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Mental Disorder During Adolescence: Evidence of Arrested Personality Development

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Abstract

The experience of a mental disorder may affect the development of personality in multiple ways, but empirical evidence regarding psychopathology effects on personality development that persist after remission of the disorder is limited and inconsistent. In the longitudinal cohort TRacking Adolescents' Individual Lives Survey (TRAILS), mental disorders during adolescence were assessed using the Composite International Diagnostic Interview and parent-reported effortful control, fearfulness, and frustration at age 11 and age 19 through the Early Adolescent Temperament Questionnaire. We found that adolescent mental disorders had small effects on personality change. Internalizing disorders predicted increases of fearfulness and frustration but hardly affected effortful control; externalizing disorders were unrelated to frustration and fearfulness but predicted a decrease of effortful control. Whereas fearfulness and frustration partially caught up after disorder remission, virtually all delay in effortful control was still present 2.9 years later, suggesting scarring effects.

Keywords

personality development, psychopathology, mental disorder, personality-psychopathology models, neuroticism, self-control, adolescence

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The relation between personality and psychopathology is highly complex. Robust evidence indicates that this association can be described by multiple models, among which are the spectrum, vulnerability, common cause, and complication models. The importance of each of these models depends on the type of disorder and personality trait (Caspi, Roberts, & Shiner, 2005; Clark, 2005; Durbin & Hicks, 2014; Jeronimus, Kotov, Riese, & Ormel, 2016; Klein, Kotov, & Bufferd, 2011; Krueger & Tackett, 2003; Laceulle, Ormel, Vollebergh, Aken, & Nederhof, 2014; Ormel, Rosmalen, & Farmer, 2004; Ormel et al., 2013; Rettew & McKee, 2005; L. G. Shiner & DeYoung, 2011; Smoller et al., 2019; Tackett, 2006; Widiger & Smith, 2008; Widiger, Verheul, & van den Brink, 1999). In contrast to these models, the evidence regarding the scar model of psychopathology is limited and inconsistent (Ormel et al., 2013; Tackett, 2006; Wichers, Geschwind, van Os, & Peeters, 2010). We targeted possible scarring effects of mental disorders on three traits: effortful control, fearfulness, and frustration. Specifically, we examined whether psychopathology effects on these traits, if any, persist after remission of the disorder episode. Effortful control reflects the capacity to voluntarily regulate behavior

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and attention, fearfulness is defined as worrying and unpleasant affect related to the anticipation of distress, and frustration denotes negative affect related to the interruption of ongoing tasks or blocking of goals (Caspi et al., 2005; Rothbart, Ahadi, & Evans, 2000). Although frustration and fearfulness are often considered major components of negative affectivity, we kept the two traits apart because their correlation changes during development (Rothbart & Putnam, 2002).

It is important to establish whether mental disorder effects on personality persist after disorder remission or personality returns to its premorbid level. This knowledge will expand insight into the burden of mental disorder in terms of maladaptive personality change. In addition, arrested personality development may increase future risk of mental disorder, role impairments, and loss of quality of life (Laceulle et al., 2014; Moffitt et al., 2011; Post, 1992). During adolescence, the lifetime prevalence of mental disorder increases substantially, from about 25% at age 11 to 45% at age 19 (Copeland, Shanahan, Worthman, Angold, & Costello, 2012; Merikangas et al., 2010; Ormel, Raven, & Oldehinkel, 2015). Hence, in theory, adverse personality effects of mental disorders during adolescence may occur in a substantial proportion of the adolescents.

We use the term *personality* throughout this article. Temperament and personality are increasingly considered two different ways of describing the same basic traits; temperament research primarily focused on early-emerging individual differences and personality research on individual differences that appear later during development and continue into adulthood (De Fruyt et al., 2006; L. G. Shiner & DeYoung, 2011). More specifically, Rothbart's often-used temperament traits of negative affectivity and effortful control overlap considerably (but are not identical) with the Five Factor model (FFM) personality constructs of neuroticism and conscientiousness (De Pauw, 2017). Although temperament and personality traits are rather stable, they are not set in stone but change with age and time (Ormel & Rijsdijk, 2000; Roberts & DelVecchio, 2000; Roberts, Walton, & Viechtbauer, 2006).

Models of the Disorder-Personality Relationship

The relationship between personality development and mental health has intrigued researchers for many decades and is considered highly complex (e.g., Clark, 2005; Durbin & Hicks, 2014; Klein et al., 2011; Krueger & Tackett, 2003; Widiger et al., 1999; Widiger et al., 2019). Traditionally, four, not mutually exclusive, basic models were proposed to explain the associations between personality and mental disorders. Which of these models are most appropriate can vary within and between different types of psychopathology and personality. The first basic model, which is known under several names, proposes that personality traits influence the risk (vulnerability model, predisposition model) or the manifestation (pathoplasty model, exacerbation model) of psychopathology. Substantial evidence supports this model. For instance, negative affectivity and low effortful control have been linked, cross-sectionally and prospectively, to many psychiatric conditions, whereas other dimensions, such as extraversion and agreeableness, vary by disorders (e.g., Kotov, Gamez, Schmidt, & Watson, 2010; Ormel et al., 2005; Spinhoven, Penelo, de Rooij, Penninx, & Ormel, 2014; Tackett, 2006).

The second basic model, the spectrum model, proposes that psychopathology and personality traits are manifestations of the same processes and thus lie on the same continua. From the perspective of the spectrum model, the distinction between traits and disorders is, to some extent, tautological (e.g., Ormel, Rosemalen, & Farmer, 2004; Rettew, 2013). For instance, it is virtually impossible to have a depressive disorder and not score high on negative affectivity. The third basic model, the common-cause model, posits that personality and disorders share similar genetic and environmental causes. In practice, the spectrum and common-cause models are difficult to separate and often considered rather similar. As with the first basic model, substantial evidence is consistent with both the common-cause and the spectrum models (e.g., Anttila, 2018; Krueger & Tackett, 2003; Tackett, 2006; Widiger et al., 2019). For instance, recent genome-wide association studies found substantial genetic correlations between the phenotypes of neuroticism and mental disorder (Anttila et al., 2018).

The fourth basic model—the focus of our study—argues that the experience of mental disorder changes an individual's personality. The model is known as the complication model, stagnation model, or scar model. Whereas the complication and stagnation models do not imply lasting personality change after disorder remission, the scar model does; it assumes a change in an individual's personality set points (Ormel, VonKorff, & Riese, 2017). This assumption does not imply that personality traits can never return to premorbid levels long after disorder remission; however, in this respect, it is different from the permanent and irreversible personality changes that can be generated by progressive or chronic brain diseases such as dementia and severe autism (Rettew, 2013; Widiger & Smith, 2008).

