REVIEW

The TRacking Adolescents' Individual Lives Survey (TRAILS): Design, Current Status, and Selected Findings

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Objectives: The objectives of this study were as follows: to present a concise overview of the sample, outcomes, determinants, non-response and attrition of the ongoing TRacking Adolescents' Individual Lives Survey (TRAILS), which started in 2001; to summarize a selection of recent findings on continuity, discontinuity, risk, and protective factors of mental health problems; and to document the development of psychopathology during adolescence, focusing on whether the increase of problem behavior often seen in adolescence is a general phenomenon or more prevalent in vulnerable teens, thereby giving rise to diverging developmental pathways. **Method:** The first and second objectives were achieved using descriptive statistics and selective review of previous TRAILS publications; and the third objective by analyzing longitudinal data on internalizing and externalizing problems using Linear Mixed Models (LMM). **Results:** The LMM analyses supported the notion of diverging pathways for rule-breaking behaviors but not for anxiety, depression, or aggression. Overall, rule-breaking (in both genders) and withdrawn/depressed behavior (in girls) increased, whereas aggression and anxious/depressed behavior decreased during adolescence. **Conclusions:** TRAILS has produced a wealth of data and has contributed substantially to our understanding of mental health problems and social development during adolescence. Future waves will expand this database into adulthood. The typical development of problem behaviors in adolescence differs considerably across both problem dimensions and gender. Developmental pathways during adolescence suggest accumulation of risk (i.e., diverging pathways) for rule-breaking behavior. However, those of anxiety, depression and aggression slightly converge, suggesting the influence of counter-forces and changes in risk unrelated to initial problem levels and underlying vulnerability. J. Am. Acad. Child Adolesc. Psychiatry, 2012; 51(10):1020–1036. **Key Words:** developmental pathways, anxiety, depression, aggression, rule-breaking

TRAILS: DESIGN AND CURRENT STATUS

Objectives of TRAILS

The TRacking Adolescents' Individual Lives Survey (TRAILS) consists of two prospective cohort studies, a population-based (N = 2,230) and a clinical (N = 543) cohort. Both follow youth from early adolescence into adulthood. In this article, we focus on the population cohort. The clinical cohort follows children who contacted specialty mental health services before the age of 10 years.[1] [Like the population cohort, it was conducted in the North of the Netherlands and includes measurement waves at regular intervals of 2 to 3 years. However, it began one assessment wave later. It collects largely the same data at the same ages as the population cohort. The clinical cohort has completed three waves: T1 from September 2004 to December 2005, T2 from September 2006 to November 2007, and T3 from September 2009 to February 2011. Hard attrition has


This article will be discussed in an editorial by Drs. James J. Hudziak and Douglas K. Novins in an upcoming issue.

TABLE 1 Specific Outcomes per Research Area

Internalizing problems[a]
Anxiety
Depression
Externalizing problems[a]
Conduct disorder problems
Oppositional defiant behavior
Substance use and dependency
Other mental health problems[a]
Attention problems
Eating problems
Psychotic symptoms
Autism spectrum problems
Social development
Prosocial behavior
Peer relationships
Romantic relationships
Life satisfaction/happiness
Academic achievements
Professional career
Selected physical disorders
COPD
Overweight
Metabolic syndrome
Functional somatic symptoms (including pain)

Note: COPD = chronic obstructive pulmonary disease.
[a]Assessed with symptom checklists; at T4 also by means of a diagnostic interview.

accumulated to 40 respondents (7.4%). T4 has started in mid-2012.] The general aims of TRAILS are as follows: to chronicle the development of mental health from early adolescence into adulthood; to identify the determinants and mechanisms of normal and deviant mental health development; to help evaluate existing interventions and to develop strategies to optimize mental health care for adolescents and young adults; and to describe the impact of mental health problems on academic, professional, and social functioning.

The study focuses on five outcome areas (Table 1). Most publications using TRAILS data to date have addressed internalizing problems, externalizing problems including substance use, or social development. Determinants include a broad variety of biological, psychological, social, and environmental factors (Table 2). One of the strengths of TRAILS is that it has repeatedly measured not only outcomes but also determinants. This makes it easier to test complex longitudinal models of the transactions and interactions that underlie developmental pathways.

Study Design

TRAILS is a population-based prospective cohort study with four assessment waves completed to date. The waves ran from March 2001 to July 2002 (T1, 10–12 years), September 2003 to December 2004 (T2, 12–15 years), September 2005 to August 2007 (T3, 15–17 years), and October 2008 to September 2010 (T4, 18–20 years). The fifth wave (T5) started in early 2012. Tables 1 (outcomes) and 2 (determinants) outline what TRAILS has measured up to the fourth assess-

TABLE 2 Determinants of the Outcomes in Table 1

Biological Markers
Genome-wide genotyping, epigenetics
Metabolic and immunological markers
Autonomic nervous system functioning
HPA axis functioning (cortisol)
Body mass index
Body fat percentage
Physical fitness
Somatic disorders
Developmental history
Perinatal complications
Early childhood behavior
Childhood adversities
Major transitions
Life events and difficulties
Cognitive and psychosocial factors
Information processing capacity (neuropsychological tasks)
Intelligence
Attributional style
Temperament/Personality
Social skills
Family functioning and parenting
Socioeconomic position
Peer status (peer nominations)
Neighborhood characteristics
Lifestyle
Physical activity
Time spending patterns
Religion
Health services use
Parental characteristics
Psychiatric history
Personality
Chronic conditions
Health behavior
Sibling information
Psychiatric history
Personality

Note: Additional blood samples (plasma, serum, cells, isolated DNA) are in stock for future determinations. HPA = hypothalamic–pituitary–adrenal.

ment wave. (Additional information is available at www.trails.nl).

The study area in the northern part of the Netherlands is defined by postal codes and includes about 1,717,000 inhabitants. The area supports a variety of economic activities, including (light) industry, services, educational facilities, and agriculture. Four-fifths of participants were recruited in the three largest towns in the area, and the rest in rural areas.[1,2] The largest ethnic group is the Dutch (89.7%).

