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Why Does Frustration Predict Psychopathology? Multiple Prospective Pathways Over Adolescence: A TRAILS Study

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Abstract: Adolescents’ temperamental frustration is a developmental precursor of adult neuroticism and psychopathology. Because the mechanisms that underlie the prospective association between adolescents’ high frustration and psychopathology (internalizing/externalizing) have not been studied extensively, we quantified three pathways: stress generation [mediation via selection/evocation of stressful life events (SLEs)], cross-sectional frustration-psychopathology overlap (’carry-over’/common causes), and a direct (non-mediated) vulnerability effect of frustration, including moderation of SLE impact. Frustration and psychopathology were assessed at age 16 with the Early Adolescent Temperament Questionnaire and the Youth Self-Report. No gender differences in frustration were observed. At age 19, psychopathology was reassessed by using the Adult Self-Report, 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, an investigator-based contextual-stressfulness rating procedure (N = 957). Half of the prospective effect of frustration on psychopathology was explained by baseline overlap, including effects of ‘carry-over’ and common causes, about 5% reflected stress generation (a ‘vicious’ cycle with the environment adolescents navigate and shape), and 45% reflected unmediated association: a direct vulnerability effect including stress sensitivity or moderation of SLE impact. After adjustment for their overlap, frustration predicted the development of externalizing but not internalizing symptoms. Copyright © 2016 European Association of Personality Psychology

Key words: negative affect; stress generation; stress sensitivity; approach and avoidance; general factor model

INTRODUCTION

Frustration is defined as irritable distress in response to limitation, exclusion, and failure. Dispositional or temperamental frustration generalizes across contexts and can have pervasive social consequences, as it colours social relationships, social interactions, and job performances (Ekman, 2007; Kuppens & Van Mechelen, 2007; Laceulle, Jeronimus, Van Aken, & Ormel, 2015; Spector, 1978). Dispositional frustration is also prospectively associated with both internalizing and externalizing psychopathologies throughout childhood and early adolescence (Eisenberg et al., 2001; Laceulle, Ormel, Vollebergh, van Aken, & Nederhof, 2014; Lengua, 2003; Muris & Ollendick, 2005; Oldehinkel, Hartman, De Winter, Veenstra, & Ormel, 2004; Ormel et al., 2005) and in adulthood (Jeronimus, Kotov, Riese, & Ormel, 2016) and antedates vandalism and violence (Dollard, Doob, Miller, Mowrer, & Sears, 1939; Munyo & Rossi, 2013).

Previous studies established a robust prospective effect of high temperamental frustration (henceforth “frustration”) on later psychopathology, but the mechanisms that underlie this association have not been studied extensively. Surprisingly, provided that adolescent frustration proves to be a developmental precursor of adult neuroticism (Oldehinkel et al., 2004; Putnam, Ellis, & Rothbart, 2001), which is the strongest prospective vulnerability factor for development of psychopathology (Jeronimus et al., 2016; Ormel et al., 2013), and is associated with immense public health costs (Cuijpers et al., 2010). In the present study, we investigate pathways that may channel the prospective association between dispositional frustration and psychopathology (defined as total psychological problem score): a direct effect of frustration, stress generation, and overlap with concurrent psychopathology.

Prospective association (main effect)

The total prospective association between baseline frustration at age 16 (t1) and psychopathology at age 19 (t2) is our main effect. In this paper, we disentangle this association to identify the direct vulnerability effect of frustration, which we defined as unmediated by major stressful life events (SLEs) and unconfounded by concurrent psychological problems at (t1), but including heightened stress reactivity (evident as moderation of SLE impact). We provide a rationale and test for each of these pathways in the succeeding texts. Additionally, to enhance our understanding of this total
prospective association between frustration and psychopathology, we consider these pathways also for internalizing and externalizing symptoms separately and test whether these pathways differ by gender.

**Stress generation (mediation)**

Frustration proneness permeates the world in which adolescents live and grow via the selection of contexts that they choose to enter and avoid, the friends and mates they select, and the reactions they evoke from their social world (Buss, 1987; Caspi, Roberts, & Shiner, 2005). Temperamental frustration also taps into patterns of information processing, including perception and interpretation of the internal (self) and external world, and causality attributions (Chan, Goodwin, & Harmer, 2007; Dodge, 1980; Finn, Mitte, & Neyer, 2013; Harris, 2009; Kuppens & Van Mechelen, 2007; Lench, Bench, Darbor, & Moore, 2015). It has been shown that adolescents prone to frustration report perceptual alterations (such as lower perceived relationship affection), which increases their probability to experience subsequent stressful social events (Laceulle et al., 2015).

And more SLEs, in turn, predict both an increase in frustration (Laceulle et al., 2015) and a heightened probability of developing internalizing problems (Ormel et al., 2005, 2013). Hence, we hypothesize that part of the prospective effect of baseline frustration ($t_1$) on subsequent disordered psychological and behavioural functioning ($t_2$) is mediated by an increased probability of SLEs ($H_1$, mediation), driven by a frustration-related change in perception and behaviour (e.g. Finn, Mitte, & Neyer, 2015; Laceulle, Jeronimus et al., 2015).

The impact of SLEs on personality and psychopathology has been shown to differ according to their perceived controllability (e.g. Brown & Harris, 1989; Jeronimus, Riese, Sanderman, & Ormel, 2014; Tennen & Affleck, 1990). SLEs can be subdivided over endogenous SLEs, defined as likely to be a consequence of one’s own behaviour (i.e. controllable, such as dismissal due to stealing, conflict with a friend, or decision to marry), versus exogenous SLEs, defined as not directly brought about by an individual’s own behaviour (i.e. uncontrollable, such as being hit by lightning or the sickness of a parent). We hypothesized that frustration increased not only the probability of endogenous SLEs ($H_{1a}$, selection effect, $F_{t+1} \rightarrow \text{EndoSLE}_t$) but also the probability of exogenous SLEs ($H_{1b}$, exposure effect, $F_{t+1} \rightarrow \text{ExoSLE}_t$). This exposure effect may index confounders that affect both frustration and the probability of exogenous SLEs, such as low socioeconomic status (SES) and neighbourhood effects (Sampson, Morenoff, & Gannon-Rowley, 2002; Tolsma, van der Meer, & Gesthuizen, 2009), and indirect effects of frustration, such as effects of social network characteristics or reputation (Harris, 2009).

Because the definition of endogenous and exogenous SLEs revolves around controllability, we expected frustration to be most predictive for endogenous SLEs [$H_{1c}$, $F_{t+1} \rightarrow (\text{EndoSLE}_t > \text{ExoSLE}_t)$].