Empirical Evidence for the Scar Model

Regarding the fourth type of models, significant evidence exists that mental disorder affects reported personality during the illness episode, which lends support to the complication and stagnation models (e.g., Ormel, Oldehinkel, & Vollebergh, 2004; Tackett, 2006). Much less research has targeted the scar model, and whether the complication/stagnation persists after remission of the mental disorder or normalizes is not well established yet. The limited research on scarring by common mental disorders has yielded inconsistent findings (e.g., Klein et al., 2011; Ormel et al., 2013; Spinhoven et al., 2014; Tackett, 2006). Three factors may account for the inconsistencies: time since remission, developmental period, and rater bias. If the reassessment of personality is too soon after disorder remission, residual stagnation may be erroneously considered scarring because catching up of personality maturation is still ongoing (Ormel, Rosmalen, & Farmer, 2004; Spijker, De Graaf, Oldehinkel, Nolen, & Ormel, 2007). If adolescence is a sensitive developmental period but adulthood not or less so, results will depend on sample age (Ormel, Rosmalen, & Farmer, 2004). Most studies in adults did not find evidence suggesting persistent effects (Ormel, Rosmalen, & Farmer, 2004; Rohde, Lewinsohn, & Seeley, 1990; Shea et al., 1996), whereas the handful of studies in adolescents often did (De Bolle, Beyers, De Clercq, & De Fruyt, 2012; Fanous, Neale, Aggen, & Kendler, 2007; Klimstra, Akse, Hale, Raaijmakers, & Meeus, 2010). Finally, if the same informant reports on both mental disorder and personality, common method bias may cause spurious association: The adult study that did find "scars" used the same informant for both personality and mental disorder exposure (Kendler, Neale, Kessler, Heath, & Eaves, 1993).

Testing Scar Effects: Methodological Considerations

The limited amount of research on the scar model is due to the complex study design required to test whether mental disorder effects on personality occur and persist after disorder remission (Tackett, 2006). The requirements differ depending on whether psychopathology is conceptualized as demarcated categories of psychiatric disorder or as dimensional continua. Study design requirements for a test of scar effects using the categorical approach include assessment of (a) onset and remission of disorder episodes during the index period (in this case, adolescence), (b) an individual's personality both before the onset of disorder and after disorder remission, (c) data on the presence of mental disorder before the index period, and (d) measurement of personality and mental disorder by different informants to exclude common method bias. Study design requirements for a test of psychopathology effects on personality using the dimensional approach include at least four widely spaced assessments of both personality and symptoms to be able to distinguish between state and trait effects (Duncan-Jones, Fergusson, Ormel, & Horwood, 1990; Ormel et al., 2013; Ormel & Schaufeli, 1991; Spinhoven et al., 2014).

Current insights clearly indicate that most psychopathology does not consist of clearly demarcated entities but is dimensional, that cutoffs in psychiatric classifications are fundamentally arbitrary and often not with clear onset and offset times, even if rules from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*; American Psychiatric Association, 1994) are used as instantiated via diagnostic psychiatric interviews (Kotov et al., 2017; Krueger et al., 2018). From this perspective, the dimensional approach seems the preferred approach because the traditional model approach does not accurately represent and capture how psychopathology is structured (Wichers et al., 2010). However, the dimensional approach has serious problems given our core question of whether the experience of mental disorder has scar effects on personality. The most optimal statistical model of the dimensional approach is the multiwave trait-state models with cross-lagged effects between the changing components of personality and psychopathology symptoms (e.g., Duncan-Jones et al., 1990; Ormel & Schaufeli, 1991; Spinhoven et al., 2014). It is not clear how to establish in this model whether cross-lagged effects of psychopathology on personality represent temporary complication/stagnation effects or scar effects that persist after symptom remission. Second, this model requires at least four assessments of both psychopathology and personality by different informants across an extended period of time, a requirement that our study (similar to virtually all others) could not deliver. Hence, we chose to use the categorical approach. It avoids the problems of the dimensional approach and represents a straightforward and conservative test of the scar hypothesis, and our study is one of the very few that meets all requirements for the categorical approach.

Personality Maturation, Stagnation, and Scarring

Up until young adulthood, personality change typically reflects maturation (Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008; Caspi et al., 2005; Durbin et al., 2016; Klimstra, Hale, Raaijmakers, Branje, & Meeus, 2009; Rettew & McKee, 2005; L. G. Shiner & DeYoung, 2011;

Soto, John, Gosling, & Potter, 2008; Van den Akker, Dekovic, Asscher, & Prinzie, 2014): Personality characteristics gradually become more adaptive and appropriate, although there probably is a temporary dip during early adolescence (De Pauw, 2017; Denissen, van Aken, Penke, & Wood, 2013), which has been denoted as the "temporary defiance of the maturity principle" (Van den Akker et al., 2014) and "disruption hypothesis" (Soto & Tackett, 2015).

Maturation is driven by biological, psychological, and social developments, including investments in new social roles and the accompanying necessary changes in goals, cognitions, appraisal, and coping behavior (Durbin et al., 2016; McCrae et al., 2000; Ormel, Riese, & Rosmalen, 2012; Roberts, Wood, & Smith, 2005; Roberts & Mroczek, 2008; R. Shiner & Caspi, 2003). If these new cognitions and behaviors become habitual with time, they may become part of a person's personality. The experience of mental disorder can arrest personality development by causing stagnation of maturation. Stagnation that persists after disorder remission denotes scarring, that is, persistently arrested development. We define scarring as stagnation that persists at least 1 year after disorder remission, an admittedly arbitrary definition.

The Current Study

Using a prospective population-based study of adolescents and young adults, we tested three hypotheses:

Hypothesis 1: The amount of personality stagnation during adolescence is proportional to the number of different mental disorders experienced (referred to as *disorder load*). The rationale for this hypothesis is the abundant evidence that comorbid disorders are more consequential than single, specific disorders (e.g., Copeland, Wolke, Shanahan, & Costello, 2015).

Hypothesis 2: Internalizing (INT) and externalizing (EXT) disorders have somewhat differential impacts on the development of personality traits: EXT disorders mainly predict reduced effortful control, and INT disorders mainly predict more fearfulness and frustration (relative to control subjects). This hypothesis is based on the relatively strong cross-sectional association found between effortful control and EXT disorders and between negative affectivity aspects and INT disorders.