Eligibility and Enrollment

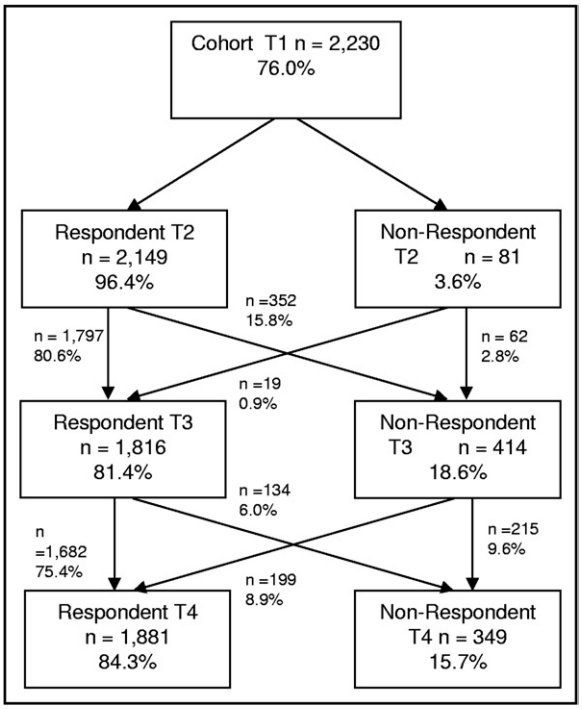
We selected the sample in two stages. First, we obtained demographic information from five municipalities for all inhabitants born between October 1, 1989, and September 30, 1990, in two of the municipalities, and October 1, 1990, and September 30, 1991, in the other three. Primary school participation was a requisite for inclusion. Of the 135 schools we approached, 13 refused to participate, thereby excluding 338 children. Next, we informed parents of eligible children about the study and then invited them to participate. Both parent and child had to give informed consent. After excluding 210 children who were unable to participate because of serious health or language problems, we invited 2,935 eligible children and their parents to enter the study, of whom 2,230 (76.0%; mean age 11.1, SD = 0.56; 50.8% girls) participated in the T1 wave.[1,2]

Response Rates and Retention Strategies

Figure 1 shows wave-specific response rates, which were consistently above 80%. Hard attrition, consisting of participants who requested not to be approached in the future, has accumulated to 171 (7.7%). Of wave-specific nonresponders, about half were willing to participate in the next wave.

At baseline, children of lower socioeconomic background, boys, and children with poor school performance were slightly less likely to participate. Participants and nonparticipants did not differ in emotional and behavior problems.[1,2] Attrition at follow-up was slightly higher in males and participants of nonwestern ethnicity, as well as in participants with divorced parents, low socioeconomic and peer status, low IQ and academic achievement, poor physical health, or externalizing problems. These differences were small but statistically significant.[1,2]

FIGURE 1 Response and attrition of the TRacking Adolescents' Individual Lives Survey (TRAILS) cohort T1 to T4.



TRAILS invested extra efforts in hard-to-recruit and hard-to-retain participants, including additional phone calls, house visits, flexibility in measurements and timing, and a lottery with attractive prizes. These extra efforts were successful both in the short and the long term: after 8 years and four assessment waves, 60% of these "difficult" participants are still in the sample.[3]

WHAT THE STUDY HAS FOUND

At the end of 2011, a total of 132 journal articles had been published or accepted for publication (www.trails.nl). Below we present a selection of findings on (dis)continuity, risk, and resilience factors.

Continuity and Discontinuity of Self-Reported Problems

One way to investigate the persistence of mental health problems is to examine correlations between self-reported emotional and behavioral problem scores assessed at consecutive measurement waves (Table 3). The TRAILS study's detailed assessments, taken multiple times across

TABLE 3 Longitudinal Correlations of Self-Reported Problems Scores Across Adolescence, Showing Both Continuity and Discontinuity of Internalizing, Externalizing, and Total Problems

	Total Problems[a]			Internalizing Problems[a]			Externalizing Problems[a]		
	T1	T2	T3	T1	T2	T3	T1	T2	T3
T2	0.53			0.51			0.47		
T3	0.41	0.61		0.40	0.59		0.34	0.56	
T4	0.37	0.49	0.60	0.35	0.46	0.59	0.31	0.41	0.53

Note: [a]*Youth Self-Report (YSR) scales.*

adolescence, support findings from previous prospective studies,[4-10] showing substantial continuity but also discontinuity of psychopathology. Discontinuities include both dysfunctional youth in early adolescence doing well later in life, and psychopathology arising de novo in adolescence. The longitudinal correlations of self-reported problem scores consistently decline over time, from around 0.56 between adjacent assessments, 2–3 years apart, to 0.35 between early and late adolescence, suggesting persistent trait effects in addition to substantial change effects. The rate of decline during adolescence levels off slightly after early adolescence.

Risk Factors of Mental Disorders

Genotypes. Consistent with evidence on the heritability of common mental disorders, parental psychiatric problems predicted adolescent problem scores.[11-16] The relationship was domain specific when adjusted for comorbidity. Thus, parental internalizing disorders predicted internalizing but not externalizing problems in children, whereas parental externalizing psychopathology predicted externalizing but not internalizing problems.[12,17]

Few genes have been consistently identified as involved in psychopathology. With a few exceptions[18] our efforts, targeting a variety of genes, were unsuccessful as well.[19,20] We had more success in relating genotypes to individual differences in sensitivity to environmental influences. We found that the short allele of *5-HTTLPR*, often related to increased sensitivity to negative experiences, also marks a greater response to positive parenting.[21] Another study showed that the association between negative childhood events and the ability to regulate behavior and attention (effortful control) was stronger in carriers of at least one *BDNF* val66met met or *5-HTTLPR* s' allele than in non-carriers.[22] We also showed that the combined *COMT* met/met-low *MAOA* genotype was associated with stronger cortisol responses to a social stress test.[23,24] Overall, these findings provide some support for the hypothesis of genetically driven individual differences in plasticity.