**Frustration-psychopathology overlap (confounding)**

One of the strongest prospective pathways between frustration and later psychopathology is probably overlap between these measures at baseline, which results from three sources: (i) common causes, (ii) correlation due to earlier bidirectional effects, and (iii) operational (measurement) overlap (see Jeronimus et al., 2016; Ormel et al., 2013). 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. Moreover, from the comparison of both models, we can also deduce whether the ‘stress generation effect’ holds after adjustment for baseline psychopathology.

**Direct effects of frustration (stress sensitivity)**

A comparison of the estimates in the models with and without baseline psychopathology also enables us to quantify the independent or ‘direct’ prospective association of frustration on psychopathology, which is neither due to overlap with baseline psychopathology nor mediated by SLEs [$H_{2a}$, direct effect: $F_{t+1} \rightarrow P_{t+2}$]. This ‘direct’ effect captures processes that are driven by frustration and which may lead to psychopathology, such as a negative bias in attention, interpretation and recall of information, increased reactivity (moderation), or ineffective coping (Laceulle, Jeronimus et al., 2015; Ormel et al., 2013). For example, high frustration is known to enhance the impact of negative environmental influences on psychopathology, such as low SES, family instability, negative parenting styles, and divorce (Kiff, Lengua, & Zalewski, 2011; Lengua, 2008; Oldehinkel, Veenstra, Ormel, De Winter, & Verhulst, 2006; Rothbart & Bates, 2007). Drawing on this moderation effect, we hypothesized that high frustration also increases the impact of SLEs on subsequent psychological problems [$H_{2b}$, vulnerability effect, $F_{t+1} \times \text{SLE}_t \rightarrow P_{t+2}$].

**Internalizing and externalizing tendencies**

Frustration associates with an increased probability of developing internalizing and externalizing symptoms in childhood and early adolescence (Laceulle, Ormel et al., 2014; Oldehinkel et al., 2004; Ormel et al., 2005) and adulthood (Jeronimus et al., 2016). Internalizing (emotional problem) and externalizing (behavioural problem) symptoms cluster into empirically and conceptually distinct domains, which correlate about $r=0.50$, a comorbidity which for long has been interpreted as the natural organization of psychopathology (Krueger & Markon, 2006). Recent factor-analytic studies, however, support a three bi-factor structure of psychopathology, in which a ‘general factor’ of psychopathology is accompanied by two more specific higher-order internalizing and externalizing factors, which capture additional shared variance in symptoms. This three-factor structure has been observed not only in childhood and adolescence (Caspi et al., 2013; Laceulle, Vollebergh, & Ormel, 2014; Tackett et al., 2013) but also in adulthood (Kotov et al., 2011; Lahey et al., 2012) and fits the data better.
than all other known structures (Laceulle et al., 2014; Lahey et al., 2012).

In this three-factor model, the internalizing and externalizing factors (InT/ExT) capture independent and inversely associated tendencies because their overlap is subsumed by the general factor (distress or negative-emotional dysregulation). In the present paper, we test whether frustration also predicts these internalizing and externalizing factors, next to the known affinity between frustration and the general factor (Ormel et al., 2005; Tackett et al., 2013). We hypothesize that as a general rule, frustration shall be more predictive for change in externalizing than internalizing symptoms [H3a, \(F(t1) \rightarrow (\Delta ExT[t_{1-2}] > \Delta InT[t_{1-2}])\], as has often been reported (e.g. Dollard et al., 1939; Munyo & Rossi, 2013; Muris, Meesters, & Blijlevens, 2007).

**Response strategies**

In the Introduction, we defined temperamental frustration as an increased sensitivity to thwarted goal approach, threat, novelty, and unexpected non-reward (i.e. an expectation violation in which reality is worse than expected), with a perceived obstruction by an (intentional) antagonistic act as the most potent contextual trigger (Amsel, 1992; Berkowitz, 1989; Carver, 2004; Carver & Harmon-Jones, 2009; Krieglmeyer, 2007; Munyo & Rossi, 2013; Panksepp & Biven, 2012; Putnam et al., 2001). Previous work showed that frustration becomes manifested as heightened emotional tension or ‘arousal’ (distress), which in turn motivates one of two interpersonal negotiating tactics (approach or avoidance), conditional on perceived control over the antagonist, which incorporates personal and contextual factors (Berkowitz, 1988; Carver, 2004; Carver & Harmon-Jones, 2009; Harmon-Jones, 2003; Lewis, Sullivan, Ramsay, & Alessandri, 1992; Sell, Tooby, & Cosmides, 2009).

Frustration is thus most likely to be triggered by emotional loaded situations that involve a barrier or obstruction (and temperamental frustration indexes the probability thereof), and frustration results in heightened arousal that drives an approach or avoidance response, conditional on properties of the given context (Berkowitz, 1988; Buss, Haselton, Shackelford, Bleske, & Wakefield, 1998; Lench et al., 2015; Panksepp & Biven, 2012; Sell et al., 2009; Shorkey & Crocker, 1981). From a functional perspective, frustration should facilitate approach tendencies when the antagonist is deemed controllable and the goal perceived as attainable (e.g. inflicting costs via anger) to overcome obstacles and persist in goal approach. However, frustration should facilitate avoidance when the antagonist is appraised as uncontrollable (withdrawal, via fear or anxiety) or low approach when the goal is perceived as unattainable (downregulation of expected benefits via sadness), which are all internalizing tendencies (Carver, 2004; Carver & Harmon-Jones, 2009; Ellsworth & Smith, 1988; Lench et al., 2015; Sell et al., 2009; Stossel, 2013).

In the previous paragraph, we postulated our overarching hypothesis (H3a) that frustration shall be more predictive for changes in externalizing than internalizing symptoms. However, drawing on the definition of endogenous SLEs as (more) controllable than exogenous SLEs, and provided that people tend to appraise endogenous events to be due to a human agent (cf. Berkowitz, 1988; Ellsworth & Smith, 1988; Mandel & Lehman, 1996; Wright, Zautra, & Braver, 1985) and tend to externalize blame for bad outcomes (see Tennen & Affleck, 1990), our functional argument asserts that adolescents are more likely to develop externalizing symptoms in response to endogenous SLEs [H3b, EndoSLEs \(\rightarrow ExT_{t2}\)] and internalizing symptoms in response to exogenous SLEs [H3c, ExoSLEs \(\rightarrow InT_{t2}\)], next to their associations with the general factor (distress).