Hypothesis 3: Most stagnation will persist at least until 1 year after remission of the mental disorder. In other words, recency of remission will not or hardly affect personality at age 19 adjusted for personality at age 11.

Method

Sample and procedure

The TRacking Adolescents' Individual Lives Survey (TRAILS) is a prospective cohort study of Dutch adolescents who were followed with biennial or triennial measurements from age 11 onward. The survey's aim is to chart and explain the development of mental health from preadolescence into adulthood. Previous publications extensively described its design, method, response rates, and bias (de Winter et al., 2005; Huisman et al., 2008; Nederhof et al., 2012; Oldehinkel et al., 2015; Ormel, Oldehinkel, et al., 2012). In brief, participants were selected from five municipalities in the north of the Netherlands. Children born between October 1, 1989, and September 30, 1991, were eligible for inclusion if they were not institutionalized and their schools were willing to participate. Over 90% of the schools, enrolling a total of 2,935 eligible children, agreed to participate in the study. Through extended efforts, 76% of these children and their parents consented to participate (Wave 1, N = 2,230; mean age = 11.1 years, SD = 0.6; 50.8% girls). Subsequent data collection waves took place biennially or triennially and had good retention rates (Wave 2: mean age = 13.6 years, SD = 0.59, retention 96%; Wave 3: mean age = 16.1 years, SD = 0.59, retention 81%; Wave 4: mean age = 19.1 years, SD = 0.60, retention 84%). To ease interpretation, we denote Wave 1 as age 11 and Wave 4 as age 19.

Ethical considerations

The study was approved by the Dutch Central Committee on Research Involving Human Subjects (CCMO: NL22114.042.08; http://www.ccmo.nl). All children and their parents provided written informed consent to participate.

Measures

Diagnostic assessment. The presence of lifetime mental disorders was assessed at age 19 using the Composite International Diagnostic Interview (CIDI) 3.0. The CIDI is a structured diagnostic interview that has been used in multiple surveys worldwide to generate diagnoses based on the *DSM–IV* (Kessler & Ustun, 2004). Clinical calibration studies found its assessment of disorders to be generally valid in comparison with blinded clinical reappraisal interviews using the Structured Clinical Interview for *DSM–IV* (SCID; Haro et al., 2006; Kessler et al., 2004; Kessler et al., 2009). CIDI-based prevalence estimates were typically comparable with SCID-based prevalence estimates, except for specific phobias and oppositional

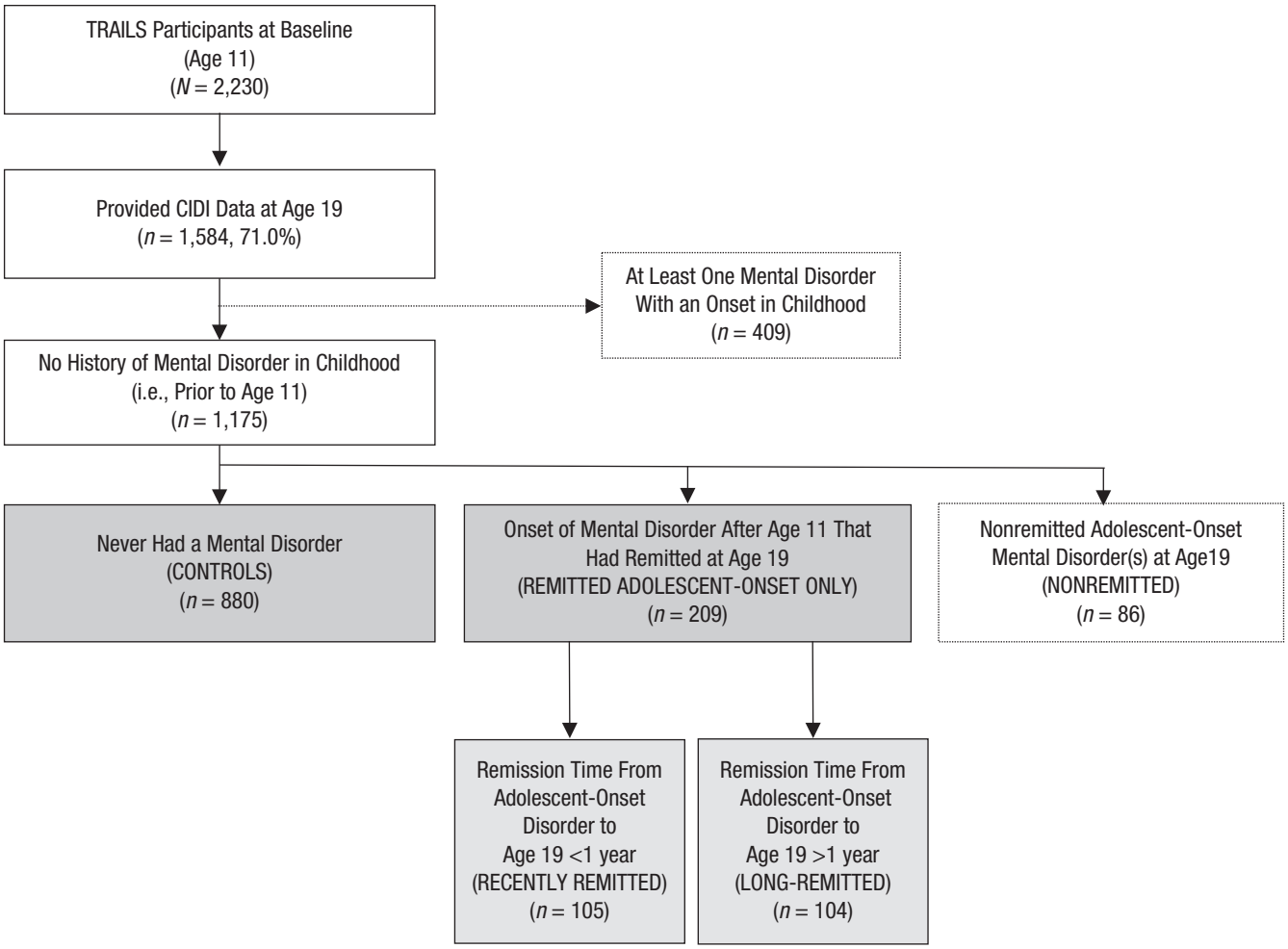

Fig. 1. Flowchart of the study and selection of remitted adolescent-onset-only group.

defiant disorders, for which CIDI estimates were higher. The CIDI 3.0 assesses age of onset of any disorder with a series of questions that have been shown to yield plausible age-of-onset data (Kessler et al., 2005). An important feature of the 3.0 version of the age-of-onset questions is the help of mnemonic aids and the sequence of onset questions, typically starting with the worst episode ever of the index disorder (when did it occur), followed by the most recent episode (when did it occur), and finally targeting the first ever episode and its age of onset.