Temperament. TRAILS has consistently demonstrated the central position of temperament in the pathways leading from genetic risk and environmental exposure to poor mental health.[23-38]

Early adolescent temperament traits mediated one-third of the association between parental psychiatric problems and psychopathology in early adolescence.[11,12] The association of temperament with psychopathology varied across trait and outcome. Frustration acted as a general risk factor, predicting nearly every dimension of psychopathology during adolescence. Fearfulness and effortful control were more dimension specific risk factors of internalizing and externalizing problems, respectively.[11,12] In addition to these main effects, poor effortful control moderated the risk of both internalizing and externalizing problems associated with high negative emotionality.[33]

Childhood Adversity. In TRAILS, early childhood adversity has been related to a variety of mental health problems.[39-42] For example, pregnancy and delivery problems predicted externalizing behavior problems,[39] and maternal postpartum depression was associated with internalizing problems.[43] Maternal smoking during pregnancy was associated with behavioral problems and substance use, but the association disappeared after adjustment for confounders such as parental psychopathology and child temperament.[41]

Stressful life events in adolescence also increased the probability of externalizing and internalizing problems.[38,44-46] The incidence of life events was not random, but depended on person and environmental characteristics.[44] Both person-

independent and person-dependent stressors predicted mental health problems. Higher rates of environment-related life stressors among adolescents of low socioeconomic position (SEP) partially explained socioeconomic inequalities in mental health.[45]

Family Factors. A range of family characteristics predicted psychopathology in the TRAILS cohort, including low SEP,[16,47] poor perceived parenting,[19,21,29,42,48-50] family adversity,[30,51,52] and divergent parental religions.[53] In particular, low maternal educational level and parental rejection proved to be important predictors. The effect of family factors often depended on the temperament, gender, and developmental phase of the adolescent, as well as the outcome under study.[15,29,30,49] These interactions with person characteristics confirm that exposure to the same challenges may entail different levels of risk for different individuals.

Peers. Peer status is highly relevant in the context of adolescent development. Important aspects of peer status include achievements (being admired) and affection (being liked); both contributing to popularity. The first contains an element of competition, whereas the second relates to being accepted or rejected by the social group. Our studies have linked peer-related factors to various mental health problems.[17,42,54-58] Gender-specific analyses revealed, among other things,[56] that depressive problems in boys were associated with not being good at sports, whereas in girls, not being liked by peers was most strongly associated with depression.[55] One study analyzed peer and parental acceptance and rejection simultaneously to investigate whether acceptance in one context can buffer rejection in the other.[42] The results suggest that peer acceptance partially buffers parental rejection but that parental acceptance does not buffer peer rejection.

Physiological Stress Sensitivity: a Mechanism Underlying Person–Environment Interactions? Overall, TRAILS findings indicate that person and environmental characteristics and their interactions shape mental health. Potential mechanisms regulating person–environment interactions involve the stress responses of the hypothalamic–pituitary–adrenal (HPA) axis and autonomic nervous system (ANS). Both have also been proposed as endophenotypes. However, so far associations of the HPA-axis and ANS with mental health problems have shown great variation and inconsistency. In TRAILS, more than 700 adolescents were subjected to a social stress task.[59,60] This provided the opportunity to relate physiological stress responses to mental health outcomes in a sample with sufficient power to detect small effects. As might be expected from the diverging findings reported previously, the associations found were usually weak or statistically insignificant. This hold true for associations with mental health outcomes[61] (Sijtsema J, Oldehinkel AJ, Ellis BJ, *et al.* Effects of family cohesion and heart rate reactivity on Aggressive/Rule-breaking behavior and prosocial behavior in adolescence: The TRAILS study. [Unpublished data].) and putative genetic risk alleles,[23,24] as well as for associations among HPA axis, autonomic, and subjective stress responses.[59] These small effect sizes are at least partly due to large interindividual variation in stress response. Prospective analyses indicate that psychophysiological stress responses during adolescence result from ongoing interactions of personal and environmental factors that may lead to either up- or down-regulation of the stress response. This depends, among other things, on gender and on the duration and timing of prior experiences (Booij S. History of depressive problems and the cortisol response to psychosocial stress in adolescents: The TRAILS study [Unpublished data].).[23,60,62] These findings further illustrate the adaptive nature of the stress response system[63] and show that, at least in adolescence, there is still a long road to the use of physiological stress responses as markers of risk or resilience in the domain of mental health.

What Next?

The fifth assessment wave of TRAILS will be completed in 2013. The five-wave data set, covering the developmental period from early adolescence into young adulthood, will provide opportunities to investigate continuity and discontinuity models of mental health that are based on the transactional developmental perspective.[64-66] In this perspective, continuity of psychopathology is driven by passive, evocative, and active person–environment (PE) correlations (rPE) and underlying PE transactions. An important subcategory includes gene–environment correlations (rGE). Parents of children with genotypes associated with a high risk for psychopathology may share up to 50% of their child's genetic risk factors for psychopathology. This may coincide with poor parenting skills (passive rGE). Furthermore, the difficult behaviors of children at high risk for developing psychopathology tend to elicit negative parental responses (evocative rPE),

reinforcing their risk. In as far as the difficult child behaviors are genetically determined, such rPEs are also denoted as rGEs. Later in development, these children will also tend to select high-risk environments (rPE, rGE) that maintain risk and may create additional risk. In contrast, continuity of mental health in low-risk children is also based on person–environment correlations and transactions, but because of low-risk genetic and environmental starting positions, the processes act in the opposite direction: adaptive behaviors elicit and select positive environments. As elaborated later here, PE transactions may not only fuel continuity but also may produce additional risk in the initially vulnerable, and therefore make developmental pathways diverge during adolescence.

The study of turning points in mental health is an important spearhead for future work in TRAILS. Because of the force of the person–environment correlation, discontinuity in mental health, such as the reversal of a maladaptive pathway into an adaptive one or vice versa, may be triggered by the behavior of others or chance events. In general, events that initiate long-term changes in personal assets, roles, and environments probably have the most potential to become turning points.[67] We found that the use of services for special educational needs during the transition from primary to secondary school was associated with discontinuity of childhood antisocial behavior.[68] Other spearheads include the interplay of childhood adversity, person characteristics, and later life events in affecting the onset and course of mental disorders, as well as the role of immunological factors, epigenetics, and mental–physical interplay. With the next measurement wave, TRAILS will also have excellent data to study long-term effects of psychopathology on social and economic outcomes and underlying mechanisms.

DEVELOPMENTAL PATHWAYS IN ADOLESCENCE: ACCUMULATION OR REDUCTION OF RISK IN ADOLESCENTS WITH HIGH INITIAL PROBLEM LEVELS?