Taken together, pertaining response strategies, we hypothesized that adolescents high on frustration (H1c) tend to experience more endogenous SLEs (H2b) to which they are most likely to respond with externalizing problems (moderation), in keeping with our overarching expectation (H3a) that high frustration is most predictive for changes in externalizing rather than internalizing symptoms. Additionally, we hypothesized that endogenous SLEs are most likely to elicit externalizing problems (H3b), whereas exogenous SLEs are most likely to elicit internalizing problems (H3c).

**Gender differences**

Gender is an essential part of our identity (Martin, 2013). Although no gender differences in frustration have been observed in childhood (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006), earlier studies in our Tracking Adolescents’ Individual Lives Survey (TRAILS) sample reported on lower frustration levels for girls than for boys in early adolescence (Oldenhinkel et al., 2006). Subsequently, and new to the literature, we examined whether levels of frustration differ by gender at age 16. Moreover, equal frustration levels do not impede cultural display rules that render assertive action (such as approach responses) and outward expression of frustration less appropriate for girls than boys (Archer, 2004; Chaplin & Aldao, 2013; Condry & Condry, 1976; Wiesner-Hanks, 2006). Such highly gendered behavioural styles align with the observation that girls score higher on internalizing symptoms (e.g. anxiety/sadness) and boys on externalizing symptoms (e.g. anger/aggression) from childhood to young adulthood (Archer, 2004; Caspi et al., 2005; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Oldenhinkel et al., 2004; Ormel et al., 2005). Although frustration conventionally predicts both internalizing and externalizing factors (Lengua, 2003; Oldenhinkel et al., 2004), we hypothesized that cultural display rules may manifest in terms of stronger associations between frustration and internalizing responses in girls [H4a, G \(\ast F(t1) \rightarrow (\Delta InT[t_{1-2}] > \Delta ExT[t_{1-2}])\)] and externalizing responses in boys [H4b, B \(\ast F(t1) \rightarrow (\Delta ExT[t_{1-2}] > \Delta InT[t_{1-2}])\)], next to the association between frustration and the general factor (Ormel et al., 2005; Tackett et al., 2013).

**Summary**

To recapitulate, three sets of hypotheses were postulated to explain the association between frustration and later psychopathology:
(1) Adolescents high on frustration experience more endogenous SLEs (H1a, selection) and exogenous SLEs (H1b, evocation/exposure), but the former is strongest (H1c).

(2) Part of the prospective effect is due to overlap with psychopathology at baseline (including ‘carry-over’ and common causes), while part of the prospective effect of frustration is a ‘direct effect’ (H2a). This direct effect is unmediated by SLEs and not due to overlap with psychopathology but includes heightened stress sensitivity, evident as moderation of the SLE impact on subsequent psychopathology (H2b).

(3) The ‘direct’ effect of high frustration on later developing psychopathology is largest for externalizing problems (H3a). Additionally, we hypothesized the direct effect of frustration on the internalizing factor to be relatively larger in girls (H4a) and the effect on the externalizing factor to be relatively larger in boys (H4b). Furthermore, the vulnerability effect of high frustration was hypothesized to increase the probability that adolescents reported externalizing problems after endogenous SLEs (H3b) and internalizing problems after exogenous SLEs (H3c), next to their association with the general factor (distress).

Finally, we fit a model adjusted for gender and family SES because previous work suggested an effect of SES on the manifestation of temperamental frustration (Jeronimus, Stavrakakis, Veenstra, & Oldehinkel, 2015).

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 (Huisman et al., 2008; Ormel et al., 2012). 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; De Winter et al., 2005]. The response rates of the third and fourth waves 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 waves as ‘age 16’ (t1) and ‘age 19’ (t2) respectively. The Dutch Central Committee on Research Involving Human Subjects approved the study. The participants were treated in compliance with American Psychological Association ethical standards, and all measurements were carried out with their adequate understanding and written consent. No different ethnicities were represented.

Stressful life events in the interval between these two waves were assessed by using an investigator-based procedure yielding contextual stressfulness rating. During the wave at age 19, we administered the Life Stress Interview (LSI; Kendler, Kessler, Walters, & MacLean, 1995), 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 Diagnostic and Statistical Manual, Fourth Edition (DSM-IV) criteria for a lifetime psychiatric disorder (Ormel, 2015). 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 Diagnostic and Statistical Manual, Fourth Edition 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 with the whole sample, LSI respondents were more often women (55% vs 50%; χ² = 4.5, p = .04) and younger [mean age 19.0, SD = 0.6 vs 19.2, SD = 0.6; t(df = 1879) = 6.3, p < .01].

Measures

Frustration
Temperamental frustration was assessed at age 16 with the Dutch (Hartman, 2000) short form of the parent version of the Early Adolescent Temperament Questionnaire-Revised (Putnam et al. (2001), five items, α = .75). The Early Adolescent Temperament Questionnaire is grounded in Rothbart’s temperament model (Rothbart, 2011; Rothbart, Ahadi, & Evans, 2000). We used the parent version because in the TRAILS sample, the factor structure of this version was superior to that of the child version (Oldehinkel et al., 2004). An additional benefit is that this prevents shared rater variance between frustration and our internalizing and externalizing symptom measures. The frustration items read: ‘My child ‘is annoyed by little things other kids do’, ‘gets very irritated when someone criticizes her/him’, ‘gets irritated when I will not take her/him some place s/he wants to go’, ‘gets irritated when s/he has to stop doing something s/he is enjoying’, and ‘hates it when people don’t agree with him/her’, which are scored on a five-point scale ranging from ‘almost never untrue’ (1) to ‘almost always true’ (5). The core of the scale thus captures irritability and frustration proneness.