The assessment included mood disorders (major depressive disorder, dysthymic disorder, and bipolar disorder I and II), anxiety disorders (panic disorder, agoraphobia, social phobia, specific phobia, generalized anxiety disorder, separation anxiety disorder, and obsessive compulsive disorder), behavior disorders (attention-deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder), and substance dependence (alcohol dependence and drug dependence). The interview was administered by trained lay interviewers. Of the total sample, 1,584 adolescents (84.2% of Wave 4 sample, mean age = 19.3 years, 54.0% girls) provided CIDI data (Ormel et al., 2015). CIDI nonresponse was not random but weakly predicted by poor physical health, behavior and substance use problems, male gender, low socioeconomic status (SES), low IQ, and low academic achievement (Nederhof et al., 2012; Ormel et al., 2015).

Exposure groups and adolescent disorder load. On the basis of the number of reported mental disorders, age of onset, and whether individuals were currently in remission (i.e., no mental disorder at age 19), we created two groups (see Fig. 1): (a) control subjects, that is, participants who never had a mental disorder ($n = 880$), and (b) remitted adolescent-onset-only participants (i.e., participants without any childhood disorder who experienced the onset of one or more mental disorders between ages 11 and 19, all of which had remitted by age 19; $n = 209$). Remission was defined as not meeting any diagnostic criteria anymore in the month preceding the CIDI interview. The 209 individuals with remitted adolescent-onset-only

disorders were subdivided into recently remitted (remission less than 1 year before age 19, n = 105) and long-remitted (remission more than 1 year before age 19, n = 104). The mean time since remission in the long-remitted group was 2.9 years (SD = 2.2). Note that mental disorders with an onset in childhood that were still present at age 11 and mental disorders with an onset in adolescence that had not remitted by age 19 were not included in any of the load measures. The reason for this exclusion is that we wanted to be as conservative as possible and therefore targeted remitted adolescent-onset-only disorders.

Adolescent disorder load. We created three adolescent disorder-load variables according to the number of remitted adolescent-onset-only disorders to which participants were exposed in adolescence: (a) any type, (b) only INT disorders, and (c) only EXT disorders.

Personality. At participant ages 11 and 19, we assessed effortful control, fearfulness, and frustration with the Dutch version of the parent-reported Adolescent Temperament Questionnaire–Revised (EATQ-R; Oldehinkel, Hartman, de Winter, Veenstra, & Ormel, 2004; Rothbart et al., 2000). The EATQ-R personality measures were limited to these three because these traits are the most relevant in the context of psychopathology and other EATQ-R scales were not reassessed at age 19. Using parent-reported personality data reduced risk of common informant bias because the CIDI was administered to the adolescent. At both ages, the instrument measuring effortful control (capacity to voluntarily regulate behavior and attention) consisted of nine items, that for fearfulness (worrying and unpleasant affect related to the anticipation of distress) consisted of five items, and that for frustration (negative affect related to interruption of ongoing tasks or goal blocking) consisted of five items as well. For the age-19 reassessment, five of the age-11 items were made more age-appropriate (see Table 1); for instance, the item "My child gets irritated when I will not take her/him someplace s/he wants to go" was replaced by "My child gets irritated quickly." Compared with age-11 scores, age-19 scores were lower for fearfulness and frustration and higher for effortful control, which probably represents maturation (see also Laceulle et al., 2014). The reliabilities as indexed by Cronbach's αs for age-11 and age-19 scores were moderate to good: effortful control (α = .85, .86, respectively), fearfulness (α = .67, .75, respectively), and frustration (α = .71, .81, respectively).

Measurement invariance. We conducted longitudinal measurement invariance tests to ascertain that observed changes actually reflect changes in the personality constructs rather than changes in measurement properties. We tested for configural (factor structure constrained across time), metric (factor loadings constrained across time), and strong invariance (factor loadings and item thresholds constrained across time; Cheung & Rensvold, 2002). Effortful control and frustration showed configural and metric measurement invariance (see Table S1 in the Supplemental Material available online), which suggests that each item had the same factor loading at age 11 and age 19. For fearfulness, we obtained only configural invariance, which indicates that the factor loadings cannot be considered equal across time, but according to fit indices, the loss of fit from configural to metric appeared minimal. Because relatively many fearfulness items were made more age-appropriate for the age-19 assessment (Table 1), it is not entirely unexpected that constraining factor loadings for the items to be exactly equal at both waves resulted in worse fit (Table S1). Not surprisingly given the length and nature of adolescence, the criteria for strong invariance were not met.

Statistical analysis

To address the first hypothesis—amount of personality stagnation is proportional to disorder load—we examined the effect of adolescent disorder load on personality change using regression analyses in the combined sample of control subjects (n = 880) and adolescents with remitted adolescent-onset-only mental disorders (n = 209); in total, there were 1,089 respondents (Fig. 1, Table 2, top). We examined the first hypothesis by testing the effect of total disorder load on age-19 personality while controlling for age-11 personality.

To test the second hypothesis about specific effects of INT load on fearfulness and frustration and of EXT load on effortful control, we repeated the analysis but with INT and EXT load as separate predictors (Table 2, bottom).

To address the third hypothesis—most stagnation will persist (at least until 1 year) after remission of the mental disorder—we performed two analyses. First, we ran a regression analysis with recency of remission (recently remitted, coded 1, n = 105, vs. long-remitted, coded 0, n = 104) and age-11 personality as predictors and age-19 personality as dependent variable (Table 3). Second, we calculated standardized difference scores for each trait, which indicated the age-11-to-age-19 trait change standardized on the age-11-to-age-19 trait change of the control subjects. If catching up does occur, the standardized difference score of long-remitted adolescents should be smaller than that of recently remitted adolescents. In other words, the larger the difference in trait scores between long-remitted and recently remitted relative to the control subjects, the more catching up will have occurred.