Although evidence suggests an increase of problem behavior during adolescence,[69,70] it is unclear whether this behavioral development is a general phenomenon affecting most adolescents or concentrated in the initially vulnerable. If children with relatively high levels of problems before they enter adolescence are especially prone to show increasing levels of problems, this would strongly argue for early interventions. Two mechanisms provide theoretical plausibility to the hypothesis of concentration of risk in the initially vulnerable: namely, person–environment transactions and person–environment interactions.

As mentioned earlier, person–environment transactions include a variety of processes by which an individual takes a direct or indirect role in shaping his or her environment.[71] People tend to select environments and provoke life events that match their personality. Thus, individuals at high risk for developing psychopathology (called high-risk individuals) seek more difficult environments and are exposed to more stressful life events than individuals at low risk for developing psychopathology (called low-risk individuals). This process is known as "social selection."[72] These environmental risk factors may lead to additional problem behaviors, increased vulnerability, and decreased resilience, which may make downward spirals more likely for the initially vulnerable than for low-risk personalities. This latter process is known as "social influence," as the environment influences behavior and personality. Both social selection and social influence produce person–environment correlation and our understanding of these mechanisms is highly relevant for possible interventions.

The second mechanism that underlies concentration of risk is person–environment interaction, which implies that exposure to the same challenge or task may entail different risk depending on an individual's personality[73]. Menarche, one of the best-known stressful events faced by girls on the threshold of adolescence, acts as a risk factor for behavioral problems, but mainly in girls who were already predisposed to behavioral problems by virtue of their personality.[74] In other words, a transition or challenge faced by all may increase the relative disadvantage of the vulnerable in comparison to the nonvulnerable.

If either accumulation of risk in the highly vulnerable or reduction of risk in the less vulnerable, or both, occur during adolescence, normal and deviant development will tend to diverge as adolescence progresses. This implies, first, that the variance of problems in the population will increase during adolescence as the vulnerable and resilient diverge; second, that baseline problem levels are associated with increasing or con-

tinuously high levels of problem behaviors in adolescence; and third, that the association of childhood problems with adolescent problems remains strong throughout adolescence. This study targeted four important problem dimensions: aggression and rule-breaking behavior in the externalizing domain, and anxious/depressed and withdrawn/depressed behavior in the internalizing domain.

METHOD

Sample

The sample is described extensively in the introductory section of this article. For these analyses, we selected participants for whom we had information from at least two data points (n = 2140, 96.0% of full sample; 51% female and 49% male).

Measures

Psychopathology. We assessed psychopathology with the Youth Self-Report (YSR) and parent-reported Child Behavior Checklist (CBCL)[75-77] at the first three assessment waves (ages 10–18 years). We did not use data from the fourth wave because, in this wave, the YSR was replaced with the Adult Self-Report, and the CBCL was not administered. The YSR and CBCL cover behavioral and emotional problems in the preceding 6 months. Participants respond on a three-point scale (0 = not or not true, 1 = somewhat true, 2 = very often or true). The strong reliability and validity of the American versions of these tests were confirmed for the Dutch versions.[77] We focused on four dimensions: aggression (17 items; $\alpha \geq 0.80$ for self reports and $\alpha \geq 0.76$ for parent reports), rule-breaking behavior (15 items; $\alpha \geq 0.64$ for self reports and $\alpha \geq 0.68$ for parent reports), withdrawn/depressed behavior (8 items, $\alpha > 0.64$ for self reports and $\alpha \geq .071$ for parent reports), and anxious/depressed behavior (13 items; $\alpha \geq 0.78$ for self reports and $\alpha \geq 0.78$ for parent reports). For each of the four scales, we computed the average of the scale scores on the YSR and CBCL. In cases in which one report was missing, we used only the available report. We chose to combine parent and self-reports of adolescent problem behavior because reports from different informants on problem behaviors tend to differ substantially. Instead of choosing one informant or running the analyses for both informants separately, we chose to combine self- and parent reports, because by combining information we retained a more complete picture of the adolescent's functioning than by merely using one informant.[36]

Childhood Problems. At the first assessment wave (ages 10–11 years), parents reported retrospectively on participants' internalizing and externalizing behavior at the age of 4 to 5 years.[78] Parents were asked how their child behaved in comparison to other children on a five-point scale, ranging from 1 = "much less" to 5 = "much more." Seven items ($\alpha = 0.79$) assessed childhood internalizing behavior (such as "Was your child fearful?" and "Was your child quickly depressed?"). Four items ($\alpha = 0.70$) assessed childhood externalizing behavior (for example, "Did your child quickly lose his/her temper?" and "Did your child bully other children?"). It should be noted that we cannot exclude possible confounding of the retrospective parental rating by current mental health status of the preteen.

Statistical Analysis

Before analysis, we restructured the original dataset, creating a format that used "age" instead of "assessment wave" as the denominator. We then calculated means and standard deviations for family characteristics, early childhood problem behavior, and the four YSR/CBCL scales from ages 10 to 17 years. Subsequently, we examined the developmental trajectories of problem behavior during adolescence using Linear Mixed Models (LMM) in PASW Statistics 18.0.[79] These models allow us to estimate fixed and random effects to adjust for dependencies in the data arising from repeated measures of the same individual. We used age as a measure of time, treating it as both a fixed and random factor, because we assumed that values would vary randomly across measurement waves depending on age.[79] In our analyses, we set age 10 years as zero, so that the intercept of the models would refer to behavior at baseline (age 10).

We standardized the variables: first, the independent variables childhood internalizing and externalizing problems to a mean of zero and a standard deviation of one; second, the dependent variables across age to be able to estimate the relative weight of the model parameters (i.e., comparable to β-weights in a standard regression analysis). By doing this, the dependent variable informs about the relative position of the participants each year.

Handling Missing Values. Because we restructured the original dataset into a format using age as the denominator, rather than assessment wave, we were left with many missing data points in the dependent variables. However, the LMM procedure in PASW 18.0 is able to handle this type of missing data, as it estimates missing data between available data points. Therefore, we reconstructed the data file into long data format (i.e., variables to cases), which allows for the nesting of several data points within one individual and is a prerequisite for performing LMM analyses.