Internalizing and externalizing symptoms
At age 16 (t1), mental health was assessed with the Youth Self-Report (YSR; Achenbach, 1991) and at age 19 (t2) with the Adult Self-Report (ASR; Achenbach & Rescorla, 2003). Achenbach constructed both instruments to standardize
assessment from childhood to adulthood (Achenbach, 1991), and both instruments assess behavioural and emotional problems in the preceding 6 months. All items rated as not true (0), somewhat or sometimes true (1), or very true or often true (2). We computed (i) a total problem score; (ii) an internalizing domain score, a composite of anxious/depressed \((k = 13, \alpha = 0.84)\) at age 16; \(k = 18, \alpha = 0.91)\) at age 19, e.g. cries a lot, fears, and feels unloved, nervous, or tense) and withdrawn-depressed \((k = 8, \alpha = 0.74)\); \(k = 9, \alpha = 0.76)\, e.g. ‘there is very little that I enjoy’, rather alone, sad, and refuses to talk); and (iii) an externalizing domain score, a composite of rule breaking \((k = 15, \alpha = 0.76)\); \(k = 14, \alpha = 0.77)\, e.g. ‘I set fires’, lies, steals at home, is truant, uses drugs, or lacks guilt) and aggression \((k = 17, \alpha = 0.81)\); \(k = 15, \alpha = 0.85)\, e.g. ‘I attack people physically’, argues a lot, is disobedient at home or at school, screams, feels suspicious, and threatens others; the somatic problems and attention deficit hyperactivity disorder scales are not used). The reliability and validity of American versions have been confirmed for the Dutch versions (Verhulst, Van der Ende, & Koot, 1997), and previous psychometric studies in Dutch samples showed that the YSR factor structure could be applied to ASR scores (Ferdinand, Verhulst, & Wiznitzer, 1995).

**Life events**

Stressful life events were assessed with Kendler et al. (1995) LSI, which was based on the Life Events and Difficulties Schedule by Brown and Harris (1989). The LSI encompasses 11 personal events, which are events occurring primarily to the respondents themselves, among which are 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 the 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 behaviours evoked by the event (Dohrenwend, 2006; Monroe, 2008). The events were rated on severity (i.e. long-term contextual threat) and dependence on the respondent’s own will or behaviour (i.e. planned actions or events caused by neglect or carelessness). Dependent events were called endogenous SLEs, while events that were deemed to lie outside the influence of the respondent were 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 2 years, separately for endogenous and exogenous events.

**Socioeconomic status**

Socioeconomic status of the family of origin was the composite of five z-scored continuous variables, including professional occupation and educational attainment of both parents/guardians and household income.

**Statistical analysis**

We performed data cleaning steps and calculated descriptives in SPSS (version 20, SPSS Inc, Chicago, IL). Endogenous SLEs were skewed to the right, and both endogenous and exogenous SLEs were kurtotic. Associations between variables were therefore expressed in terms of Spearman rho \((r_s)\) and Spearman partial rho \((r_{sp})\) coefficients (Bishara & Hittner, 2012). Gender differences were tested with bootstrapped t-tests \((k = 10000)\, two-tailed significance, \(H_0: b = 0)\) and mean-level differences with ANOVA (bootstrapped, \(k = 10000\)). Standardized beta weights \((\beta)\) report the change in outcome per SD change in a predictor and quantify the strength of the direct effect of one variable on the other in the context of multiple comparisons (Geiser, 2013). We classified correlations \((r)\) and betas as small if between .10 and .20, moderate between .20 and .30, and large if above .30 (Peterson & Brown, 2005; Richard, Bond, & Stokes-Zoota, 2003).

Our hypotheses were tested with a system of multiple regression equations specified in structural equation models (SEMs) in Mplus 7.11 software (Muthen & Muthen, 1999). We applied Maximum Likelihood estimations with Robust standard errors (MLR) to account for the non-normality of the data (Asparouhov & Muthén, 2012; Bryant & Satorra, 2012). MLR uses full maximum likelihood, which, in this context, is superior over multiple imputation (Allison, 2012). We tested all paths for their contribution to the fit of a model in terms of significant change in Akaike and Bayesian information criteria (see Burnham & Anderson, 2004). 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, see Table 3). For our gender models, we also tested all paths for gender equality and were possible constrained paths to be equal for boys and girls.

A latent variable approach would adjust for part of the measurement error in frustration and psychopathology but cannot do this for the endogenous and exogenous SLE measures (because many SLEs are stochastically independent). Therefore, a conservative observed-variable approach was applied, which enables for a more reliable and fair comparison of the life event and frustration effects. The 957
participants enabled a free parameter-to-sample ratio above 1:50 in all models tested (Bentler & Chou, 1987) and a reasonably precise estimation of local effects beyond Cohen’s $f^2 = 0.013$ in our most complex models ($R^2 > 0.01$, $r = 0.10$, $d = 0.20$), given 80% power, five predictors, and $\alpha = 0.05$ (Selya, Rose, Dierker, Hedeker, & Mermelstein, 2012).

The small number of degrees of freedom ($df \leq 17$ to 23) combined with seven variables $[k(k+1)/2] = 27$ known values suffices for reliable model identification but not for reliable secondary fit indices (e.g., Root Mean Square Error of Approximation) which in this situation often falsely indicate a poor fit for correctly specified models (see Barrett, 2007; Geiser, 2013; Kenny, Kaniskan, & McCoach, 2015).

It has therefore been advised not to calculate secondary fit indices but to check the robustness of the model by testing all possible paths to see whether they improve fit (Kenny et al., 2015). The results suggest that our models were correctly specified, whereas the statistically insignificant model $\chi^2$ values indicate that the models reproduced the data rather closely. Additionally, we have fitted comparable multiple linear regression models in SPSS (version 20, SPSS Inc., Chicago, IL) and calculated partial Spearman rho coefficients.

Because these models supported the same conclusions, only the final SEM models are reported. Finally, we fit a model adjusted for gender and SES of the family of origin.

### Internalizing and externalizing factors

To derive our three-factor internalizing and externalizing factors, we fit the linear regression analyses of concurrent measurements on one another at both waves, analogous to the calculation of partial $r_t$. We used the mutual adjusted residuals, from which the ‘general factor’ had been subtracted, as internalizing and externalizing factors in SEM. In other words, all overlap with externalizing problems was removed from our internalizing factor, whereas all overlap with internalizing problems was removed from our externalizing factor. The removed overlap from both measures is called the general factor. All symptoms together or the total problem score is henceforth referred to as psychopathology.

