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## Environmental influences on neuroticism : a story about emotional (in)stability

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# Chapter 6

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## **Why Does Frustration Predict Psychopathology? Multiple Prospective Pathways over Adolescence: A TRAILS Study**

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## ABSTRACT

Temperamental frustration predicts psychopathology in adolescence. This study tested four hypotheses about pathways that may underlie this prospective association: stress generation (mediation via selection and evocation of stressful life events), heightened stress sensitivity (more psychopathology after events), cross-sectional frustration-psychopathology overlap (“carry-over”), and a direct (non-mediated) effect of frustration. Additionally, we explored whether high frustration predicts increases in internalizing and externalizing tendencies (mutually adjusted). Data were derived from assessment waves at age 16 and 19 of the longitudinal Tracking Adolescents’ Individual Lives Survey (TRAILS). At age 16, frustration and psychopathology were assessed with the Early Adolescent Temperament Questionnaire (EATQ-R) and the Youth Self Report (YSR). At age 19, psychopathology was reassessed using the Adult Self Report (ASR), while occurrence of endogenous (self-generated) and exogenous (not self-generated) SLEs during the interval (ages 16-19) were ascertained with the Life Stress Interview and assessed with an investigator-based contextual stressfulness rating procedure ( $n=957$ ). We observed that frustration-prone adolescents generated more SLEs, and were more sensitive to endogenous SLEs, but not to exogenous SLEs. Stress-generation and sensitivity explained little of the two-year prospective association of frustration on psychopathology, while over half of the association was due to overlap between frustration and psychopathology at baseline, and 45% reflected unmediated association. Furthermore, frustration was associated with externalizing but not internalizing behavioural tendencies, when controlling for overlap between the two. Our results underscore the pivotal role of high frustration in adolescent development, its prospective effect on psychopathology, and high affinity with externalizing-specific features thereof.

## INTRODUCTION

Frustration is a temperamental trait that captures distress in response to limitations, exclusion, and failure. Frustration generalizes across contexts; affecting social relationships, social interactions, and job performances [662,855,856], and antedates vandalism and violence [857,858]. High frustration reportedly predicts both internalizing and externalizing psychopathology in childhood and early adolescence [196,220,822,859,860]. Hitherto the mechanisms that underlie the association between temperamental frustration and psychopathology have not been studied extensively. In the present study we investigate four possible pathways that may channel the prospective association between frustration and psychopathology (defined as total problem score): stress generation, stress sensitivity, overlap with concurrent psychopathology, and a direct effect of frustration (*i.e.*, a pathway unmediated by stressful events and unconfounded by previous psychopathology). In the following we provide a rationale for each of these pathways. Finally, to enhance our understanding of the prospective association between temperamental frustration and psychopathology, we explore these pathways for direction markers of internalizing or externalizing tendencies separately; and test whether observed effects differ by gender.

### Stress Generation (Mediation)

Individuals with a particular disposition tend to select themselves into environments that cultivate this predisposition, a process known as the corresponive principle [64]. For example, this widening of intrinsic differences via reciprocal causality has been reported for neuroticism, a strong predictor for internalizing psychopathology, and stressful life experiences (SLEs), *e.g.* [100,239]. Our first hypothesis (H1) held that individuals who score high on temperamental frustration conduct their lives in ways that increase the probability of SLEs, causing more psychopathology (mediation). We subdivided SLEs into the commonly used categories of *endogenous* SLEs, judged to be brought about by an individual's own behavior (personal), and *exogenous* SLEs, not directly brought about by individual's own behavior. We hypothesized that individuals high (*vs.* low) on frustration selected themselves into more endogenous SLEs (H1a), but were also exposed to more exogenous SLEs (H1b). Based on the definition of endogenous and exogenous SLEs we expected the active selection for endogenous SLEs to be stronger than the effect of high frustration on exogenous SLEs (H1c).

### Stress Sensitivity (Moderation)

Temperamental frustration has been found to enhance the impact of negative environmental influences, such as low social-economic status, family instability, negative parenting styles, and divorce, on psychopathology [198,861-863]. We hypothesize

in line therewith that high frustration increases the impact of SLEs on subsequent psychopathology (H2a). The most fundamental concepts underlying individual differences in sensitivity to stressful environments and emotion are approach and avoidance processes; approach of appetitive stimuli, and avoidance of threat, novelty, and frustrated non-reward [22,864]. Frustration is the common emotional response after opposed approach [219,698], especially when individuals feel blocked by (intentional) antagonistic acts [865-867]. Frustration can in turn lead to an approach or avoidance process, dependent on the perceived level of control over the antagonist [698,867-869]. From a functional perspective, frustration is supposed to facilitate approach persistence if the antagonist is perceived as controllable (*e.g.*, via anger, *cf.* [698]), but to low approach (or avoidance) if the antagonist is perceived as uncontrollable, and the incentive is unattainable (*e.g.*, via sadness or anxiety, *cf.* [867]). Since frustration tends to follow thwarted goal approaches, and endogenous (*vs.* exogenous) SLEs are commonly perceived as more controllable [870], we hypothesize that high (*vs.* low) frustration leads to more psychopathology after endogenous SLEs than after exogenous SLEs (H2b).

