	.05 (.11)/.00 (.91)	**.18 (<.001)**/**.13 (.003)**	.03 (.31)/−.02 (.56)	**.14 (.001)**/**.13 (.007)**	**.13 (.001)**/.05 (.27)
SU – T1	**.14 (<.001)**/**.11 (.004)**	**.12 (.001)**/−.03 (.49)	.05 (.23)/.08 (.095)	−.06 (.13)/−.06 (.13)	.01 (.76)/.01 (.77)
EC – T1	**−.21 (<.001)**/**−.08 (.029)**	−.08 (.060)/.08 (.053)	−.03 (.46)/−.09 (.056)	−.05 (.30)/−.05 (.30)	−.03 (.54)/−.03 (.62)
T3					
NA – T2	**.15 (<.001)**/.06 (.12)	**.16 (<.001)**/**.15 (.001)**	.06 (.19)/**.09 (.037)**	**.24 (<.001)**/**.18 (<.001)**	**.22 (<.001)**/**.23 (<.001)**
SU – T2	**.21 (<.001)**/.08 (.067)	**.13 (.002)**/.03 (.51)	**.11 (.007)**/.07 (.17)	.08 (.052)/−.09 (.050)	.02 (.73)/.01 (.81)
EC – T2	**−.20 (<.001)**/**−.16 (<.001)**	**−.09 (.047)**/−.02 (.64)	**−.15 (<.001)**/.01 (.86)	−.03 (.44)/−.03 (.46)	**−.12 (.016)**/−.02 (.64)
T4 total direct and indirect effects					
NA – T2	**.10 (.006)**/**.10 (.036)**	**.13 (.002)**/.08 (.21)	.04 (.28)/.07 (.30)	**.23 (<.001)**/**.23 (<.001)**	**.17 (<.001)**/.12 (.095)
SU – T2	**.17 (<.001)**/**.16 (.004)**	**.09 (.012)**/−.01 (.92)	**.09 (.038)**/.09 (.078)	−.05 (.18)/−.03 (.56)	−.06 (.13)/.02 (.69)
EC – T2	**−.26 (<.001)**/**−.13 (.003)**	**−.11 (.010)**/−.09 (.071)	−.07 (.066)/.03 (.55)	**−.10 (.005)**/−.05 (.23)	−.01 (.73)/−.05 (.41)

ADHD, attention-deficit/hyperactivity disorder; ODD, oppositional defiant disorder; CD, conduct disorder; NA, negative affectivity; SU, Surgency; EC, Effortful control. T1: age 4 in Norway, age 3 in Spain; T2: age 6 in Norway, age 5 in Spain; T3: age 8 in Norway, age 7 in Spain; T1: age 10 in Norway, age 9 in Spain. Standardized regression coefficients, p-values in parentheses; $p < .05$ in bold.

Domenech, & Reich, 2011) is a computerized semistructured diagnostic interview with parents for assessing the most common psychiatric disorders at ages 3–9 years according to the DSM-IV criteria. The interrater reliabilities (ICCs) from blinded recordings of the DICA interview for 12 children were as follows: ODD = .99, CD = .93, ADHD = .87, depressive disorders = .84, and anxiety disorders = .83.

CBQ-SF. CBQ-SF was used at ages 3 years (T1) and 5 years (T2) with satisfactory internal consistencies (α): negative affectivity .83/.85, surgency .81/.84, and effortful control .79/.75.

Results

Descriptive of the study variables can be found in Table 1.

Predisposition

Because a fixed effects model with three time-invariant factors did not converge, the model with two time-invariant factors (behavioral and emotional) was selected (Table S2). Neither the two time-invariant random effects nor the hybrid model identified in Study 1 fit worse than the fixed effects model; thus, the hybrid model was preferred to ease comparisons with Study 1. Next, we tested a model in which the effects of the two time-invariant factors were free to vary over time (M6), and the model fit improved significantly. We therefore retained M6.

Negative affectivity predisposed children to increased symptom levels of anxiety at all ages (Table 2), ODD at contiguous follow-ups (T1–T2, and T2–T3), depression from T2 to T3, and ADHD from T2 to T4 indirectly through T3. Surgency predicted more symptoms of ADHD at all ages except from T2 to T3. Low effortful control predicted symptoms of ADHD at all ages.