### RESULTS

#### Descriptives

We present our descriptives in Table 1. The participants of both genders reported an equal number of endogenous SLEs [$t_{505} = 0.33$, $p = .75$, men $\bar{x} = 3.4$ vs women $\bar{x} = 3.3$], but women reported more exogenous SLEs [$t_{505} = 4.9$, $p < .001$, $\bar{x} = 6.9$ vs $5.3$, $r_1 = .16$, $d = 0.32$]. The associations between the studied variables are presented in Table 2. Frustration-prone adolescents (at $t_1$) associated with concurrent psychopathology ($t_1$) and predicted psychopathology at follow-up ($t_2$). Frustration-prone adolescents (at $t_1$) reported more endogenous SLEs, but not more exogenous SLEs at $t_1$. More endogenous and exogenous SLEs associated with subsequent psychopathology (at $t_2$). Psychological symptom scores were rather stable between ages 16 and 19 ($t_{1,2} = .62$), and the gender gap narrowed by half (from $r_1 = -.21$ to -.11, as can be seen in the first column of Table 2). The three-factor internalizing factor was somewhat more stable than the

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### Table 1. Descriptives of the study variables

<table>
<thead>
<tr>
<th>Wave</th>
<th>N</th>
<th>Range</th>
<th>Mean (SD)</th>
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<td></td>
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<td>Frustation</td>
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<td>1.00 to 4.80</td>
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<td>0.00 to 2.37</td>
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<td>$T_1$</td>
<td>866</td>
<td>-0.53 to 1.24</td>
</tr>
<tr>
<td>Externalizing</td>
<td>$T_1$</td>
<td>866</td>
<td>-0.38 to 0.85</td>
</tr>
<tr>
<td>Age (in years)</td>
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<td>957</td>
<td>17.99 to 20.84</td>
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<tr>
<td>Endogenous SLEs</td>
<td>$T_1-T_2$</td>
<td>957</td>
<td>0.00 to 26.00</td>
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<tr>
<td>Exogenous SLEs</td>
<td>$T_1-T_2$</td>
<td>957</td>
<td>0.00 to 35.00</td>
</tr>
<tr>
<td>Psychopathology</td>
<td>$T_2$</td>
<td>942</td>
<td>0.00 to 2.41</td>
</tr>
<tr>
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<td>$T_2$</td>
<td>942</td>
<td>-0.39 to 0.72</td>
</tr>
<tr>
<td>Externalizing</td>
<td>$T_2$</td>
<td>942</td>
<td>-0.51 to 0.75</td>
</tr>
</tbody>
</table>

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 Methods section. SD, standard deviation; SE, standard error; N, sample size; SLEs, stressful life events; $T_1$, baseline (age 16); $T_2$, follow-up (age 19).

### Table 2. Spearman correlations between all variables ($r_t$) in the lower half

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
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<tbody>
<tr>
<td>1 Gender</td>
<td></td>
<td>-.42***</td>
<td>.19***</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2 Frustation</td>
<td>$T_1$</td>
<td>-.01</td>
<td>.16**</td>
<td>.19***</td>
<td>.06</td>
<td>-08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Psychopathology</td>
<td>$T_1$</td>
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<td>.26***</td>
<td>.62**</td>
<td>.48***</td>
<td></td>
<td></td>
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<td>4 Internalizing symptoms</td>
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<td>.03</td>
<td>.13***</td>
<td>.45***</td>
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<td>-21***</td>
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<tr>
<td>5 Externalizing symptoms</td>
<td>$T_1$</td>
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<td>.76***</td>
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<td>.09***</td>
<td>.25***</td>
<td>-.09***</td>
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<td>6 Endogenous SLEs</td>
<td>$T_1-T_2$</td>
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<td>-.10**</td>
<td>.23***</td>
<td>.12***</td>
<td>.28***</td>
<td></td>
<td></td>
<td></td>
<td>-.06</td>
</tr>
<tr>
<td>7 Exogenous SLEs</td>
<td>$T_1-T_2$</td>
<td>-.16***</td>
<td>.06</td>
<td>.17***</td>
<td>.16***</td>
<td>.13***</td>
<td>.24***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 Psychopathology</td>
<td>$T_2$</td>
<td>-.11***</td>
<td>.23***</td>
<td>.62***</td>
<td>.58***</td>
<td>.44***</td>
<td>.28***</td>
<td>.20***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 Internalizing symptoms</td>
<td>$T_2$</td>
<td>-.22***</td>
<td>-.16***</td>
<td>.57***</td>
<td>.63***</td>
<td>.28***</td>
<td>.18***</td>
<td>.18***</td>
<td>.90***</td>
<td>.74***</td>
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<td>10 Externalizing symptoms</td>
<td>$T_2$</td>
<td>.03</td>
<td>.26***</td>
<td>.53***</td>
<td>.38***</td>
<td>.54***</td>
<td>.27***</td>
<td>.07*</td>
<td>.87***</td>
<td>.59***</td>
</tr>
<tr>
<td>11 General factor</td>
<td>$T_2$</td>
<td>-.10**</td>
<td>.24***</td>
<td>.62***</td>
<td>.43***</td>
<td>.27***</td>
<td>.29***</td>
<td>.20***</td>
<td>.99***</td>
<td>.37***</td>
</tr>
</tbody>
</table>

Note: The upper half of the table shows partial $r_t$ with the internalizing (InT) and externalizing (ExT) factors after removal of their overlap (distress, see the Methods section for details).

$N = 957$ (523 women, 54.6%, coded 0). SLEs, stressful life events; $T_1$, baseline (age 16); $T_2$, follow-up (age 19). Psychopathology refers to the total problem scores. A gender-stratified table is given in Table S19.

***$p < .001$, **$p < .01$, *$p < .05$, two-tailed.

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externalizing factor between ages 16 and 19 ($t_{1-2}$, $r_s = .56$ vs .44, upper panel).

**Modelling**

All modelling details are provided in the supplement, but the key differences in fit between baseline models and the final models are presented in Table 3.

**Stress generation (H1)**

The ‘frustration model’ in Figure 1 showed that adolescents prone to frustration (at $t_1$) reported more endogenous SLEs ($\Delta \chi^2(1) = 14.7, \ p < .001, \ H1a$) and more exogenous SLEs ($\Delta \chi^2(1) = 3.8, \ p < .05, \ H1b$). Both effects were small, and though the effects of frustration on endogenous SLEs appear to be twice as large as on exogenous SLEs ($\beta = 0.14$ vs 0.07, $H1c$), both CIs showed substantial overlap (Figure 1). We observed no gender differences (all tests $p \geq .18$, Table S13).

The stress generation effect of frustration (at $t_1$) on SLEs disappeared after adjustment for baseline psychopathology (Figure 2). This indicates that concurrent psychological problems (at $t_1$) rather than temperament (at $t_1$) accounted for most stress generation. These results were robust to adjustment for SES and gender (Figure 3). Hence, the stress generation effect of frustration was confounded by concurrent psychological problems (Figure 4).