### **Two Other Pathways: Overlap and Direct Effects**

Two other possible pathways between frustration and later psychopathology were hypothesized. We felt that the prospective association between frustration and psychopathology is confounded by the overlap between these measures at baseline (H3). This overlap results from three sources: i) common causes, ii) correlation due to earlier bi-directional effects, and iii) operational (measurement) overlap. To quantify the part of the prospective effect that is due to overlap at baseline we compared two models, one with and one without baseline psychopathology included as predictor. The comparison of the estimates in these models enabled us to quantify a fourth possible pathway, an independent “direct” (*i.e.* non-mediated) prospective association of frustration on psychopathology (H4); which is neither due to overlap with baseline psychopathology, nor mediated by SLEs.

### **Direction Markers**

Psychopathology (the total of problems) is conventionally subdivided over a broadband of internalizing tendencies directed towards the self (such as anxiety or depression) and a broadband of externalizing tendencies directed towards others (such as aggression and rule breaking), see [871-873]. This internalizing and externalizing broadband share a core of pathological distress (the deviation from normality), which has been labeled as a ‘severity marker’, and which associated cross-sectional at age 11 with frustration [220] and at age 13 with earlier frustration [822]. We extend upon these analyses and asked whether frustration predicts both internalizing- and externalizing-*specific*

tendencies. We derived such tendencies via mutual adjustment of both broadband measures, yielding residuals that could be used as ‘direction markers’ of pathological manifestation in adolescence. Based upon notions described above in the paragraph on stress sensitivity, we hypothesized that high (*vs.* low) frustration predicted increases in both internalizing and externalizing broadband-specific tendencies (H5a), but is most predictive for change in the externalizing direction (H5b). Additionally, we hypothesized that more endogenous SLEs resulted in increases in externalizing (more than internalizing) tendencies (H6a) whereas more exogenous SLEs resulted in increases in internalizing (more than externalizing) tendencies (H6b).

### Gender Differences

No gender differences in frustration have been observed in childhood [874]. Earlier studies in our TRAILS sample reported on lower frustration levels for girls than boys in early adolescence [861]. Subsequently, and new to the literature, we examined whether levels of frustration differ by gender at age sixteen. Equal frustration levels do not impede cultural display rules, which render assertive action and outward expression of frustration less appropriate for girls than boys [683,875,876]. Such highly gendered behavioural styles [64] align with the observation that girls score higher on internalizing psychopathology and boys on externalizing psychopathology over childhood and adolescence [220,822,877]. As high frustration predicts increases in both internalizing and externalizing psychopathology (*e.g.*, [220,860]), we hypothesized that these cultural display rules manifest in stronger associations between high frustration and internalizing tendencies in girls (H7a, more than externalizing) and externalizing tendencies in boys (H7b, more than internalizing).

## METHODS

### Sample

We used data from two waves of the ongoing longitudinal Tracking Adolescents’ Individual Lives Survey (TRAILS). TRAILS is a prospective cohort study of Dutch adolescents described before [878,879]. Briefly, five assessment waves have been completed to date. The study started with 2230 (pre)adolescents (response rate 76%, mean age 11.1, SD= 0.6, 51% girls [880]). Response rates of the third and fourth wave were 81% ( $n= 1816$ , mean age 16.3 years, SD= 0.7, 52% girls) and 83% ( $n= 1881$ , mean age 19.1 years, SD= 0.6, 52% women). In the rest of this paper, we denote the third and fourth wave as ‘age 16’ and ‘age 19’, respectively. The Dutch Central Committee on Research Involving Human Subjects (CCMO) approved the study. Participants were

treated in compliance with APA ethical standards, and all measurements were carried out with their adequate understanding and written consent.

Stressful life events (SLEs) in the interval between these two waves were assessed using an investigator-based procedure yielding contextual stressfulness rating. During the wave at age 19, we administered the Life Stress Interview (LSI; [881], a labour-intensive interview. Because of the high costs, the LSI was not administered to all participants. Of the 1584 participants who had completed the Composite International Diagnostic Interview (CIDI), 45% ( $n = 659$ ) met the DSM-IV criteria for a lifetime psychiatric disorder during the past year. All of those were eligible for the LSI, and 580 (89%) were actually interviewed (72 were not interviewed due to logistic constraints). Of the adolescents without a lifetime DSM-IV diagnosis ( $n = 808$ ), nearly half were randomly selected and 377 actually interviewed. Thus, a total of 957 adolescents were interviewed (mean age = 19.1,  $SD = 0.6$ , 55% women). Compared to the whole sample, LSI respondents were more often women (55% versus 50%;  $\chi^2_1 = 4.5$ ,  $p = .04$ ) and younger (mean age 19.0,  $SD = 0.6$  versus 19.2,  $SD = 0.6$ ;  $t(df=1879) = 6.3$ ,  $p < .01$ ).

## Measures

### *Frustration*

Temperamental frustration was assessed at age 16 with the Dutch [882] short form of the parent version of the Early Adolescent Temperament Questionnaire-Revised (EATQ-R [219], 5 items,  $\alpha = .75$ ). The EATQ is grounded in Rothbart's temperament model [123,193]. We used the parent version because in the TRAILS sample the factor structure of this version was superior to that of the child version [220].