Complication

ADHD at T1 predicted higher surgency and lower effortful control at T2, whereas ODD at T1 predicted lower surgency at T2 (Table S4).

Common cause

Because temperamental traits were uncorrelated with the two time-invariant symptom factors, common causes could not explain the observed prospective associations between temperament and symptoms.

Pathoplasty/exacerbation

No interaction was significant at $p < .030$, and none proved significant after adjustment for the false discovery rate.

Brief discussion

Of the four examined explanations, only the predisposition and, to a lesser extent, the complication explanations were supported. Regarding predisposition, negative affectivity consistently predicted anxiety at all time-points and predicted depression and ODD at younger ages. ADHD was predicted by high surgency and low effortful control. There was some evidence of complication effects suggesting that ADHD-symptoms and, to a lesser extent, ODD-symptoms early in life increase surgency and decrease effortful control. Therefore, in the Spanish children, negative affectivity emerged as a potential shared cause of emotional disorders and ODD, whereas surgency and effortful control did so for ADHD. For the Spanish children, the presence of early disruptive psychopathology (ADHD or ODD) may enhance negative affectivity and reduce effortful control.

General discussion

Four explanations (predisposition, complication, common cause, and pathoplasty/exacerbation) of the prospective relations between childhood temperament and symptoms of DSM-IV-defined disorders were examined in large and representative community samples of Norwegian and Spanish children during four waves of data collection. The predisposition explanation was generally supported in both countries, whereas the complication explanation was somewhat supported in Spain.

Predisposition

ADHD. An increased number of ADHD-symptoms was forecasted by low effortful control and higher levels of surgency in both Norway and Spain, whereas inconsistent relations between higher negative affectivity and future ADHD-symptoms were detected in both countries. Lower effortful control and its cognitive counterpart, executive functioning, are associated with ADHD or ADHD-linked pathology (Ullsperger, Nigg, & Nikolas, 2016). Given that surgency contains temperamental facets such as impulsivity and high activity level, which resembles ADHD at face value, surgency is indeed expected to correlate with ADHD-symptoms. In fact, one may question whether low effortful control/executive functioning and high surgency are part of the disorder and not a cause of it, i.e., that overlapping constructs causes the correlations. Although associations between temperament and pathology seem to remain when identical or overlapping items are excluded (Lengua, West, & Sandler, 1998), one cannot be certain that allegedly overlap-free items of temperament and psychopathology are completely semantically and pragmatically separated. Other researchers have found that temperamental

traits share genetic origins with ADHD (Coolidge, Thede, & Young, 2000), and thus may not be etiological *vis-a-vis* the disorder. As our methodological approach ruled out the confounding effects of such alleged common causes, the results presented herein suggest that low effortful control and surgency do play an etiological role in the development of ADHD-symptoms.

Negative emotions such as sadness, anger, and anxiousness are often seen in children with ADHD-symptoms (De Pauw & Mervielde, 2011; Forslund, Brocki, Bohlin, Granqvist, & Eninger, 2016). The present results indicate that when such negativity is dispositional (i.e. temperamental negative affectivity), increased risk of sustained or enhanced ADHD-symptomatology emerge even when prior symptoms of all common disorders and all time-invariant confounders are adjusted for, although no such relationship was found in the Spanish children at the youngest ages. While our findings are consistent with the possibility that low effortful control, and high surgency and negative affectivity play an etiological role in ADHD-symptomatology, our design does not shed light on the causal mechanisms. Although ADHD is highly heritable, some of its variability is attributable to unique environmental effects, possibly involving parents and peers. Children with low effortful control, and high surgency and negative affectivity may evoke negative parenting (Eisenberg, Taylor, Widaman, & Spinrad, 2015), which further exacerbates ADHD-symptoms or hinder the normative age-related decline in some ADHD-symptoms (Sasser, Kalvin, & Bierman, 2016). Moreover, such temperament also increases the risk of peer problems including conflict, rejection, and exclusion (Verlinden et al., 2014), which may increase the risk of ADHD-symptomatology (Stenseng, Belsky, Skalicka, & Wichstrøm, 2016).