Linear Mixed Models. In the first step, we estimated an unconditional mean model, in which we determined the extent of variance in problem behavior that could be attributed to within- and between-individual differences. To do this, we added a random term for all individuals to indicate that there is some nonzero

covariance between the observations of problem behaviors within the same individual and to estimate the amount of between-subject variance in the model. For the random effects, we also included an intercept associated with each individual. We defined the covariance structure as "unstructured" to allow for estimation of variance and covariance of the random effects.[79]

In the second step, we used age as a predictor of difference between individuals, as well as a random effect, to account for changes in individuals over time. This is the linear growth model. We also added a random effect for age to specify a slope coefficient for each individual, denoting the changes within individuals over time.

If the pathways of high- and low-risk individuals diverge, it would be indicated by a positive covariance between the intercept and time (the linear growth parameter, or slope). However, if the covariance between intercept and slope is negative, pathways converge. A nonsignificant covariance indicates independence: the intercept and slope are unrelated.

In the third step, the quadratic growth model, we examined the curvilinear function of age to predict differences in problem behaviors between individuals. In the fourth step, the complete model, we added gender and childhood behavior problems. We considered gender to be a categorical fixed factor and childhood problem behavior a fixed covariate. We also tested interactions between both gender and childhood behavior problems, and the functions of age.

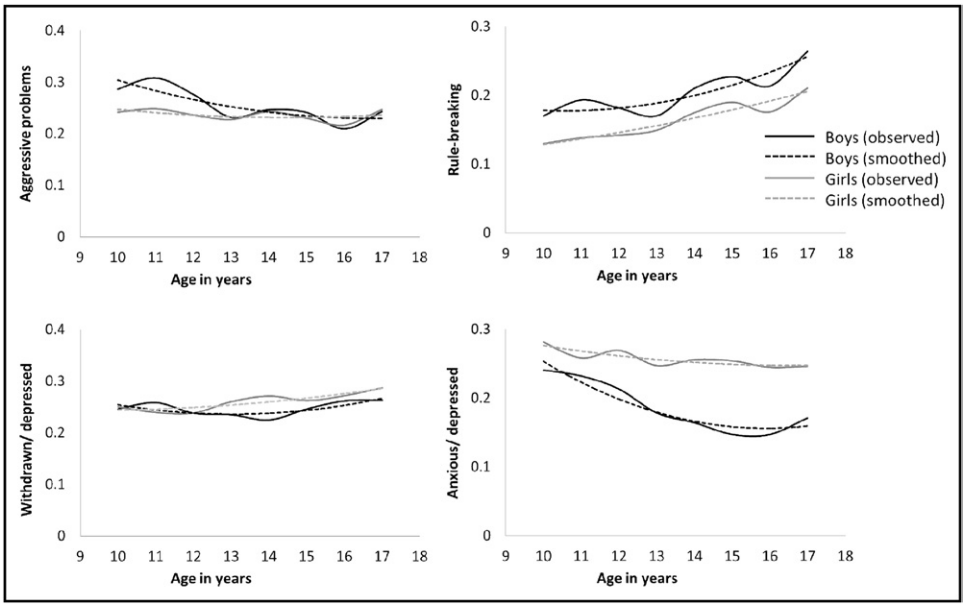
RESULTS

Table 4 shows the means and standard deviations of the study variables, and Figure 2 depicts the observed and smoothed gender-specific problem levels across adolescence. Mean levels of Aggressive Behavior and Anxious/Depressed problems decreased during adolescence, especially in boys, whereas those of the Rule-Breaking Behavior and Withdrawn/Depressed behavior increased in both genders. The variance of the Rule-Breaking and Withdrawn/Depressed Behavior scales increased substantially, with the variance of Rule-Breaking Behavior more than doubling. This means that individuals within the population tended to show increasing differences in their problem scores on the Rule-Breaking Behavior and Withdrawn/Depressed scales during development across adolescence. This increased variance is consistent with the diverging pathways hypothesis: it is a necessary, but not a sufficient, condition. The variance of the Aggressive Behavior scale was stable, and the variance of Anxious/Depressed Behavior increased only a little during adolescence, suggesting neither diverging nor converging pathways. Because decreasing variance suggests convergence, our results for the Aggressive Behavior and Anxious/Depressed scales did not support our hypothesis of diverging pathways.

TABLE 4 Means, Standard Deviations, and Variances of Childhood and Adolescent Behavioral Problems

	Mean (SD)	Variance	n
Childhood externalizing	2.56 (0.65)	0.425	1972
Childhood internalizing	2.63 (0.62)	0.386	1973
Aggressive Behavior	**Mean (SD)**	**Variance**	**n**
Age 10	0.26 (0.15)	0.024	1045
Age 11	0.28 (0.16)	0.026	908
Age 12	0.26 (0.16)	0.026	510
Age 13	0.23 (0.15)	0.022	1341
Age 14	0.24 (0.16)	0.026	465
Age 15	0.23 (0.16)	0.025	722
Age 16	0.21 (0.15)	0.023	744
Age 17	0.24 (0.17)	0.030	267
Withdrawn/Depressed	**Mean (SD)**	**Variance**	**n**
Age 10	0.25 (0.17)	0.028	1044
Age 11	0.25 (0.17)	0.029	907
Age 12	0.24 (0.16)	0.026	510
Age 13	0.25 (0.18)	0.031	1341
Age 14	0.25 (0.18)	0.032	465
Age 15	0.25 (0.19)	0.035	721
Age 16	0.27 (0.19)	0.038	744
Age 17	0.28 (0.20)	0.041	290
Rule-breaking Behavior	**Mean (SD)**	**Variance**	**n**
Age 10	0.15 (0.10)	0.009	1045
Age 11	0.17 (0.11)	0.011	907
Age 12	0.16 (0.12)	0.013	510
Age 13	0.16 (0.12)	0.014	1341
Age 14	0.19 (0.14)	0.020	465
Age 15	0.21 (0.14)	0.021	722
Age 16	0.19 (0.14)	0.020	744
Age 17	0.23 (0.15)	0.022	290
Anxious/Depressed	**Mean (SD)**	**Variance**	**n**
Age 10	0.26 (0.16)	0.025	1046
Age 11	0.25 (0.15)	0.023	907
Age 12	0.24 (0.16)	0.026	510
Age 13	0.21 (0.16)	0.026	1342
Age 14	0.21 (0.17)	0.028	465
Age 15	0.20 (0.17)	0.030	722
Age 16	0.20 (0.17)	0.028	744
Age 17	0.21 (0.17)	0.029	290

FIGURE 2 Observed and smoothed gender-specific problem levels in adolescence (ages 10–17 years).