### *Psychopathology*

At age 16, mental health was assessed with the Youth Self-Report (YSR) [883] and at age 19 with the Adult Self-Report (ASR) [884]. Both instruments assess behavioural and emotional problems in the preceding six months. We computed (i) a total problem score, (ii) an internalizing domain score, a composite of anxious/depressed ( $k = 13$ ,  $\alpha = .84$  at age 16;  $k = 18$ ,  $\alpha = .91$  at age 19) and withdrawn-depressed ( $k = 8$ ,  $\alpha = .74$ ;  $k = 9$ ,  $\alpha = .76$ ); and (iii) an externalizing domain score, a composite of rule breaking ( $k = 15$ ,  $\alpha = .76$ ;  $k = 14$ ,  $\alpha = .77$ ) and aggression ( $k = 17$ ,  $\alpha = .81$ ;  $k = 15$ ,  $\alpha = .85$ ). The reliability and validity of American versions have been confirmed for the Dutch versions [885].

### *Life Events*

SLEs were assessed with Kendler's [881] Life Stress Interview (LSI), which was based on the Life Events and Difficulties Schedule by Brown and Harris [271]. The LSI encompasses eleven personal events, that is, events occurring primarily to the

respondents themselves, among which assault, breakup of romantic relationship, illness or injury, trouble with police, loss of a confidant and difficulties at work or school. In addition, there are four classes of events occurring primarily to an individual in the respondent's social network (*e.g.*, a serious crisis, illness, or death). Each reported SLE was dated as accurately as possible by means of mnemonic aids such as personal calendars.

A distinguishing feature of the LSI is that the events are not rated by the respondent, but by the interviewer. Furthermore, the ratings are contextual, that is, based on what most people would feel about an event given the circumstances and biography, taking no account of respondents' reaction or any following mental health problems. Interviewer-based contextual ratings are essential to prevent intra-category variability and to disentangle objective event characteristics from the emotions and behaviors evoked by the event [268,269]. Events were rated on severity (*i.e.*, long-term contextual threat) and dependence on respondent's own will or behavior (*i.e.*, planned actions called endogenous SLEs or events caused by neglect or carelessness called exogenous SLEs). Severity ratings ranged from 1= minor to 4= severe; dependence ratings ranged from 1= clearly independent, 2= probably independent, 3= probably dependent, to 4= clearly dependent.

All interviewers were extensively trained and regularly attended booster sessions in order to ensure reliable and valid scores. All interviews were recorded and scored by a second rater blind to the initial interviewer's scores. In case of discordant ratings, the two raters discussed the scores until consensus was reached or a third rater made the final judgment. We calculated the summed severity scores of all events that occurred within the time frame of two years, separately for endogenous and exogenous events.

### Statistical Analysis

We performed data cleaning steps and calculated descriptives in SPSS (version 20, SPSS Inc., Chicago, Illinois). Endogenous SLEs were skewed to the left, and both endogenous and exogenous SLEs were kurtotic. We explored associations between variables using Spearman rho ( $r_s$ ) and Spearman partial rho ( $r_{sp}$ ) coefficients [419]. Gender differences were tested with bootstrapped *t*-tests ( $k= 10,000$ , two-tailed significance,  $H_0: b= 0$ ). Standardized beta weights ( $\beta$ ) report the change in outcome per standard deviation change in a predictor, and quantify the strength of the direct effect of one variable on the other in the context of multiple comparisons [424]. We classified correlations ( $r$ ) and betas as small if between .10 and .29, moderate between .30 and .50, and large if above .50 [373,466].

Our hypotheses were tested with a system of multiple regression equations specified in structural equation models (SEM) in Mplus 7.11 software [421]. We applied maximum likelihood estimations with robust standard errors (MLR) to account for



the non-normality of the data [422,886]. We tested all paths for their contribution to the fit of a model in terms of significant change in Akaike and Bayesian information criteria (AIC, BIC), see [887]. Nested model modifications that improved on baseline fit converged in our final models, in which all-insignificant paths were fixed at zero, and only paths that influenced fit were estimated.

For each final model we report change in fit relative to an unmodified baseline model in which the final model was nested (and in which all paths were estimated freely). For our gender models we also tested all paths for gender-equality, and where possible constrained paths to be equal for boys and girls (Jöreskog tradition). The small number of degrees of freedom in our models ( $df \leq 17$  to 23) combined with seven variables ( $(k(k+1)/2) = 27$  known values) suffices for model identification but not for reliable secondary (e.g., RMSEA) fit indices, see [424,888,889]. We checked the robustness of our results with equivalent models in SEM [890] and with multiple linear regression models in SPSS (version 20, SPSS Inc., Chicago, Illinois), but because results led to the same conclusions, we only report the SEM models.

### ***Direction Markers***

Direction markers for internalizing and externalizing tendencies were derived with linear regression analyses of concurrent measurements on one another at both waves, analogous to the calculation of partial  $r_s$ . We used the mutual adjusted residuals as ‘direction markers’ or internalizing and externalizing broadband-specific tendencies in SEM. Psychopathology henceforth refers to *total* problem scores.