ODD and CD. The temperamental predictors of ODD resembled those of ADHD, although less consistently so, especially among the Spanish children. Negative affectivity predicted ODD over the first two data collection waves in both countries. Low effortful control and high surgency were weakly to modestly predictive across ages in Norway, but not in Spain. Given that the symptoms of angry/irritable mood are among the defining ODD characteristics, it should come as no surprise that prior research has found that negative affect predicts ODD (Antunez, de la Osa, Granero, & Ezpeleta, 2016). Because common genetics may explain at least parts of the concurrent association between negative affectivity and ODD (Silberg et al., 2015) concerns could be raised as to whether prospective associations between negative affectivity and ODD actually reflect a predisposition pathway. The finding that negative affectivity proved uncorrelated with the time-invariant causes of ODD-symptoms in both Norway and Spain

strengthens the case for the etiological role of negative affectivity.

With some scattered exceptions, temperament was not predictive of CD-symptoms. Given the high correlations between ODD and CD, this was not expected. However, the low frequency of CD-symptoms in both Norway and Spain may have caused power issues, which would have resulted in high uncertainty and fluctuation of the estimates.

Anxiety and depression. Negative affectivity predicted symptoms of anxiety disorders in both samples, and symptoms of depressive disorders in Norwegians at all ages and in Spaniards at the third assessment. Longitudinal associations between negative affect and anxiety and depression have long been recognized (Laceulle et al., 2014). However, such prospective associations also fit a common cause explanation (see Laceulle et al., 2014). As such, the present results clarify that the effect of negative affectivity cannot be attributed to stable common causes, covariation with other temperamental traits or comorbidities. Note that the coefficients from negative affect to depression in Spain were similar to those in Norway; hence, the low rate of symptoms, combined with the smaller number of participants in Spain, may have led to power problems. We therefore cannot rule out the possibility that negative affectivity may also influence later depression in younger Spanish children.

Low effortful control and executive dysfunction have been implicated in the etiology of anxiety and depression (Laceulle et al., 2014), but these studies have generally not accounted for comorbid conditions or initial level of anxiety and depression (Gulley, Hankin, & Young, 2016; Laceulle et al., 2014). We here extend these studies by demonstrating that negative affectivity indeed may be a predispositional factor, and not fully attributable to heterotypic continuity in anxiety and depression, comorbid conditions, or common time-invariant factors.

Complication

ADHD. In Spain, complication effects were observed for ADHD: an increased number of ADHD-symptoms at age three boosted surgency and decreased effortful control at age 5. These results indicate that early ADHD symptomatology might interfere with the consolidation of the recently emerging reactivity and self-regulation abilities during the preschool years. As high surgency and low effortful control predicted more symptoms of ADHD during middle childhood both reciprocal and cascading processes may be at work, at least in some cultural contexts.

ODD. A higher number of ODD-symptoms at age three predicted lower surgency at age 5 in the Spanish children. This is a surprising result, as previous literature reports associations between high surgency and ODD (De la Osa, Granero, Penelo, Doménech, & Ezpeleta, 2014). However, complication effects indicate that oppositional symptoms prospectively decrease positive emotionality and activity, which are adaptive qualities, although the magnitude found was low. A possible explanation is that oppositional children, because of their conflictual interactions with peers and parents alike, do not find interpersonal relationships very rewarding and thus exhibit less approach behavior, another attribute of surgency.

Complication effects were observed only in the Spanish children. Several cultural differences may explain this. Spanish children begin school at age three, whereas Norwegian children begin when they are six. Norwegian child-rearing practices are generally considered democratic and permissive as opposed to a more authoritarian style in Spain (Wahler & Cerezo, 2005). A high symptom load implies less impairment in young Norwegian children than observed elsewhere (Wichstrøm et al., 2012). Finally, throughout the follow-ups, the Spanish children tended to present a higher number of symptoms of ADHD. In consequence, the potentially higher demands regarding behavioral and attentional regulation put on young Spanish children, in the face of more behavioral symptoms and with more negative social consequences resulting from these symptoms, may imply that symptoms generate long-lasting trait-like (i.e., temperamental) changes, not seen in Norway.