All analyses were performed on complete cases. In the text, we present standardized coefficients; unstandardized

Table 1. Items and Factor Loadings of Personality Traits at Age 11 and Age 19 (n = 1,089)

	Effortful control			Fearfulness				Frustration			
				Item wording				Item wording			
	Item	Age 11	Age 19	Age 11	Age 19	Age 11	Age 19	Age 11	Age 19	Age 11	Age 19
1	Finishes homework early	.72	.71	Fears thought of parents dying	—[a]	.64	—	Becomes irritated when criticized		.73	.79
2	Starts immediately with difficult task	.76	.77	Worries about family	Worries a lot[b]	.66	.78	Becomes irritated if parent doesn't go places	Is quickly irritated[b]	.71	.84
3	Difficulty finishing tasks on time	–.76	–.71	Is scared going into dark room	Is uncomfortable in a small room[b]	.69	.41	Hates it when others disagree		.70	.67
4	Finds it easy to concentrate	.74	.64	Worries about getting into trouble		.69	.74	Becomes irritated if he/she needs to stop doing things		.72	.62
5	Postpones tasks to last minute	–.73	–.79	Is scared being home alone	Is afraid of being embarrassed[b]	.54	.73	Is annoyed by things others do		.61	.73
6	Pays attention	.64	.59					—[c]	Is easily upset	—	.70
7	Starts new things without finishing others	–.65	–.69								
8	Does something fun before work	–.52	–.60								
9	Sticks with plans and goals	.50	.54								

Note: Some fear and frustration items were made more age-appropriate at the age-19 reassessment wave. Both the age-11 and age-19 item wordings are presented in this table.
[a]This item was not assessed at age 19. [b]These items were changed to be more age appropriate. [c]This item was not assessed at age 11.

Table 2. Regression Effects of Adolescent Disorder Load (0–3+) on Personality Change (n = 1,089)

	Age-19 Effortful control				Age-19 Fearfulness				Age-19 Frustration			
Predictors	*b*	*SE*	β	95% CI for *b*	*b*	*SE*	β	95% CI for *b*	*b*	*SE*	β	95% CI for *b*
Disorder load based on all mental disorders with onset after Age 11												
Age-11 personality	0.40	0.03	0.40	[0.34, 0.46]***	0.18	0.02	0.27	[0.14, 0.22]***	0.48	0.04	0.38	[0.40, 0.55]***
Sex (0 = F; 1 = M)	–2.44	0.36	–0.20	[–3.15, 1.73]***	–0.95	0.14	–0.20	[–1.23, 0.67]***	–1.34	0.24	–0.17	[–1.81, 0.88]***
Adolescent disorder load	–1.34	0.30	–0.13	[–1.92, 0.76]***	0.52	0.12	0.13	[0.28, 0.75]***	1.01	0.20	0.15	[0.63, 1.40]***
Disorder load separate for INT and EXT disorders with onset after Age 11												
Age-11 personality	0.40	0.03	0.40	[0.34, 0.46]***	0.17	0.02	0.26	[0.13, 0.21]***	0.48	0.04	0.38	[0.40, 0.55]***
Sex (0 = F; 1 = M)	–2.39	0.36	–0.20	[–3.10, 1.68]***	–0.92	0.14	–0.20	[–1.20, 0.64]***	–1.32	0.24	–0.17	[–1.79, 0.86]***
Adolescent INT disorder load	–0.92	0.38	–0.07	[–1.67, 0.19]*	0.85	0.15	0.17	[0.55, 1.14]***	1.17	0.25	0.14	[0.68, 1.66]***
Adolescent EXT disorder load	–2.30	0.61	–0.11	[–3.49, 1.12]***	–0.24	0.24	–0.03	[–0.72, 0.23]	0.65	0.40	0.05	[–0.14, 1.15]

Note: Table contains unstandardized regression coefficients (*b*), standard errors (*SE*), standardized regression coefficients (β), and 95% confidence intervals (CI) of *b* for all regression analyses. All load variables reflect remitted adolescent-onset-only disorders. F = female; M = male; INT = internalizing; EXT = externalizing.
*$p < .05$. **$p < .01$. ***$p < .001$.

Table 3. Regression Effects of Recency of Remission on Personality Change (n = 209)

Outcomes	Predictors	
	Recently remitted adolescent-onset-only (1) vs. long-remitted adolescent-onset-only (0)	Age-11 personality
Age-19 effortful control		
b	–0.80	0.49
SE	0.91	0.08
β	–0.06	0.45
95% CI for *b*	[–2.59, 1.00]	[0.34, 0.64]***
Age-19 fearfulness		
b	0.88	0.17
SE	0.39	0.05
β	0.17	0.24
95% CI for *b*	[0.12, 1.65]*	[0.07, 0.27]***
Age-19 frustration		
b	1.54	0.33
SE	0.65	0.10
β	0.18	0.25
95% CI for *b*	[0.26, 2.82]*	[0.13, 0.53]***

Note: Table contains unstandardized regression coefficients (*b*), standard errors (*SE*), and standardized regression coefficients (β).
*$p < .05$. ***$p < .001$.

coefficients, standard errors, and 95% confidence intervals are presented in the tables. We consider effect sizes (indexed by β) smaller than 0.10 to be irrelevant. Given the possibility of substantial measurement error in the data, we paid attention to trend significance ($.05 < p < .10$) if the effect size exceeded 0.10.

Results

Descriptive statistics

Of all participants that were interviewed with the CIDI (n = 1,584), 30.5% (n = 483) developed at least one adolescent-onset mental disorder, of whom 41.2% (n = 199) experienced two or more disorders. In this CIDI sample of 1,584 adolescents, mood disorders affected 17.4%, whereas 28.0% met criteria for an anxiety disorder, 15.5% for a behavior disorder, and 7.0% for substance dependence. In the CIDI sample without the 409 participants who experienced childhood-onset mental disorder (n = 1,175), 25.1% (n = 295) developed at least one adolescent-onset mental disorder, of whom 36.9% (n = 109) experienced two or more, and 70.8% (n = 209) were disorder free at age 19. Of the 483 adolescents with at least one adolescent-onset disorder, mood disorders affected 14.8% of the sample, whereas 13.5% met criteria for an anxiety disorder, 7.6% for a behavior disorder, and 7.0% for substance dependence; 55.3% were disorder free at age 19.

Table 4 presents the correlations among age-11 and age-19 personality traits and Table 5 raw mean scores. The between-trait correlations indicate that frustration at age 11 is associated with both fearfulness and effortful control but at age 19 is more strongly associated with fearfulness. The across-time correlations indicate substantial rank-order stability but substantial rank-order change as well. At age 19, mean effortful control and frustration scores were hardly different from those at age 11. In contrast, fearfulness scores dropped substantially and showed the least rank-order stability.