**Frustration-psychopathology overlap**

We hypothesized that part of the prospective association between frustration (at $t_1$) and psychopathology (at $t_2$) was ‘confounded’ by their overlap at baseline. High frustration levels (at $t_1$) associated concurrently with more psychopathology [at $t_1$, $\beta = 0.27$, $\Delta \chi^2(1) = 56.11, \ p < .001$, Figure 4] and predicted increases in psychopathology between ages 16 and 19 [$t_{1-2}$, $\beta = 0.11$, $\Delta \chi^2(1) = 14.39, \ p < .001$]. A comparison of the betas in the model with and without baseline psychopathology as predictor (Figures 1 and 2) suggests that overlap at baseline explained about half of the prospective association between frustration (at $t_1$) and psychopathology [at $t_2$, $\beta = 0.21$ ($\Delta \chi^2(1) = 33.45, \ p < .001$) vs $\beta = 0.11$ ($\Delta \chi^2(1) = 14.39$, $p < .001$)].

---

Table 3. Key differences in fit between the baseline models and the final models

<table>
<thead>
<tr>
<th>Models</th>
<th>Results</th>
<th>$\Delta \chi^2$</th>
<th>$\Delta df$</th>
<th>$p$</th>
<th>$\Delta$BIC</th>
<th>$\Delta$AIC</th>
<th>Supp. details</th>
</tr>
</thead>
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<tr>
<td>Frustration model</td>
<td>Figure 1</td>
<td>0.74</td>
<td>1</td>
<td>.40</td>
<td>5.7</td>
<td>0.8</td>
<td>Tables S1 to S4</td>
</tr>
<tr>
<td>Psychopathology model</td>
<td>Figure 2</td>
<td>3.56</td>
<td>3</td>
<td>.31</td>
<td>16.8</td>
<td>2.2</td>
<td>Tables S1 to S4</td>
</tr>
<tr>
<td>Gender and SES adjusted model</td>
<td>Figure 3</td>
<td>1.52</td>
<td>5</td>
<td>.91</td>
<td>25.6</td>
<td>2.2</td>
<td>Tables S20 and S21</td>
</tr>
<tr>
<td>Internalizing model</td>
<td>Table 4</td>
<td>7.22</td>
<td>5</td>
<td>.21</td>
<td>26.2</td>
<td>2.6</td>
<td>Tables S6 to S8</td>
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<tr>
<td>Externalizing model</td>
<td>Table 4</td>
<td>4.59</td>
<td>5</td>
<td>.47</td>
<td>28.5</td>
<td>4.2</td>
<td>Tables S9 to S11</td>
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<tr>
<td>Gender psychopathology model</td>
<td>Table 5</td>
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<td>Tables S12 to S14</td>
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<td>Gender internalizing model</td>
<td>Table 5</td>
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<td>.20</td>
<td>69.5</td>
<td>6.3</td>
<td>Tables S15 and S16</td>
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<tr>
<td>Gender externalizing model</td>
<td>Table 5</td>
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<td>16</td>
<td>.38</td>
<td>90.2</td>
<td>12.4</td>
<td>Tables S17 and S18</td>
</tr>
</tbody>
</table>

Note: SES, socioeconomic status; $\chi^2$, chi-squared; $df$, degrees of freedom; AIC, Akaike information criteria; BIC, Bayesian information criteria; $\Delta$, change.

---

Figure 1. Frustration model.

Note. $N=810$. 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 supplement Table S1 for model fit and Tables S3 and S4 for tests of all paths). Significance ***$p<.001$, **$p<.01$, *$p<.05$
In sum, we observed that baseline overlap between frustration and psychopathology (at $t_1$) explained a substantial part of the prospective association with psychopathology (at $t_2$).

**Direct effects of frustration (stress sensitivity)**

A comparison of the betas in the model with and without baseline psychopathology as predictor (Figures 1 and 2) suggests that the significant direct prospective pathway, via the diagonal path downwards from frustration at $t_1$ to psychopathology at $t_2$ in Figure 2, accounted for about 40% of the association ($H2a$, $\beta=0.11$). An indirect effect model supported this perspective and showed that the full association between baseline frustration and psychopathology at follow-up ($\beta=0.26$, $p<.001$, 100%) could be compared in direct (44%, $\beta=0.11$) and indirect (carry-over) effects via baseline psychopathology ($56%$, $\beta=0.14$, $p<.001$ (95% confidence interval [CI] = 0.11 to 0.18)), of which 5% are from exogenous SLEs ($\beta=0.01$, $p<.005$ (0.01 to 0.02), Table S5). Notably, there was no reverse effect (namely $p<.001$)].

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*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 and the modeling details are given in the method section (see supplement Table S1 for model fit and Tables S3-S4 for tests of all paths). All path coefficients were significant at $^{***}p<.001$

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Figure 2. Change in psychopathology model.

Figure 3. Adjusted psychopathology model.
indirect prospective effect of baseline psychopathology via baseline frustration). The model adjusted for gender and SES in Figure 3 shows similar results.

With regard to stress sensitivity, the model in Figure 1 indicates that high (vs low) frustration at baseline moderated the impact of endogenous SLEs \( \Delta \chi^2(1) = 28.69, p < .001 \) and exogenous SLEs \( \Delta \chi^2(1) = 13.44, p < .001 \) on psychopathology (at \( t_2 \)). High baseline frustration associated with a stronger impact of endogenous SLEs on psychopathology (at \( t_2 \)) but a weaker impact of exogenous SLEs on psychopathology (at \( t_2 \)). However, detailed graphs (Figures 4 and 5 and Figures S1 and S4) showed a smooth linear increase in psychopathology for higher levels of frustration and endogenous SLEs (Figure 5), but the results were less clear for low and medium levels of exogenous SLEs. Moreover, the group with high levels of exogenous SLEs was very small (range 0–36, 90% of the 956 participants scored <12, and only 36 adolescents scored in the highest half of the scale); thus, a larger or more disadvantaged population is needed for firmer conclusions.

Despite that baseline frustration was no longer predictive of SLEs after adjustment for baseline psychopathology (Figure 2, no stress generation), adolescents prone to frustration at baseline were still more likely to develop psychopathology (at \( t_2 \)) after experiencing SLEs (primarily in terms of the general factor, as the effects for InT/ExT were weak, Table 5). In sum, we observed a substantial ‘direct’ prospective association for frustration (H2a) and evidence that adolescents high on frustration are vulnerable to develop psychopathology after endogenous SLEs (H2b, stress sensitivity), irrespective of their baseline psychopathology levels.