Common cause

In both countries, time-invariant causes of symptoms of psychiatric disorders were not correlated with temperament; thus, they could not explain the prospective associations between symptoms and temperament. This is seemingly at odds with twin studies showing that at least part of the associations between early temperament and later psychopathology is due to shared genetics (Gjone & Stevenson, 1997; Goodnight et al., 2016; Silberg et al., 2015). Furthermore, the heterotypic stability of symptoms was moderate at 2-year intervals but was merely modest over longer time intervals (except for ADHD). This, again, contrasts the literature regarding the continuity of psychopathology during the preschool and middle childhood years (e.g., Kim-Cohen et al., 2005). However, all these continuity studies applied questionnaire-type rating scales or respondent-based interviews completed by the same informant (typically parents), whereas we used interviewer-based clinical interviews in which decisions regarding the presence of a symptom are made by the interviewer and not the interviewee (e.g., the parent). Possibly, therefore, the higher stability obtained in questionnaires taps into the stability of the rater – and the raters' opinion of the child – and not just the stability of the child's problems.

Moreover, when temperament is measured using questionnaires completed by the same informant

that completed the psychopathology measures, heritability estimates based on questionnaires will arguably tap into the heritability of the raters' opinions and the common methods bias. However, when interviewer-based clinical interviews are applied, rater bias and common methods biases are reduced. As a result, less – and potentially more accurate – stability of the psychiatric symptoms is observed. In the face of the modest stability of symptoms (except for ADHD-symptoms), the contribution of time-invariant factors can only be modest. Such time-invariant psychopathology factors are therefore expected to correlate even less with *other* factors (e.g., temperament) than with psychopathology, especially when obtained through different methods. Importantly, the abovementioned prospective twin studies did not adjust for earlier psychopathology. Thus, prospective findings could reflect concurrent correlations at prior time-points and common rater effects. Consequently, the contribution of common genetics to the real prospective relation, net of common methods and overlapping items, between temperament and symptoms of psychiatric disorders may have been inflated in prior research. The present lack of support for the common methods explanation may hence not be deeply discrepant with earlier findings.

Pathoplasty/excerbation

In neither sample did we find support for the pathoplasty/excerbation explanation. Common to all conceptualizations of pathoplasty/excerbation is the notion that temperament and psychopathology have different etiology. In consequence, any effects of psychopathologyXtemperament interactions on the course of psychopathology should not be due to common causes. This implication has not been directly examined in prior work. Moreover, many (e.g., Tackett, 2006) posit that the temperament trait examined should predate the onset of disorder. Evidence suggest that common psychiatric disorders may be present already at 2 years of age (Egger & Angold, 2006), and at subclinical levels even before this time (Ivanova et al., 2010). Given the predominant continuous nature of psychopathology (Haslam, Holland, & Kuppens, 2012) it is difficult, perhaps with the exception of young infants, to document that the temperamental trait in question did indeed predate the disorder or initial symptoms thereof. The predate criterion for pathoplasty/exacerbation is therefore not testable in most settings. Perhaps more useful is to adjust for the effect of covarying and prior psychopathology (i.e., complication effects) when examining the effect of temperament, as was done in this study. Also, according to most conceptualizations (e.g., Nigg, 2006; Tackett, 2006) the temperament trait in question must be present when psychopathology is present (not just predate it) *and* alter its course, which is in essence an interaction.

Others, however, have examined pathoplasty as a main effect, e.g., changes in personality predicting later changes in psychopathology (De Bolle et al., 2012). As we adjusted for common (time-invariant) etiology while examining interaction effects, our results do not compare directly to those of others; however, according to our data, there are no indications that the courses of psychiatric disorder symptoms (i.e., homotypic continuity) vary according to temperament.