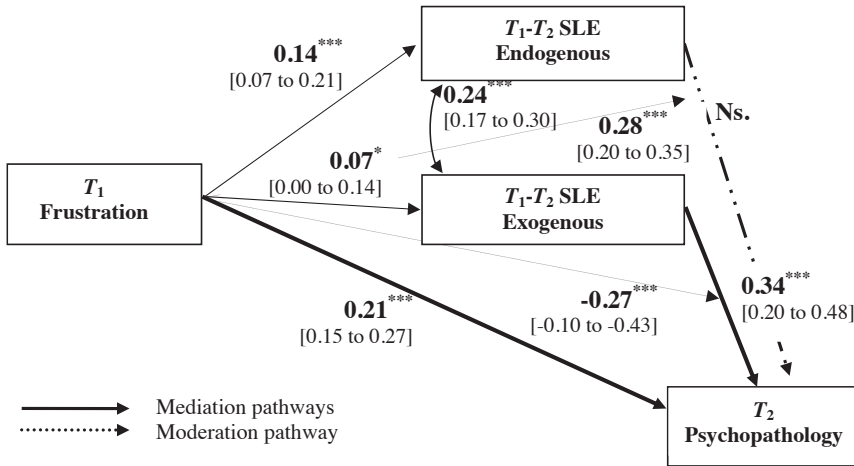
## **Model Fitting**

### ***Frustration Model***

The stress generation (H1) and stress sensitivity (H2) hypotheses were tested in a ‘frustration model’ fit with frustration as predictor of SLEs, and frustration and SLEs predicted psychopathology. The final model is presented in Figure 7 ( $\Delta X^2 = 0.74$ ,  $\Delta df = 1$ ,  $p = .40$ ,  $\Delta BIC = 5.7$ ,  $\Delta AIC = 0.8$ ); all modelling details are provided in Appendix Table A9.

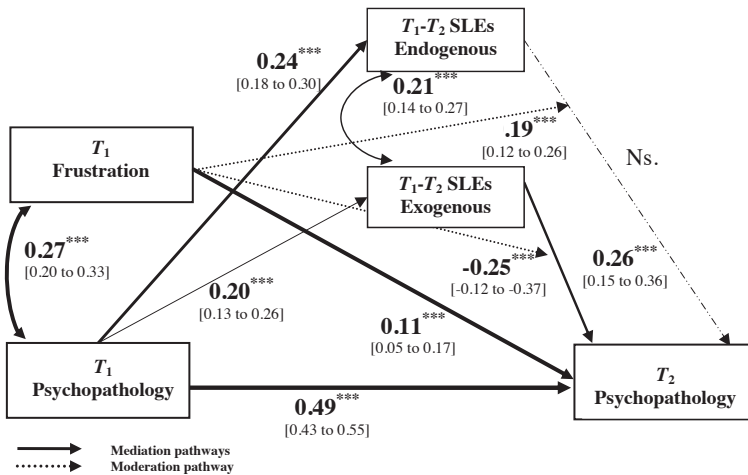
### ***Change in Psychopathology Model***

To quantify the overlap between frustration and psychopathology at baseline (H3) we fit a ‘change in psychopathology model’ (henceforth ‘change model’); the final model is presented in Figure 8 ( $\Delta X^2 = 3.56$ ,  $\Delta df = 3$ ,  $p = .31$ ,  $\Delta BIC = 16.8$ ,  $\Delta AIC = 2.2$ ), but all modelling details are provided in Appendix Table A9. The change model was extended with baseline psychopathology as a predictor, and a comparison of betas in both models (as depicted in Figure 7 and 8) enable for a rough quantification of the concurrent



**Figure 7.** Frustration Model

Note.  $N = 810$ . Ns. = non-significant. The model reports standardized estimates and confidence intervals between brackets [95% CI].  $T_1$  = baseline (age 16),  $T_2$  = follow-up (age 19); SLEs = stressful life events. Definitions of and details on each variable is given in the method section, as well as modeling details (see Appendix Table A9 for model fit, and Table A11 and A12 for tests of all paths). Significance \*\*\* $p < .001$ , \*\* $p < .01$ , \* $p < .05$ , two-tailed.



**Figure 8.** Change in Psychopathology Model

Note.  $n = 957$  (523 women, 54.6%). The model reports standardized estimates and confidence intervals between brackets [95% CI].  $T_1$  = baseline (age 16),  $T_2$  = follow-up (age 19); SLEs = stressful life events. Definitions of and details on each variable is given in the method section, as well as modeling details (see Appendix Table A9 for model fit, and Table A11 and A12 for tests of all paths). Significance \*\*\* $p < .001$ , \*\* $p < .01$ , \* $p < .05$ , two-tailed.

overlap and of the independent prospective effect of frustration on psychopathology (in terms of percentage of the betas in Figure 7, and on base of indirect effects in the change model).

### ***Direction Markers***

To explore whether frustration predicts both internalizing and externalizing broadband-specific tendencies (H5a/H5b) we fit the ‘frustration model’ and ‘change model’ for both direction markers separately. We compared the betas from each model to see whether endogenous SLEs were most predictive for externalizing tendencies (H6a); and whether exogenous SLEs were most predictive for internalizing tendencies (H6b). The final internalizing change model ( $\Delta X^2= 7.22, \Delta df=5, p= .21, \Delta BIC= 26.2, \Delta AIC= 2.6$ ) and final externalizing change model ( $\Delta X^2= 4.59, \Delta df=5, p= .47, \Delta BIC= 28.5, \Delta AIC= 4.2$ ) are presented in Table 22. Both frustration models, and all modelling details, can be found in Appendix Table A14-16 and A17-19, respectively.

### ***Gender Differences***

We stratified our SEM models by gender, and the final change models are presented in Table 23: for psychopathology ( $\Delta X^2= 15.21, \Delta df= 14, p= .36, \Delta BIC= 79.8, \Delta AIC= 11.8$ ), internalizing tendencies ( $\Delta X^2= 16.99, \Delta df= 13, p= .20, \Delta BIC= 69.5, \Delta AIC= 6.3$ ) and externalizing tendencies ( $\Delta X^2= 17.10, \Delta df= 16, p= .38, \Delta BIC= 90.2, \Delta AIC= 12.4$ ). Both frustration models, and all modeling details, are presented in Appendix Table A20-A26.