Regression analyses: adolescent disorder load

The regression analyses in Table 2 (top) indicate significant effects of age-11 personality and adolescent disorder load on age-19 personality. Tests on linearity indicated that age-11-to-age-19 personality change was proportional to disorder load, which confirms the first hypothesis. Quadratic and logarithmic effects of load did not significantly improve the model with the linear effect (see Table S2 in the Supplemental Material). Figure 2 illustrates the dose-response relationship between

Table 4. Correlations Between Age-11 and Age-19 Personality Traits (n = 1,089)

Variable	Age 11			Age 19		
	Effortful control	Fearfulness	Frustration	Effortful control	Fearfulness	Frustration
Age 11						
Effortful control	—	–.18***	–.32***	.44***	–.09**	–.19***
Fear		—	.29***	–.06	.29***	.17***
Frustration			—	–.23***	.21***	.37***
Age 19						
Effortful control				—	–.19***	–.35***
Fear					—	.57***
Frustration						—

**$p < .01$. ***$p < .001$.

Table 5. Mean Scores for Age-11 and Age-19 Personality Traits

	Mean scores					
	Effortful control		Fearfulness		Frustration	
Sample	Age 11	Age 19	Age 11	Age 19	Age 11	Age 19
Whole CIDI sample (n = 1,584)	29.4 (6.3)	29.2 (6.4)	12.0 (3.6)	7.5 (2.5)	13.8 (3.2)	14.0 (4.2)
Group						
Remitted (n = 209)	30.1 (6.1)	28.7 (6.5)	12.2 (3.8)	7.9 (2.6)	13.8 (3.2)	14.7 (4.3)
Recently remitted (< 1 year) (n = 105)	29.4 (6.4)	28.0 (7.1)	12.5 (3.9)	8.4 (2.7)	14.4 (2.9)	15.7 (4.1)
Long remitted (> 1 year) (n = 104)	30.9 (5.7)	29.4 (6.0)	12.0 (3.7)	7.5 (2.5)	13.3 (3.4)	13.8 (4.2)
Control subjects (n = 880)	30.1 (6.1)	30.2 (6.0)	11.6 (3.5)	7.0 (2.2)	13.5 (3.2)	13.1 (3.9)

Note: Effortful control is based on nine items assessed on a 5-point Likert scale from 1 to 5, resulting in scores between 9 and 45. Age 11 fear and frustration are based on five items (range = 5–25), Age 19 fear is based on four items (range = 5–20), and frustration is based on six items (range = 5–30). CIDI = Composite International Diagnostic Interview

disorder load and decreased effortful control and increased frustration. The relation between disorder load and fearfulness showed a ceiling effect—that is, a strong increase in fearfulness from 0 to 1 disorder load, but no further increase thereafter.

The data supported the second hypothesis on specific effects as well (Table 2, bottom). Adjusted for age-11 personality, EXT load was unrelated to fearfulness and frustration but predicted a decrease of effortful control (β = −0.11). In contrast, INT load was hardly related to effortful control (β = −0.07) but predicted an increase of fearfulness (β = 0.17) and to a lesser extent frustration (β = 0.14).

Recency of remission

The regression analysis with recency of remission and age-11 personality as predictors of age-19 personality

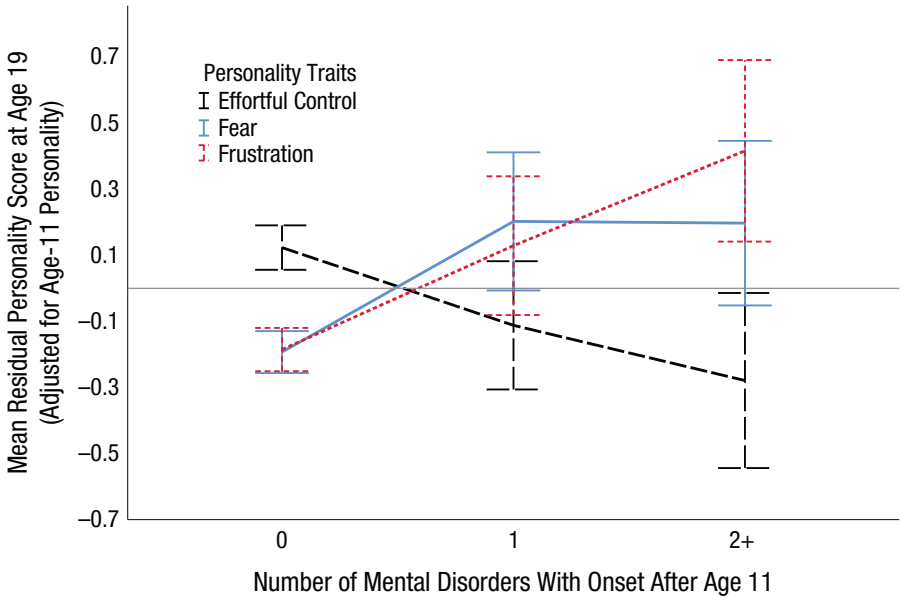

Fig. 2. Relationship between load of remitted adolescent-onset-only mental disorders and personality development (n = 1,089). Personality values of individuals without any disorder come from the 880 control subjects. On the y-axis, the mean residual age-19 personality scores (i.e., age-19 scores adjusted for age-11 personality) are presented. Positive values suggest that the observed age-19 fear and frustration has increased more than predicted by age-11 fear and frustration. Negative values suggest that the observed age-19 effortful control has decreased more than predicted by age-11 effortful control. Both associations suggest stagnation of personality development.

showed significant effects of recency of remission on fearfulness and frustration (β = −0.17, 0.18) but not on effortful control (Table 3), which indicates catching up for fearfulness and frustration but not effortful control. This interpretation is supported by the comparison of the standardized age-11-to-age-19 difference scores between recently remitted and long-remitted adolescents (remember: standardized on the mean trait change in control subjects). These differences amounted to 1.0 as opposed to 0.4 for fearfulness, 1.8 as opposed to 1.0 for frustration, and −1.8 as opposed to −1.6 for effortful control. Thus, regarding fearfulness and frustration, the age-11-to-age-19 trait change in long-remitted adolescents is more similar to the trait change in control subjects than in the age-11-to-age-19 trait change in recently remitted adolescents is. This indicates catching up, probably partially, because in the long-remitted group (a) the standardized difference scores are still larger than zero (0.4, 1.0) and (b) age-19 fearfulness and frustration scores are still higher than those of the control subjects. Regarding effortful control, the standardized across-time differences of long-remitted and recently remitted adolescents hardly differed and were still substantially different from control subjects, which suggests very limited catching up.