Tables 5 and 6 presents the maximum likelihood estimates and standard errors of the LMM analyses for each of the four dependent variables. For the dependent variables, each progressive step of growth model fit the data better than the previous model, based on the −2 log-likelihood comparisons. Thus, the complete model fit the data better than the quadratic growth curve model, which fit better than the linear growth model, which, in turn, fit better than the unconditioned mean model. For reasons of parsimony, we do not present the estimates of the unconditional mean model (available on request). Between 50.4% and 60.4% of the variance in problem behaviors during adolescence was due to within-individual differences, rather than differences between individuals. This means that more than half of the differences in problem scores across adolescence in the general population are due to differences within individuals as a result of developmental changes.

Aggressive Behavior

The left panel of Table 5 shows the estimates and standard errors of the LMM analyses of Aggressive Behavior. The unconditional mean model (not presented in Table 5) showed that approximately 54% of the variation in aggression was due to interindividual differences. Comparison of the linear and unconditional model showed that the linear rate of change accounted for nearly 24% of the variation in aggressive behavior. The linear growth model shows that participants, on average, had an aggression score of 0.16 at age 10 and that, overall, this score decreased during adolescence (i.e., fixed effect of age, −0.05). The variances in intercept and age indicate that there are significant between-subject differences in both the initial level of aggressive behavior and its development during adolescence.

As indicated by the negative covariance of −0.04 between intercept and time in the linear growth parameter, the level of aggression in adolescents with higher initial scores decreased more than did the level of aggression in adolescents with lower initial scores. Thus, adolescents' Aggressive Behavior scores became more similar over time: their pathways converged. This is inconsistent with the hypothesis of diverging pathways.

The quadratic growth curve model shows a positive quadratic shape, indicating that, although aggression decreased overall, there was a slight increase in late adolescence. According to the complete model, including gender and childhood externalizing behavior, the initial aggression level was 0.35 for boys. This score was significantly lower for girls (0.05). Childhood externalizing behavior was a very strong predictor of aggression at age 10 years. Furthermore,

TABLE 5 Linear Mixed Models of the Effect of Age, Gender, and Childhood Externalizing Behavior on Aggressive and Rule-Breaking Behaviors in Adolescence

	Aggressive Behavior			Rule-Breaking Behavior		
	Linear Growth Model Estimate (SE)	Quadratic Growth Model Estimate (SE)	Complete Model Estimate (SE)	Linear Growth Model	Quadratic Growth Model Estimate (SE)	Complete Model Estimate (SE)
Fixed effects						
Intercept	0.16 (0.02)***	0.20 (0.02)***	0.35 (0.03)***	−0.22 (0.02)***	−0.17 (0.02)***	0.00 (0.03)
Age	−0.05 (0.00)***	−0.10 (0.01)***	−0.17 (0.02)***	0.08 (0.00)***	0.01 (0.01)	−0.05 (0.02)*
Age squared	—	0.01 (0.00)***	0.01 (0.00)***	—	0.01 (0.00)***	0.02 (0.00)***
Girl (ref = Boy)	—	—	−0.30 (0.05)***	—	—	−0.37 (0.04)***
Childhood externalizing	—	—	0.40 (0.02)***	—	—	0.20 (0.02)***
Interactions with age						
Girl	—	—	0.12 (0.03)***	—	—	0.11 (0.03)***
Childhood externalizing	—	—	−0.04 (0.01)**	—	—	0.01 (0.01)
Interactions with age squared						
Girl	—	—	−0.01 (0.00)*	—	—	−0.01 (0.00)**
Childhood externalizing	—	—	0.00 (0.00)	—	—	−0.00 (0.00)
Random effects						
Residual	0.35 (0.01)***	0.34 (0.01)***	0.33 (0.01)***	0.42 (0.01)***	0.41 (0.01)***	0.40 (0.01)***
Variance (intercepts)	0.70 (0.04)***	0.71 (0.04)***	0.53 (0.03)***	0.27 (0.03)***	0.29 (0.03)***	0.20 (0.02)***
Covariance (intercepts, time)	−0.04 (0.01)***	−0.04 (0.01)***	−0.03 (0.01)***	0.02 (0.01)**	0.01 (0.01)*	0.01 (0.01)**
Variance (age)	0.01 (0.00)***	0.01 (0.00)***	0.01 (0.00)***	0.01 (0.00)***	0.02 (0.00)***	0.02 (0.00)***
−2 Log Likelihood	15242.06	15226.51	13446.56	15481.64	15458.32	13812.45

Note: *p < .05, **p < .01, ***p < .001 (two-sided tests).

TABLE 6 Linear Mixed Models of the Effect of Age, Gender, and Childhood Internalizing Behavior on Depressive/Withdrawn and Anxious/Depressed Behaviors in Adolescence