### ***Power***

The 957 participants enabled a free-parameter to sample ratio above 1:50 in all models tested [423]. This enabled reasonably precise estimation of local effects beyond Cohen’s  $f^2= 0.013$  in our most complex models ( $R^2 > 0.01, d= 0.20$ ), given 80% power, 5 predictors, and  $\alpha= 0.05$  [891]. Robustness checks with linear regression analyses are reported in Appendix Table A27.

## **RESULTS**

### **Descriptives**

We present our descriptives in Table 20. Participants of both genders reported an equal number of endogenous SLEs ( $t_{(955)}= 0.33, p= .75, \text{men} = 3.4 \text{ vs. women} = 3.3$ ), but women reported more exogenous SLEs than men ( $t_{(955)}= 4.9, p < .001, = 6.9 \text{ vs. } 5.3$ ). The associations between the studied variables are presented in Table 21. Temperamental frustration associated with psychopathology, both concurrently and prospectively.

**Table 20** Descriptives of the study variables

	Wave	N	Range	Mean (SD)
Frustration	$T_1$	810	1.00 to 4.80	2.72 (0.68)
Age (in years)	$T_1$	915	14.81 to 18.48	16.23 (0.67)
Psychopathology	$T_1$	866	0.00 to 2.37	0.66 (0.38)
Internalizing	$T_1$	866	-0.53 to 1.24	0.00 (0.01)
Externalizing	$T_1$	866	-0.38 to 0.85	0.00 (0.01)
Age (in years)	$T_2$	957	17.99 to 20.84	18.99 (0.57)
Endogenous SLEs	$T_1$ - $T_2$	957	0.00 to 26.00	3.36 (3.87)
Exogenous SLEs	$T_1$ - $T_2$	957	0.00 to 35.00	6.15 (5.17)
Psychopathology	$T_2$	942	0.00 to 2.41	0.52 (0.41)
Internalizing	$T_2$	942	-0.39 to 0.72	0.00 (0.01)
Externalizing	$T_2$	942	-0.51 to 0.75	0.00 (0.01)

Note. The sample contained 523 girls (54.6%) and 434 boys (45.4%). The calculation of the internalizing and externalizing tendencies is described in the method section. SD= standard deviation; SE= standard error; N= sample size; SLEs= stressful life events;  $T_1$ = baseline,  $T_2$ = follow-up.

**Table 21.** The lower half shows Spearman correlations ( $r_s$ ) between all variables. The above half shows partial  $r_s$  between frustration and endogenous and exogenous SLEs (SLEs mutually adjusted).

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	
1. Gender												
2. Frustration	$T_1$		-.01				.06	-.08				
3. Psychopathology	$T_1$		-.21***	.26***								
4. Internalizing Tendencies	$T_1$		-.42***	.10**	.62***			-.01	.13***			
5. Externalizing Tendencies	$T_1$		.19***	.19***	.48***	-.30***		.23***	.03			
6. Endogenous SLEs	$T_1$ - $T_2$		-.03	.10***	.23***	.03	.24***			-.08*	.27***	
7. Exogenous SLEs	$T_1$ - $T_2$		-.16***	.06	.17***	.13***	.09**	.24***		.10**	-.11	
8. Psychopathology	$T_2$		-.11***	.23***	.62***	.45***	.25***	.28***	.20***			
9. Internalizing Tendencies	$T_2$		-.30***	-.02	.29***	.56***	-.28***	-.06	.08*	.41***		
10. Externalizing Tendencies	$T_2$		.21***	.19***	.19***	-.21***	.49***	.27***	.07*	.35***	-.63***	
11. Severity Marker	$T_2$		-.10**	.24***	.62***	.43***	.27***	.29***	.20***	.99***	.37***	.39***

Note. N= 957 (523 women, 54.6%, coded 0). SLEs= stressful life events;  $T_1$ = baseline,  $T_2$ = follow-up. A gender-stratified table is given in Appendix Table A28. Psychopathology refers to the total problem scores. Significance \*\*\* $p$ < .001, \*\* $p$ < .01, \* $p$ < .05, two-tailed.

Individuals high (vs. low) on frustration reported more endogenous SLEs, but not more exogenous SLEs. More endogenous and exogenous SLEs were predictive of subsequent psychopathology. Psychopathology (total) scores were rather stable between age 16 and 19 ( $r_s$ = .62), and the gender gap narrowed substantially (from  $r_s$ = -.21 to -.11, cf. the first column of Table 21). The internalizing broadband-specific tendencies were

somewhat more stable than the externalizing tendencies between age 16 and 19, as correlations were large and moderate respectively.

### **Stress Generation (H1)**

The ‘frustration model’ in Figure 7 showed that individuals with high (*vs.* low) temperamental frustration reported more endogenous SLEs ( $\Delta X^2_{(1)} = 14.7, p < .001$ ) and more exogenous SLEs ( $\Delta X^2_{(1)} = 3.8, p < .05$ ). Both effects were small, and though effects of frustration on endogenous SLEs appear twice as large as on exogenous SLEs ( $\beta = 0.14$  *vs.*  $0.07$ ), both confidence intervals (in Figure 7) show substantial overlap. We observed no gender differences (all tests  $p \geq .12$ , see Appendix Table A22).