Three-factor model: internalizing and externalizing factor

Our models replicated high temperamental frustration at age 16 (\( t_1 \)) as a direct predictor for the severity of psychopathology at age 19 (\( t_2 \)). We extended upon these findings via separate models for the internalizing and externalizing factors, which are presented in Table 4. At baseline, high frustration showed more overlap with the externalizing factor than with the internalizing factor (4% vs 1% explained variance and 6% for the general factor, Table S22). Moreover, high frustration was not predictive for change in the internalizing factor [\( t_{1-2} \), Table 4, \( \Delta \chi^2(1) = 2.82, p = .09 \)] but predicted increases in the externalizing factor (\( t_{1-2} \)). This prospective association with externalizing problems seemed largely independent from overlap at baseline [H3a, \( \beta = 0.09, p < .01, \Delta \chi^2(1) = 9.13, p < .005, \beta = 0.16, p < .001 \) in a model
without baseline externalizing factor], Finally, endogenous SLEs predicted increases in the externalizing but not in the internalizing factor (H3b), whereas more exogenous SLEs predicted increases in the internalizing but not externalizing factor (H3c). In sum, frustration proved most kindred to the internalizing factor [at age 16 \( t_{855}=6.37, p<.001 \), mean difference = (0.16)] and at age 19 [follow-up, \( t_{855}=3.39, p<.001 (0.09) \)].

In terms of the conventional binary approach to internalizing and externalizing problems, girls scored higher on the internalizing scale at ages 16 \( t_{855}=11.11, p<.001 (0.19) \) and 19 \( t_{855}=6.16, p<.001 (0.11) \), but there was no gender difference in the externalizing scale [at age 16, \( t_{855}=1.66, p=.10 (0.02) \); at age 19, \( t_{855}=0.79, p=.43 (0.01) \)]. However, in terms of three-factor internalizing and externalizing factors (mutually adjusted), the difference in the internalizing factor was even more pronounced [namely girls scored higher at ages 16 \( t_{855}=12.47, p<.001 [0.20] \) and 19 \( t_{855}=8.52, p<.001 (0.11) \), but boys scored higher on the externalizing factor at both ages 16 \( t_{855}=5.27, p<.001 (0.07) \) and 19 \( t_{855}=5.78, p<.001 (0.06) \)].

### Table 4. Frustration and the internalizing and externalizing factor

<table>
<thead>
<tr>
<th>Topic</th>
<th>Path</th>
<th>Internalizing factor</th>
<th>Externalizing factor</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Beta (95% CI)</td>
<td>Beta (95% CI)</td>
</tr>
<tr>
<td>Overlap</td>
<td>Frustration →</td>
<td>InT/ExT ( T_1 )</td>
<td>0.11*** (0.04 to 0.19)</td>
</tr>
<tr>
<td>Prospective</td>
<td>Frustration →</td>
<td>InT/ExT ( T_2 )</td>
<td>0.09** (0.03 to 0.15)</td>
</tr>
<tr>
<td>Stress</td>
<td>Frustration →</td>
<td>Endo SLEs</td>
<td>0.12*** (0.05 to 0.19)</td>
</tr>
<tr>
<td>generation</td>
<td>Frustration →</td>
<td>Exo SLEs</td>
<td>0.15*** (0.09 to 0.21)</td>
</tr>
<tr>
<td>Stress</td>
<td>Endo SLEs</td>
<td>InT/ExT ( T_2 )</td>
<td>0.24*** (0.18 to 0.31)</td>
</tr>
<tr>
<td>sensitiv.</td>
<td>Exo SLEs</td>
<td>InT/ExT ( T_2 )</td>
<td>0.15** (0.05 to 0.25)</td>
</tr>
<tr>
<td>Fr * Endo</td>
<td>InT/ExT ( T_2 )</td>
<td>–0.17** (–0.06 to –0.27)</td>
<td></td>
</tr>
</tbody>
</table>

Note: \( T_1 = \text{baseline (age 16)}; T_2 = \text{follow-up wave (age 19)} \). Endo, endogenous; Exo, exogenous; SLEs, stressful life events; InT/ExT, internalizing or externalizing factor (mutually adjusted, see the Methods section). All paths contributed significantly to the model fit (namely, \( *p<.05 \) from \( \Delta G^2 = \Delta \chi^2 = 3.84, **p<.01 \) from 6.64, ***p<.001 from 10.83), or were constrained to zero. The Methods section provides information about all variables, model-fit indices, and modelling procedures (details for the internalizing factor are reported in Tables S6–S8 and for the externalizing factor in Tables S9 to S11). The structure of the model is visualized in Figure 2.

### Table 5. Gender stratified models of change in psychopathology (total scores) or in internalizing (InT) or externalizing (ExT) factors

<table>
<thead>
<tr>
<th>Topic</th>
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<th>Psychopathology total score</th>
<th>Internalizing factor</th>
<th>Externalizing factor</th>
</tr>
</thead>
<tbody>
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<td>Overlap</td>
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<td>Psych ( T_1 )</td>
<td>0.26***</td>
<td>0.10***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>InT/ExT</td>
<td>0.30***</td>
<td>0.13***</td>
</tr>
<tr>
<td>Prospective</td>
<td>Frustration →</td>
<td>Psych ( T_1 )</td>
<td>0.10***</td>
<td>0.12***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Endo SLEs</td>
<td>0.51***</td>
<td>0.44***</td>
</tr>
<tr>
<td>Stress</td>
<td>Frustration →</td>
<td>Endo SLEs</td>
<td>0.11***</td>
<td>0.12***</td>
</tr>
<tr>
<td>generation</td>
<td>Frustration →</td>
<td>Exo SLEs</td>
<td>0.25***</td>
<td>0.21***</td>
</tr>
<tr>
<td></td>
<td>Psych ( T_1 ) →</td>
<td>Endo SLEs</td>
<td>0.25***</td>
<td>0.18***</td>
</tr>
<tr>
<td>Stress</td>
<td>Frustration →</td>
<td>Exo SLEs</td>
<td>0.16***</td>
<td>0.15***</td>
</tr>
<tr>
<td>sensitiv.</td>
<td>Endo SLEs</td>
<td>Exo SLEs</td>
<td>0.20***</td>
<td>0.24***</td>
</tr>
<tr>
<td>Fr * Endo</td>
<td>Exo SLEs</td>
<td>Exo SLEs</td>
<td>0.28***</td>
<td>0.25***</td>
</tr>
</tbody>
</table>

Note: \( * \) the beta estimates of the path differed significantly between genders. All paths attributed significantly to the model or were constrained at zero (see Tables S14, S16, and S18 respectively). The Methods section provides information about all variables and model-fit indices (all details are reported in Tables S12 to S18). The structure of the model is visualized in Figure 2.