	Withdrawn/Depressed			Anxious/Depressed		
	Linear Growth Model Estimate (SE)	Quadratic Growth Model Estimate (SE)	Complete Model Estimate (SE)	Linear Growth Model	Quadratic Growth Model Estimate (SE)	Complete Model Estimate (SE)
Fixed effects						
Intercept	−0.05 (0.02)*	−0.02 (0.02)	−0.00 (0.03)	0.18 (0.02)***	0.23 (0.02)***	0.15 (0.03)***
Age	0.02 (0.00)**	−0.02 (0.01)	−0.06 (0.02)**	−0.06 (0.00)***	−0.12 (0.01)***	−0.18 (0.02)***
Age squared	—	0.01 (0.00)**	0.01 (0.00)**	—	0.01 (0.00)***	0.01 (0.00)***
Girl (ref = Boy)	—	—	−0.08 (0.05)	—	—	0.14 (0.05)**
Childhood Internalizing	—	—	0.25 (0.02)***	—	—	0.25 (0.02)***
Interactions with age						
Girl	—	—	0.10 (0.03)**	—	—	0.12 (0.03)***
Childhood Internalizing	—	—	−0.04 (0.01)**	—	—	−0.03 (0.02)
Interactions with age squared						
Girl	—	—	−0.01 (0.00)*	—	—	−0.01 (0.00)
Childhood Internalizing	—	—	0.00 (0.00)*	—	—	0.00 (0.00)
Random effects						
Residual	0.39 (0.01)***	0.39 (0.01)***	0.37 (0.01)***	0.37 (0.01)***	0.37 (0.01)***	0.36 (0.01)***
Variance (intercepts)	0.53 (0.03)***	0.53 (0.03)***	0.46 (0.03)***	0.57 (0.03)***	0.57 (0.03)***	0.50 (0.03)***
Covariance (intercepts, time)	−0.02 (0.01)**	−0.02 (0.01)***	−0.02 (0.01)*	−0.02 (0.01)***	−0.02 (0.01)***	−0.02 (0.01)***
Variance (age)	0.02 (0.00)***	0.02 (0.00)***	0.02 (0.00)***	0.01 (0.00)***	0.01 (0.00)***	0.01 (0.00)***
−2 Log Likelihood	15572.41	15564.15	14078.62	15392.87	15375.85	13749.95

Note: *p < .05, **p < .01, ***p < .001 (two-sided tests).

interactions with age indicated that boys' aggression decreased annually by 0.17, whereas girls' aggression decreased by 0.05, suggesting that boys and girls became more similar during adolescence although they started off at different levels. Furthermore, the interaction between age and childhood externalizing shows that the effect of childhood externalizing decreased during adolescence by 0.04 per year.

Rule-Breaking Behavior

The right panel of Table 5 presents estimates and standard errors of the LMM analyses of Rule-Breaking Behavior. The unconditional mean model (not presented in Table 5) showed that approximately 44% of the variation in rule breaking was due to interindividual differences. Comparison of the linear and unconditional model showed that the linear rate of change accounted for nearly 25% of the variation in rule-breaking. The linear growth model shows that adolescents on average had a rule-breaking score of −0.22 at age 10, and that this score increased during adolescence by 0.08 annually. The quadratic growth curve model shows a positive quadratic shape, indicating that this increase in rule-breaking behavior largely occurred in the second half of adolescence. The linear effect was no longer significant.

The positive covariance between intercept and time indicates that the level of rule-breaking behavior in adolescents with higher initial scores increased more than in adolescents with lower initial scores. Individuals thus became more dissimilar over time in terms of rule breaking. In other words, their pathways diverged, consistent with the diverging pathways hypothesis.

According to the complete model, the level of Rule-Breaking Behavior scale scores at age 10 was estimated at 0.0 for boys. This score was significantly lower for girls (−0.37). Childhood externalizing was a strong predictor of rule breaking during adolescence, and its effect did not decay. This is indicated by the lack of negative interaction of childhood externalizing with age. Interactions of gender with age indicate that girls' rule-breaking behavior increased during adolescence (0.06 annually), whereas boys decreased (−0.05 annually). Girls, however, began at a much lower level of rule breaking than boys. Furthermore, boys increased more in rule breaking during the second half of adolescence than girls, as indicated by the quadratic age effect. The significant variances in the intercept and age indicate that there are significant differences between individuals in both the initial levels of rule breaking and its development.

Withdrawn/Depressed

The linear and quadratic growth models indicate that Withdrawn/Depressed scale scores (Table 6) began to increase at age 15 years, resulting in an overall increase during adolescence. Participants had a mean Withdrawn/Depressed score of −0.05 at age 10 years. The unconditional mean model (not presented in Table 6) showed that approximately 51% of the variation in Withdrawn/Depressed scores was due to interindividual differences. Comparison of the linear and unconditional model showed that the linear rate of change accounted for about one-fifth (21.7%) of the variation in Withdrawn/Depressed scale scores.

A negative covariance of −0.02 between the intercept and linear growth parameter implies that withdrawal and depression in adolescents with high initial levels of withdrawal and depression increased less compared to adolescents with low initial levels of withdrawal and depression, which increased more strongly. Individuals thus became more similar over time. This tendency to converge does not support the diverging pathways hypothesis.

The complete model shows that childhood internalizing strongly predicted Withdrawn/Depressed Behavior scores in early adolescence. However, that effect decayed during adolescence, at least in part, as indicated by the negative interaction with age (−0.04). Interactions of gender with age indicate that boys' Withdrawn/Depressed level decreased slightly, followed by an increase in the second half of adolescence, whereas girls' Withdrawn/Depressed level increased by 0.04 annually. At age 10 years, boys and girls did not have significantly different depression levels; their divergence occurred after age 14.

Anxious/Depressed

The linear and quadratic growth model estimates for Anxious/Depressed in Table 6 indicate that this behavior decreased in the first half of the study period, with a smaller increase in the second half. The linear model shows that participants had a mean Anxious/Depressed

score of 0.18 at age 10. The unconditional mean model (not presented in Table 6) showed that approximately 52% of the variation in Anxious/Depressed was due to interindividual differences Comparison of the linear and unconditional model showed that 23% of the variation in Anxious/Depressed was explained by the linear rate of change.

Significant variances in the intercept and age indicate that there are significant differences between individuals in both the initial level of anxious/depressed behavior and its development.

As indicated by the negative covariance of −0.02 between the intercept and time, the level of anxiety and depression in adolescents with high initial scores decreased more than the level of anxiety and depression in adolescents with lower initial scores, which decreased less steeply. Individuals' Anxious/Depressed scores thus became more similar over time. In other words, their anxious/depression pathways converged. This is inconsistent with the hypothesis of diverging pathways.

The complete model estimates indicate that, at age 10, boys had an Anxious/Depressed scale score of 0.15 and girls 0.29. Again, childhood internalizing behavior was a strong predictor of higher Anxious/Depressed scores at age 10, and its predictive value hardly fell during adolescence, because the negative interaction with age was marginally significant ($p = 0.06$). Interactions of gender with age indicate that boys' Anxious/Depressed scores decreased more strongly during the first half of adolescence than girls' scores. Girls, however, started off at a higher level than boys. In both genders, Anxious/Depressed scores increased slightly in late adolescence (Figure 2).