### **Stress Sensitivity (H2)**

The frustration model in Figure 7 indicates that high (*vs.* low) frustration moderated the impact of endogenous ( $\Delta X^2_{(1)} = 28.69, p < .001$ ) and exogenous ( $\Delta X^2_{(1)} = 13.44, p < .001$ ) SLEs on psychopathology, but in opposite directions. Individuals high (*vs.* low) on frustration appear more sensitive to endogenous SLEs, but less sensitive to exogenous SLEs. Based on detailed graphs (Appendix Figure A3–A6) the finding can be interpreted as that individuals scoring high on frustration are already at risk for psychopathology and are not further affected by additional exogenous SLEs (ceiling effect).

### **Two Other Pathways: Overlap (H3) and Direct effects (H4)**

We hypothesized that part of the prospective association between frustration and psychopathology was ‘confounded’ by their overlap at baseline. High (*vs.* low) frustration levels associated concurrently with more psychopathology at baseline ( $\beta = 0.27, \Delta X^2_{(1)} = 56.11, p < .001$ ), and predicted increases in psychopathology between age 16 and 19 ( $\beta = 0.11, \Delta X^2_{(1)} = 14.39, p < .001$ ). A comparison of the betas in the model with and without baseline psychopathology as predictor (Figure 7 and 8) suggests that overlap at baseline explained about half of the prospective association between frustration and psychopathology ( $\beta = 0.21 [\Delta X^2_{(1)} = 33.45, p < .001]$  *vs.*  $\beta = 0.11 [\Delta X^2_{(1)} = 14.39, p < .001]$ ). The significant direct prospective pathway accounted thus for about 40% of the association ( $\beta = 0.11$ , via the diagonal path downward from frustration to psychopathology in Figure 8). An indirect effects model (see Appendix Table A13) supported this perspective, and showed that the full association between frustration and psychopathology at follow-up ( $\beta = 0.26, p < .001, 100\%$ ) could be comparted in a direct effect (44%,  $\beta = 0.11$ ) and an indirect (carry-over) effect via baseline psychopathology (56%,  $\beta = 0.14, p < .001$  [95%CI= 0.11 to 0.18], of which 5% via exogenous SLEs ( $\beta = 0.01, p < .005$  [0.01 to 0.02])). In sum, we observed both overlap and a direct prospective association.

**Direction Markers (H5)**

Results replicated high temperamental frustration as a direct predictor for the severity of psychopathology. We extended upon these findings via separate models for internalizing and externalizing broadband-specific tendencies, which are presented in Table 22. At baseline high frustration overlapped more with externalizing tendencies than with internalizing tendencies (4% vs. 1% explained variance, see Appendix Table A27). Moreover, Table 22 shows that frustration lacked independent prospective effects on change in internalizing tendencies ( $\Delta X^2_{(1)} = 2.82, p = .09$ ), but predicted increases in externalizing tendencies, and this association seemed largely independent from overlap at baseline ( $\beta = 0.09, p < .01, \Delta X^2_{(1)} = 9.13, p < .005$  versus  $\beta = 0.16, p < .001$ , in a model without baseline externalizing). Finally, endogenous SLEs predicted increases in externalizing but not in internalizing tendencies, whereas more exogenous SLEs predicted increases in internalizing but not in externalizing tendencies. We conclude that high frustration is most kindred with externalizing broadband-specific tendencies, but proved uninformative for changes in internalizing tendencies.

**Table 22.** The association between Frustration and Internalizing and Externalizing Direction Markers

Topic	Path	Internalizing Tendencies		Externalizing Tendencies	
		Beta	[95% CI]	Beta	[95% CI]
<b>Overlap</b>	Frustration ↔ Int/Ext $T_1$	0.11 <sup>***</sup>	0.04 to 0.19	0.20 <sup>***</sup>	0.13 to 0.27
<b>Prospective</b>	Frustration → Int/Ext $T_2$			0.09 <sup>**</sup>	0.03 to 0.15
	Int/Ext $T_1$ → Int/Ext $T_2$	0.55 <sup>***</sup>	0.51 to 0.60	0.44 <sup>***</sup>	0.37 to 0.50
<b>Stress Generation</b>	Frustration → Endo SLEs	0.12 <sup>***</sup>	0.05 to 0.19		
	Frustration → Exo SLEs				
	Int/Ext $T_1$ → Endo SLEs			0.26 <sup>***</sup>	0.19 to 0.33
	Int/Ext $T_1$ → Exo SLEs	0.15 <sup>***</sup>	0.09 to 0.21	0.07 <sup>*</sup>	0.01 to 0.14
	Endo SLEs ↔ Exo SLEs	0.24 <sup>***</sup>	0.18 to 0.31	0.23 <sup>***</sup>	0.17 to 0.30
<b>Stress Sensitivity</b>	Endo SLEs → Int/Ext $T_2$			0.19 <sup>***</sup>	0.12 to 0.26
	Exo SLEs → Int/Ext $T_2$	0.15 <sup>**</sup>	0.05 to 0.25		
	Fr*Endo → Int/Ext $T_2$				
	Fr*Exo → Int/Ext $T_2$	-0.17 <sup>**</sup>	-0.06 to -0.27		

*Note.*  $T_1$ = baseline (age 16);  $T_2$ = follow-up wave (age 19); Endo= Endogenous; Exo= Exogenous; SLEs= stressful life events; Int/Ext= internalizing or externalizing tendencies (mutually adjusted, see method section). All paths contributed significantly to the model fit ( $\hat{c} p < .05$  from  $\Delta X^2_{(\Delta f=1)} = 3.84$ ,  $** p < .01$  from 6.64, and  $*** p < .001$  from 10.83), or were constrained to zero. The method section provides information about all variables, model-fit indices, and modelling procedures (details for internalizing tendencies are reported in Appendix Table A14-A16, for externalizing tendencies in Appendix Table A17-A19). The structure of the model is visualized in Figure 8.