Limitations

Despite a range of strengths described earlier in this work, our findings should be interpreted in the context of some limitations. First, although there is much to indicate that psychopathology is continuous in nature (Haslam et al., 2012), our findings do not necessarily translate to psychiatric *disorders*, as these would require judgment of, onset, duration, and impairment. Second, we examined broad temperamental traits that subsume facets that may relate differently, and perhaps more strongly, to psychopathology, such as a fearfulness/behavioral inhibition-anxiety link (Wichstrøm, Belsky, & Berg-Nielsen, 2013) and an attention focusing (or shifting)–ADHD link (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Our approach may therefore have concealed or deflated specific temperament-psychopathology associations. Future work should address this possibility. Third, although we accounted for time-invariant common causes, time-*varying* causes (e.g., negative life events or aspects of parenting that are not consistent over time and not evoked by child temperament or psychiatric symptoms) were not included, and could influence both temperament and psychopathology and produce spurious relationships, also prospectively.

Conclusions

Although prior work has repeatedly tested the four common explanations (i.e., predisposition, complication, common causes, and pathoplasty/exacerbation) for the strong relations between temperament/personality and psychopathology, the validity of findings has been uncertain because researchers have not been able to discount the effect of common causes of temperament/personality and confounding results. The research reported herein demonstrated that temperament effects on later symptoms of psychiatric disorders – across the preschool and early school years – could not be attributed to time-invariant common causes among either the Norwegian or Spanish children. Rather, we found cross-age and cross-national support for the notion that certain temperamental traits predispose children to certain types of psychopathology; negative affect came across as predisposing children to all symptoms of disorders (except CD), low effortful control

may predispose children to ODD-symptoms, and surgery predisposes children to increased rates of ADHD-symptoms. We also found that early symptoms of ADHD and ODD in Spanish children may interfere with the development and maturation of reactivity and self-regulation, and ADHD-symptoms were prospectively associated with high surgery and lower effortful control, whereas ODD-symptoms were associated with lower surgery. Although temperament influences the later development of symptoms, there was no indication that the homotypic continuity of symptoms differed according to temperament (pathoplasty/exacerbation). Thus, overall, our findings support models of childhood psychopathology that place difficult temperament or problems with self-regulation as an early risk factor for psychopathology (Beauchaine & McNulty, 2013; Bridgett, Burt, Edwards, & Deater-Deckard, 2015). Given that temperament is moderately stable and modifiable, interventions targeting children's reactive and self-regulatory capacities may prove successful in reducing their symptoms of psychiatric disorders.

Supporting information

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

Figure S1. (A) Predisposition and complication explanations in a hybrid fixed/random effects model: autoregressive cross-lagged part (normal font) and time-invariant factor part (in bold). (B) Common cause explanation (in bold). (C) Pathoplasty/exacerbation explanation (in bold).

Table S1. Sample characteristics.

Table S2. Correlations between symptoms and temperamental dimensions in Norwegian and Spanish children.

Table S3. Results of model fitting of random, fixed and hybrid models explaining the impact of temperamental traits on later symptoms of psychiatric disorders.

Table S4. Complication model: Effects of symptoms of psychiatric disorders at T1 on temperament at T2 in Norwegian (left) and Spanish (right) children. Partial results from hybrid fixed/random regression analysis (standardized regression coefficients and *p*-values).

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Key Points

- A range of specific relations between children's temperament and psychopathology has been identified. Four explanations for these correlations have been suggested: predisposition, complication, common cause, and pathoplasty/exacerbation. The possibility that temperament and psychopathology are caused by common factors has not been directly tested and thus not adjusted for in prior work examining other explanations.
- We applied a hybrid fixed and random effects regression that could adjust for all unmeasured time-invariant common causes. When analyzing data from two community samples of Norwegian and Spanish children, we demonstrated that in both countries and across ages, negative affect predisposed children to symptoms of all disorders except conduct disorder; low effortful control predisposed children to attention deficit/hyperactivity (ADHD) and oppositional defiant disorder symptoms; and surgery predisposed children to increased ADHD-symptoms. Some complication effects were observed in Spanish children. Common cause and pathoplasty/exacerbation were not supported as explanations.
- Successful early interventions targeting maladaptive forms of temperament, i.e., high negative affect and surgery and low effortful control, may reduce later symptoms of psychiatric disorders.

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