DISCUSSION

In this study, we examined the development of internalizing and externalizing behaviors during adolescence. Our analyses produced two important sets of findings. The first set emphasizes that the study of developmental pathways needs to take into account the individual's gender, type of psychopathology, and age. Normative development differs not only between internalizing and externalizing problems, but also between dimensions within these broad domains. Rule-Breaking Behavior, such as stealing, substance use, and truancy, increased during adolescence, whereas aggressive behaviors, mostly behaviors involving physical aggression such as fighting, decreased. This finding is consistent with other recent studies.[80,81] The decrease in aggression was weaker for girls and aggression tended to increase slightly, in both boys and girls, later in adolescence. In the internalizing domain, Anxious/Depressed and Withdrawn/Depressed scale scores decreased over time in boys, with a small increase in late adolescence. However, in girls, these behaviors first remained stable and then increased, especially withdrawn/depressed behaviors. Consistent with an earlier report,[80,81] this evidence suggests that normative developmental pathways are dimension and gender specific. From a clinical point of view, it is important to know what developmental changes are to be expected that can be regarded as normal.

Our second set of findings addresses the diverging pathway hypothesis, which was supported only by Rule-Breaking Behavior scores. The developmental pathways of aggression and the two internalizing problem scales, unexpectedly, tended to converge rather than diverge. We do not have a straightforward explanation for these findings. Rule-Breaking Behavior is the dimension with the strongest mean increase during adolescence, and also the dimension that is probably most strongly affected by peer influences. It is possible that peer influences play an important role in the person–environment transactions that fuel the diverging pathways of rule-breaking behaviors. There is evidence showing that individuals with relatively high levels of rule-breaking behavior in late childhood or early adolescence tend to socialize with peers with similar rule-breaking and associated behaviors.[82] Social pressures in these deviant peer groups may reinforce and expand rule-breaking behavior during adolescence.[83,84] This hypothesized role of peers fits the "corresponsive principle" that the most likely effect of life experiences on behavior and personality development is to enhance the characteristics that led people to those experiences in the first place.[72] The corresponsive principle links the mutually reinforcing person–environment transactions of social selection and social influence. In the context of rule breaking, social selection refers to the selection of young rule breakers into deviant peer groups. Social influence refers to the impact of a group on the rule-breaking behavior of its members by means of social pressure and exposure to group-created rule-breaking opportunities. Recent evidence

supports this hypothesis.[82] This finding has implications for both researchers and clinicians. If we want to bend the negative spiral of those adolescents with rule-breaking behavior toward better adjustment, we need to intervene as soon as these behaviors emerge and especially look into strategies that will interfere with the adolescents' tendency to interact with peers who are deviant.

Why did we find converging, and not diverging or stable, pathways for internalizing and aggressive problems? We offer six post hoc explanations for the unexpected findings. First, selective attrition. Nonresponse analyses showed that attrition is associated with baseline problem level, in particular of the externalizing type.[1,2] The effect of relatively high attrition in those with high baseline and increasing problem levels is a bias toward converging pathways. However, it should be noted that we do not know whether baseline problem level in the attrition group is associated with increasing problem levels. Second, high and increasing problem levels may set into motion counter-forces that reduce or reverse deviant development. Examples of potential counter-forces include mental health treatment and prevention, police and judicial involvement, and school and parental interventions. This comports with evidence that we previously documented, that treatment for emotional and behavioral problems in adolescence is concentrated in those with high or increasing problem levels.[85] Third, in contrast to rule-breaking behavior, it is difficult to envision how peers could play a similar corresponsive role in influencing anxiety and depression.[86] Fourth, adolescents with high initial problem levels may benefit more from the maturation of brain structures involved in the regulation of emotions and behaviors.[87-89] Fifth, the converging trend may be due to a transitory increased level of problem behaviors of some individuals at baseline who subsequently return to their characteristic set point level of problem behaviors.[90] Finally, it should be noted that these findings for internalizing problems and aggression do not preclude the possibility that risks of these problems accumulate in particular individuals and subgroups. However, the data clearly showed that such an accumulation is unrelated to initial vulnerability as indexed by baseline problem levels.

Childhood problems strongly predicted problems in early adolescence. However, the magnitude of their association with problem levels later in adolescence gradually decreased (except for rule breaking, for which the association with early adolescent levels was weaker but remained stable throughout adolescence). Again this suggests that adolescence is not a life stage where risk accumulates in the initially vulnerable, with the exception of rule-breaking behavior; rather, the collective evidence points to stability of risk on one hand, and changes of risk unrelated to childhood problem levels on the other. Thus, the diverging pathways observed in rule-breaking behaviors may not only be due to accumulating risk in the vulnerable, but also to increased opportunity to break rules.

These findings should be interpreted in the context of their limitations and strengths. Some notable strengths of this study include its large sample of adolescents and 8-year follow-up period, multi-informant ratings on a range of problem behaviors throughout adolescence, limited nonresponse at baseline, and limited attrition at follow-ups. This allowed us to examine whether developmental pathways diverge in adolescence. However, an important limitation should also be mentioned. We could not use the data collected at T4, when the cohort was 18 to 20 years, both because of a change from the Youth Self Report to the Adult Self Report, which contains somewhat different items, and because of a lack of parental ratings. Therefore, the data analyzed do not cover the last phase of adolescence.

Although reasonable arguments support the hypothesis of diverging pathways during adolescence, derived from person–environment transactions and interactions that promote an ongoing concentration of risk in the vulnerable, we found evidence of diverging pathways only in rule-breaking behavior. We think that these diverging rule-breaking pathways arise from early socialization with deviant peers. Pathways for aggression and internalizing problems did not diverge. This may be due to effective counter-forces, such as mental health treatment and judicial involvement, or because these dimensions of psychopathology are less sensitive to environmental influences, including peers. Investigation of the validity of these hypotheses is one of the challenges facing the field. Finally, the view that problem levels increase during adolescence needs refinement, in terms both of problem type

and gender. Notably, although rule-breaking (in both genders) and withdrawn/depressed behavior (particularly in girls) increased during adolescence, we found no increase in aggression or anxious/depressed behavior. &

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