### Gender Differences (H7)

At age 16 we observed no gender differences in mean level of temperamental frustration ( $t_{(808)} = 0.67, p = .51$ , boys = 2.73 [SD= 0.57] vs. girls = 2.76 [SD= 0.61]). Gender-stratified models are presented in Table 23. High frustration was not significantly more predictive for internalizing tendencies in girls (vs. boys) and for externalizing tendencies in boys (vs. girls), contrary to our hypotheses.

**Table 23.** Gender Models of change in Psychopathology (total scores), or in Internalizing or Externalizing Direction Markers

Topic	Path	Psychopathology Total score		Internalizing Tendencies		Externalizing Tendencies	
		♀	♂	♀	♂	♀	♂
<b>Overlap</b>	Frustration ↔ Psych $T_1$	0.26***	0.30***	0.10***	0.13***	0.22***	0.19***
<b>Prospective</b>	Frustration → Psych $T_2$	0.10***	0.12***			0.10***	0.10**
	Psych $T_1$ → Psych $T_2$	0.51***	0.44***	0.53***	0.47***	0.43***	0.41***
<b>Stress Generation</b>	Frustration → Endo SLEs			0.11***	0.12***		
	Frustration → Exo SLEs						
	Psych $T_1$ → Endo SLEs	0.25***	0.21***	0.06	-0.18*** ≠	0.16***	0.40*** ≠
	Psych $T_1$ → Exo SLEs	0.16***	0.15***			0.09**	0.12**
	Endo SLEs ↔ Exo SLEs	0.20***	0.24***	0.23***	0.27***	0.21***	0.26***
<b>Stress Sensitivity</b>	Endo SLEs → Psych $T_2$					0.20***	0.18***
	Exo SLEs → Psych $T_2$	0.28***	0.25***	0.14 <sup>†</sup>	0.15 <sup>†</sup>		
	Fr*Endo → Psych $T_2$	0.25***	0.13**	≠ 0.09	-0.15 <sup>†</sup>	≠	
	Fr*Exo → Psych $T_2$	-0.27***	-0.25***	-0.21***	-0.08	≠	

*Note.* ≠ = The beta estimates of the path differed significantly between genders. All paths attributed significantly to the model or were constrained at zero (see Appendix Table A22, A24, and A26, respectively). The method section provides information about all variables and model-fit indices (all details are reported in Appendix Table A20-A26). The structure of the model is visualized in Figure 8.

Significance, \*  $p < .05$ , \*\*  $p < .01$ , and \*\*\*  $p < .001$ , two tailed.

## DISCUSSION

In this study we tested four pathways that may underlay the prospective association between frustration and severity of psychopathology as indexed by total problems: stress generation (H1, mediation), heightened stress sensitivity (H2, moderation), overlap with baseline psychopathology (H3, ‘carry-over’), and an independent (*i.e.* unmediated) prospective effect of frustration (H4). Our results supported all four pathways. We fit models with frustration and SLEs and with or without psychopathology as a predictor at baseline. A comparison of these models suggests that 50% of the prospective effect of frustration on psychopathology was due to overlap at baseline, about 45% was independent, while 5% was mediated and moderated by SLEs. Regard-

ing internalizing and externalizing broadband-specific tendencies (H5), we found that frustration predicted increases in externalizing tendencies, but proved uninformative for change in internalizing tendencies. Finally, endogenous SLEs predicted increases in externalizing (but not internalizing) tendencies, whereas exogenous SLEs predicted increases in internalizing (but not externalizing) tendencies (H6). We observed no gender differences in levels of frustration at age sixteen (H7). After having summarized our main findings, these will be discussed in more detail below.

### **Stress Generation (H1: Mediation)**

The corresponsive principle posits that individuals prone to frustration select themselves into environments that enhance this disposition. We hypothesized (H1) in analogy that individuals high (*vs.* low) on frustration experience more stressful life events (SLEs). This was indeed observed. Moreover, as expected, the effect of high frustration on the ‘active selection’ of more endogenous SLEs was stronger than the more indirect evocative effects on exogenous SLEs ( $\beta = 0.14$  *vs.*  $0.07$ ). Though both effects were small, they exemplify the mutual accommodation between an individual and the environment in which one lives and grows [100]. Frustration predicted 1-5% of the variance in SLE-occurrences, which is negligible from a prevention-perspective, but meaningful given all possible person-environment transactions, and the notion that such effects may accumulate over time [427,892]. Nevertheless, our results dismiss SLEs as an important mediating pathway between high temperamental frustration and later psychopathology.

### **Stress Sensitivity (H2: Moderation)**

We hypothesized that individuals high on temperamental frustration were more sensitive to stress (H2). High (*vs.* low) frustration levels indeed enhanced the effect of endogenous SLEs on psychopathology, in line with a stress-sensitivity mechanism (H2a). Unexpectedly, high (*vs.* low) frustration weakened the average impact of exogenous SLEs on psychopathology (for both genders). To our knowledge this protective effect of frustration after exogenous SLEs has never been reported before, and opposes our hypothesis of higher stress-sensitivity of high (*vs.* low) frustration individuals. The small protective effect requires replication in an independent sample as it may well be a chance finding. However, it could be due to a ceiling effect, *viz.* high frustration scores associated with heightened baseline psychopathology, which might have impeded an increase in symptoms after exogenous SLEs compared to individuals low on frustration, unhindered by this restriction of range.