Discussion and Conclusion

We examined the influence of adolescent psychopathology on personality development. To be as conservative as possible, we considered only disorder episodes that had an onset in adolescence after the baseline assessment of personality and had remitted before its reassessment. In addition, we excluded all adolescents who experienced an episode in childhood (hence, before the baseline assessment of personality) as well. Collectively, the findings point at some stagnation of maturation in effortful control and to a lesser extent in fearfulness and frustration. EXT disorder load was associated with a decrease in effortful control but did not affect fearfulness and frustration. For INT disorder load, the opposite was found: It predicted less maturation in fearfulness and frustration but hardly affected effortful control. The extent of stagnation increased with each additional adolescent disorder. Most stagnation in effortful control and some of that in fearfulness and frustration was still present long after disorder remission (on average, 2.9 years).

The effects of disorder load on personality were statistically significant but not very strong: The standardized effect sizes of disorder load on personality ranged from 0.13 to 0.15 compared with 0.27 to 0.40 for the effect of age-11 personality on age-19 personality. Although the disorder-load effects were small, we do think they are relevant given the wide-ranging consequences of small to modest differences in childhood self-control for functioning later in life (Moffitt et al., 2011). In addition, it is important to realize that we excluded adolescents with childhood-onset disorders and those with adolescent-onset disorder that had not remitted at age 19 to prevent biased estimates of premorbid and postmorbid personality. Thus, effects of mental disorder on personality may be stronger than observed in our study because adolescent-onset disorders tend be more severe when preceded by a childhood disorder and chronic.

The findings suggest arrested development (scarring), especially in effortful control. Recency of remission predicted age-19 fearfulness and frustration but not effortful control. Contrasting the recently and long-remitted group showed hardly any difference for effortful control, whereas the magnitude of increased fearfulness and frustration was significantly smaller in the long-remitted group than in the recently remitted group. In line, fearfulness and frustration scores of long-remitted individuals deviated less from healthy control subjects than those of recently remitted individuals, whereas the deviation in effortful control scores from healthy control subjects was similar between recently and long-remitted individuals. These findings suggest catching up after disorder remission for fearfulness and frustration, personality aspects related to negative affectivity. However, there is an alternative explanation for the differences between long-remitted and recently remitted individuals: These differences may be due to less severe or shorter episodes in the long-remitted participants than in the recently remitted ones. The CIDI data did not allow us to accurately investigate this alternative. Given the differential findings for the three personality traits, we feel this alternative is less likely.

It is unclear what will happen to the stagnation in personality if remitted individuals stay free of mental disorder for many years. It is possible that the traits will gradually catch up with those of the control subjects in adulthood and not become permanent scars. That said, the average time since remission in the long-remitted group was 2.9 years, which is a considerable length of time already. Therefore, part of the stagnation may be persistent indeed and perhaps even accumulate when new episodes of mental disorder arise (Post, 1992; Wichers et al., 2010).

It is not clear why stagnation in components of negative affectivity is partially recuperating after disorder remission, whereas this hardly seems to occur for effortful control. Two characteristics of negative affectivity may play a role. First, fearfulness is the least stable trait across adolescence in terms of both mean score and

rank order and hence may be more malleable by mental disorder and its remission than effortful control. Second, several studies suggested that at the conceptual as well as the measurement levels, the distance between negative affectivity and INT disorders is smaller than the distance between effortful control and EXT disorder (Jeronimus et al., 2016; Kotov et al., 2010). If negative affectivity is actually more strongly interwoven with mental disorder than effortful control, it may be more sensitive to the presence or absence of mental disorder and its residual symptoms. In other words, the spectrum and common-cause models may be particularly relevant for negative affectivity (especially fearfulness), whereas the scar model holds more value for effortful control.

Are the associations found attributable to third variables or spectrum effects?

The common-cause model postulates shared determinants of psychopathology and personality: Third variables may account for changes in both psychopathology and personality. If true, the association between experiencing a mental disorder and personality change may be spurious. Obvious candidates for common causes are genetic influences (Anttila et al., 2018) and major life events (Jeronimus et al., 2016). Although these factors may explain both the episode itself and personality change during the episode, it is unlikely that they also explain scars. Scars require persistence of stagnation after remission of the episode, and it is hard to understand why the effects of genetic variants and life events on mental health would remit and effects on personality would continue given that personality traits are at least as stable as mental disorders. On the other hand, it is possible that personality returns less easily to its original set point if its development has been stagnated than mental health does.

Similar to the common-cause model, the spectrum model postulates shared determinants of personality and psychopathology. In addition, it conceptualizes both as consisting of dimensional continua on which "mental disorders" represent the more extreme states of normal personality traits. As mentioned earlier, the common-cause and spectrum models are empirically difficult to distinguish (Durbin & Hicks, 2014; Ormel et al., 2013; Rettew, 2013). In terms of the spectrum model, the categorical approach adopted in this study "reconstructs" a complex dimensional space into categories. That approach obviously generates some loss of information, but this reconstruction is unlikely to have biased our findings.

Possible mechanisms?

Adult personality is rather stable (McCrae et al., 2000; Roberts & DelVecchio, 2000; Roberts & Mroczek, 2008). In the set-point model of personality, most personality changes are even considered temporary because homeostatic processes will return trait values to their person-characteristic set point (Ormel et al., 2017; Ormel, Riese, & Rosmalen, 2012). Major role transitions and life events do have some impact on personality, but these effects are typically temporary (Jeronimus, Riese, Sanderman, & Ormel, 2014; Löckenhoff, Terracciano, Patriciu, Eaton, & Costa, 2009; Mroczek & Spiro, 2003; Specht et al., 2014). So why can episodes of mental disorder have persistent effects? In theory, both the mental disorder itself and its determinants, in particular stress, could cause personality stagnation and scarring. The social investment principle provides one mechanism (Roberts, Hill, & Davis, 2017; Roberts, Wood, & Caspi, 2008). According to this principle, personality maturation starts with taking on new roles, and the changes in identity and behavior that these new roles necessitate in turn fuel maturation. Mental disorders may delay, impair, or block these processes because they delay taking on new roles or leave less psychological resources for commitment to these roles.