### Two Other Pathways: Overlap (H3) and Direct Effects (H4)

About 55% of the prospective effect of frustration on psychopathology could be explained by overlap at baseline between these measures (‘a carry-over effect of frustration on future psychopathology via concurrent psychopathology’), in line with H3. About 45% of the prospective effect of high frustration on psychopathology was independent from overlap at baseline, and unmediated by SLEs. This ‘unique’ frustration-driven effect on psychopathology is medium-sized ( $d = \sim 0.35$ ). The mechanisms underlying this direct effect are unclear, as we remain unwitting regarding the kind of factors we are looking for, or how they manifest themselves. We do know that frustration modulates cognitive and emotional processes that color how individuals perceive and construe their environments [22,32], and we speculate that these cognitive processes render them more vulnerable for future psychopathology [32,893].

### Direction Markers

Temperamental frustration predicted the severity of adolescent psychopathology as indexed by total problems. To enhance our understanding of the workings of frustration, we fit the frustration and change model for internalizing and externalizing broadband-specific tendencies separately, the ‘direction markers’ of pathological manifestation. Though this demarcation of variance over internalizing and externalizing tendencies is an artificial theoretical fiction, we felt it might serve heuristic purposes. Direction markers were derived via mutual adjustment of both measures for their shared core, which we argued captures the severity of pathological deviation from normality. Severity showed a strong association with general psychopathology (see Table 22), in keeping with our argument of a ‘severity marker’ [116,894]. This implies that our ‘direction markers’ capture non-pathological behavioral tendencies, arguably more akin to temperament than psychopathology per se, yet indicative for the direction of psychopathological manifestation.

Overlap between frustration and externalizing tendencies at baseline seemed to be larger than with internalizing tendencies (see Appendix Table A29). Previous studies reported that temperamental frustration predicts both internalizing and externalizing psychopathology (see introduction). Our results suggest this to be due to a shared ‘severity marker’, that is, non-specific overlap, while frustration is only predictive for additional externalizing broadband-*specific* tendencies (a direct effect independent from overlap at baseline). These results support the idea that the mechanisms that underlie the direct prospective effect of frustration on psychopathology are kindred to low approach behavior [22,867], alike anger [662,698], rather than in terms of avoidance (*e.g.*, anxiety/fear), as has often been argued [196]. This is in keeping with the idea of frustration as the common emotional response to thwarted goal approach [865-867]. Results did not support the hypothesis that frustration predicts increases

in both broadband-specific tendencies (H5a), but align with H5b; high frustration is most predictive for externalizing-specific tendencies. Some studies in children report similar findings [895].

To conclude, when Burt [896] identified his emotionality dimension in 1930s, he distinguished an ‘active or aggressive conduct’ pole that comprised facets of frustration and anger from a ‘non-assertive negative-reactivity to threat’ pole characterized with feelings of anxiety and fear. Our data can be interpreted in line with Burt’s division in the following way: high temperamental frustration predicted externalizing broadband-specific tendencies, which are sensitive to endogenous SLEs (H2c/H6b), but is not predictive for internalizing broadband-specific tendencies which are sensitive to (often unanticipated) exogenous SLEs (H2c/H6b).

### Gender Effects

The mean level of temperamental frustration was equal for men and women at age sixteen, which is new to the literature. Frustration was not more predictive for increases in internalizing broadband-specific tendencies in women than men (H7a was not supported). Similarly, frustration was not more predictive for externalizing tendencies in men than women (H7b was not supported). We observed some gender differences that may warrant further study (*e.g.*, women reported more exogenous SLEs), but did not support the hypothesized gender-specificity of behavioral styles. We conclude that divergent cultural display rules for the expression of frustration [876] in adolescents could not be substantiated in our models (H7a/H7b); while the few gender effects we did observe were modest at best (see Appendix Table A20-26).

### Limitations

The results of our study should be interpreted in light of the following strengths and limitations. Strengths of this study are our sample of almost thousand adolescents from the general Dutch population in which SLE-occurrences were assessed with Life Stress Interviews. We applied powerful statistical tools to analyze the associations between frustration, SLEs, and psychopathology, which should reliably detect effects from  $d = 0.20$  onwards. The limitation that retrospective self-reports of SLEs inherently incorporate response components that may be influenced by current mental state, such as cognition, appraisal, interpretation and recall, was addressed by our panel (see method section) who rated SLEs independently of the respondent, which forms the current gold standard of life stress research [268,269].

## CONCLUSION

In this study four pathways were identified that underlie the prospective effect of frustration on total psychopathology: stress-generation and stress-sensitivity, a carry-over effect of frustration on future psychopathology via concurrent psychopathology, and an independent direct effect. Our findings suggest that high temperamental frustration, externalizing broadband-specific tendencies, and endogenous SLEs, form a transactional ‘vicious’ cycle between adolescents and the environment they navigate and shape, which can give rise to more psychopathology, and an even deeper ingrained tendency for negative affect [100]. We hope future studies test additional mechanisms that may explain the SLE-unmediated prospective association between frustration and externalizing psychopathology.

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