Another mechanism is sensitization (Post, 1992; Segal, Williams, Teasdale, & Gemar, 1996). The episode and the involved stress may induce a cascade of neurobiological events that have long-lasting consequences, such as altered gene expression. Some empirical support for stress sensitization in the context of depression was found (Wichers et al., 2010). Finally, because individuals influence their environment, the experience of mental disorder may result in persistent environmental change, which might exert an ongoing impact on personality. Given the persistence of the mental disorder effects, especially on effortful control, the observed effects might represent set-point change. As a reliable assessment of an individual's set point requires at least two personality assessments before and two after a potential change agent (Ormel et al., 2017), we could not perform a critical test of set-point change with the current data set.

Strengths and limitations

A strength of our study is that its design allowed investigating the stagnation and scar models of delayed and arrested personality development in detail, which allowed distinctions between stagnation, catching up, and scars. This approach has rarely been done. Our mouse trap to catch stagnation and scarring is better

than previous work because of the large sample size and clearly delineated psychopathology groups based on a standardized diagnostic interview. Another strength is the conservative analytical approach with exclusion of adolescents with childhood-onset and nonremitted disorders to ensure minimally biased personality scores.

Additional strengths include (a) a well-documented sample, which was followed throughout adolescence; (b) consideration of comorbidity as indexed by the number of mental disorders; (c) adjustment for preadolescent personality in the analyses; and (d) use of parent-reported personality measures and respondent-reported information on mental disorders, which reduces the risk of common informant bias (Kotov et al., 2010; Ormel et al., 2013; Tackett, 2006). Another strength is the use of the same personality measure at both preadolescence (age 11) and young adulthood (age 19).

Our study has limitations as well. First, a potentially serious limitation is measurement error in the assessment of personality and mental disorder. It is important to distinguish random and systematic measurement error. The latter produces bias and increases risk of false negatives and false positives; random measurement error produces noise, which weakens observed associations and thus increases the risk of false negatives but rarely false positives. Administering the EATQ-R at age 19 helped comparison across time but had the disadvantage that some items were not age-appropriate anymore. To reduce this limitation, we replaced these items with more age-appropriate versions. Internal consistencies were similar, and age-11 personality remained a strong predictor of age-19 personality. The measurement invariance analyses found configural (equal factor structure) and metric invariance (equal factor loadings) for effortful control and frustration, which indicates that their items had the same factor loading across time. For fearfulness, only configural invariance was found, but the loss of fit from configural to metric appeared minimal. Item thresholds could not be constrained equal across time, which indicates that the "difficulty" of items probably changed across adolescence. Hence, we cannot guarantee that the latent trait constructs remained the same across adolescence; there may have been subtle changes in the meaning of the latent constructs. It is difficult to see whether and if so, in what direction and how these changes might have biased our findings. Most likely, the assessment at age-19 personality was less accurate, with more noise than the administration at age 11.

Although reliability and validity studies of the CIDI reported satisfactory findings (Haro et al., 2006; Kessler et al., 2004; Kessler et al., 2009), a single administration of the CIDI has the drawbacks that the data on lifetime exposure are retrospective and that recall errors may depend on the mental health status during the interview. Furthermore, it is difficult to date onset and remission of episodes. We reduced the possibility of bias by excluding adolescents with nonremitted disorder at age 19, so it is unlikely that recall errors and dating problems caused serious bias that could account for our findings. However, they will have created random measurement error.

Second, it is important to note that most cited studies on the personality-psychopathology models have used personality measures derived from the FFM, that is, neuroticism and conscientiousness. The concepts of negative affectivity (fearfulness and frustration) and effortful control share core components with their FFM counterparts but cannot be set completely equal. Thus, our results may not entirely generalize to the FFM-based measures (De Pauw, 2017).

Third, the nonresponse at follow-up was not random but—weakly—predicted by poor physical health, behavior and substance use problems, male gender, low SES, low IQ, and low academic achievement at baseline (Nederhof et al., 2012; Ormel et al., 2015). Nonresponse bias in psychiatric epidemiological studies tends to be conservative (Kessler & & Ustun, 2004; Merikangas et al., 2010), so the actual associations between mental disorders and personality are probably stronger than the effect sizes reported in this study, especially for the EXT disorders.

Fourth, residual symptoms may have affected the measurement of personality. The CIDI mainly establishes whether diagnostic criteria are met. To reduce respondent burden, no information is collected on individual symptoms of a disorder when it is already clear that the diagnostic threshold of that disorder will not be reached (e.g., because a required core symptom is absent). In other words, subclinical symptom levels are not assessed validly, and hence we could not check whether residual symptoms of childhood-onset or adolescence-onset disorders might have biased the personality measures at, respectively, age 11 and age 19. Because we used parent-reported personality measures, we consider it unlikely that our findings are substantially biased by residual symptoms.

Concluding comments

Our findings indicate that exposure to mental disorder during adolescence causes stagnation in the maturation of effortful control and to a lesser extent, fearfulness and frustration. This finding suggests that in addition to the vulnerability, common-cause, spectrum, and complication models, the stagnation and scar model are needed to account for the association between personality and psychopathology. That said, the need for replication and expansion to dimensional approaches

should be stressed (Wichers et al., 2010). Although our findings did not prove that the relationship is causal (Magidson, Roberts, Collado-Rodriguez, & Lejuez, 2014), they are nonetheless important because the effects of mental disorders on personality, especially on effortful control, seem to persist and may increase future risk of mental disorder, role impairments, and loss of quality of life (Moffitt et al., 2011; Ormel et al., 2005). Given the substantial incidence of episodes of mental disorder during adolescence, up to 30% (Copeland et al., 2012; Merikangas et al., 2010; Ormel et al., 2015), persistent stagnation of personality maturation due to mental disorder may occur in a significant proportion of adolescents.

Transparency

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Author Contributions

J. Ormel conceived and managed the study, developed the argument, and wrote various drafts of the manuscript. D. Raven, A. J. Oldehinkel, and J. Ormel collected the data. A. M. Oerlemans performed the statistical analyses. A. J. Oldehinkel and O. M. Laceulle added important material. All of the authors contributed to the interpretation of the statistical results, provided critical feedback on earlier drafts, and approved the final version of the manuscript for submission.

Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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Open Practices

Under the European Union's General Data Protection Regulation, our data set is considered pseudonymized rather than anonymized and is still regarded as personal data. Given that participants have not given informed consent to have their personal data publicly shared, we are legally and ethically not allowed to publicly post our data set. Data are therefore only available from the authors by request. The conditions imposed on the access to and use of TRAILS data are laid down in the general conditions (https://www.trails.nl/en/hoofdmenu/data/data-use). The complete Open Practices Disclosure for this article can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702619896372.

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Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702619896372

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