\(*p<.001, **p<.01, *p<.05 \) (two-tailed).
Post-hoc three-way interaction

In the present paper, we hypothesized (i) that people might show a functional response to SLEs (H3b, EndoSLEs → ExTt2, H3c, Exo SLEs → InTt2), and (ii) that despite equal mean levels of baseline frustration, cultural display rules would increase the probability that frustration-prone girls develop internalizing problems after SLEs (H4a), whereas boys were more likely to develop externalizing problems (H4b). Combined, this implies that gender moderates the effect of high frustration (at t1) on the impact of endogenous versus exogenous SLEs, in terms of change in subsequent internalizing and externalizing symptoms (t1–2, a three-way interaction). To test this prediction, we fit two post-hoc SEM models to predict changes in the internalizing and externalizing factors between ages 16 and 19, which are both presented in Table 6.

The internalizing model showed that boys were less likely to develop internalizing problems after exogenous SLEs (moderation by gender, in support of H4a). Only one three-way interaction was observed (Frustrationt1 * Gender * Exogenous SLEs, $\beta = 0.16$, $p < .01$), suggesting that only frustration-prone boys at age 16 developed internalizing problems after exogenous SLEs (at age 19, Figure 6, right panel). This observation aligns with the hypothesized function (H3a/3b) and display rules (H4a/4b) and is not a ceiling effect because frustration-prone boys already reported more internalizing symptoms at baseline (Figure 4). Nonetheless, it may still reflect a chance finding. The externalizing model indicated that frustration-prone adolescents (at $t_1$) were more likely to develop externalizing symptoms (at $t_2$) after endogenous SLEs (supporting H3b) and less likely to develop externalizing symptoms (at $t_2$) after exogenous SLEs (supporting H3c). However, no three-way interactions were observed for the externalizing factor (Table 6), in discord with our display rule.

DISCUSSION

Frustration is an important part of our development from birth to death and inevitable in daily life. In this study, we tested pathways that may underlay the prospective association between dispositional frustration and severity of psychopathology as indexed by total problems: stress generation (mediation), overlap with baseline psychopathology (including ‘carry-over’ and common causes), and an independent

Table 6. Post-hoc three-way interactions (Frustration * Gender * SLEs)

<table>
<thead>
<tr>
<th></th>
<th>Internalizing factor</th>
<th>Externalizing factor</th>
</tr>
</thead>
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<td></td>
<td>Beta</td>
<td>(95% CI)</td>
</tr>
<tr>
<td>Baseline InT/ExT</td>
<td>0.56*** (0.52 to 0.59)</td>
<td>0.44*** (0.40 to 0.47)</td>
</tr>
<tr>
<td>Frustration</td>
<td>0.03 (−0.01 to 0.07)</td>
<td>0.10** (0.02 to 0.18)</td>
</tr>
<tr>
<td>Gender</td>
<td>−0.01 (−0.05 to 0.07)</td>
<td>0.02 (−0.01 to 0.05)</td>
</tr>
<tr>
<td>Endogenous</td>
<td>0.25*** (0.20 to 0.31)</td>
<td>0.27*** (0.22 to 0.32)</td>
</tr>
<tr>
<td>Exogenous</td>
<td>0.01 (−0.04 to 0.05)</td>
<td>0.08 (0.04 to 0.12)</td>
</tr>
<tr>
<td>Fr*Gender</td>
<td>0.09 (0.04 to 0.15)</td>
<td>0.10 (0.03 to 0.12)</td>
</tr>
<tr>
<td>Fr*Endo SLEs</td>
<td>0.05 (0.00 to 0.10)</td>
<td>0.09* (0.05 to 0.13)</td>
</tr>
<tr>
<td>Fr*Exo SLEs</td>
<td>−0.12** (−0.16 to −0.08)</td>
<td>−0.11** (−0.15 to −0.07)</td>
</tr>
<tr>
<td>G*Endo SLEs</td>
<td>−0.23*** (−0.30 to −0.16)</td>
<td>−0.06 (−0.11 to −0.01)</td>
</tr>
<tr>
<td>G*Exo SLEs</td>
<td>0.05 (−0.02 to 0.12)</td>
<td>−0.01 (−0.05 to 0.03)</td>
</tr>
<tr>
<td>Fr<em>Gender</em>Endo SLEs</td>
<td>−0.00 (−0.06 to 0.06)</td>
<td>−0.02 (−0.06 to 0.02)</td>
</tr>
<tr>
<td>Fr<em>Gender</em>Exo SLEs</td>
<td>0.16** (0.10 to 0.22)</td>
<td>0.07 (0.10 to 0.03)</td>
</tr>
</tbody>
</table>

Note: CI, confidence interval; Fr, frustration; G, gender; Endo, endogenous; Exo, exogenous; SLEs, stressful life events. Beta is the standardized regression coefficient. ***p < .001, **p < .01, *p < .05 (two-tailed).

Significant betas ($p < .05$, two-tailed) are reported in bold.

Figure 6. The effect of exogenous stressful life events on the internalizing factor stratified by gender. [Colour figure can be viewed at wileyonlinelibrary.com]
(i.e. unmediated) prospective effect of frustration that may also manifest as heightened stress sensitivity to SLEs (moderation). Our results supported all three pathways.

The models were fit 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 association of frustration at age 16 ($t_1$) with psychopathology at age 19 ($t_2$) was due to overlap at baseline, about 45% was independent, while 5% was mediated by SLEs, albeit indirectly, via concurrent psychopathology at age 16. High frustration at age 16 increased the likelihood of developing psychopathology at age 19 after experiencing endogenous SLEs (stress sensitivity), irrespective of their baseline psychopathology levels, in keeping with frustration as a vulnerability factor.

High frustration at age 16 proved uninformative for change in the internalizing factor over the following 3 years but predicted increases in the externalizing factor, next to more general distress. Finally, we observed that exogenous SLEs predicted increases in the internalizing (but not externalizing) factor, while endogenous SLEs between ages 16 and 19 predicted increases in the externalizing (but not internalizing) factor (Table 4). No gender differences in levels of frustration at age 16 were observed, and both genders showed comparable responses to SLEs in terms of changes in internalizing and externalizing problems between ages 16 and 19. After having summarized our main findings, these will be discussed in more detail in the succeeding texts.

Stress generation

Our models support the often reported stress generation effect of frustration (mediation). The ‘active selection’ for endogenous SLEs (H1a) was stronger than the more indirect ‘evocation’ of exogenous SLEs (H1b). This aligns with our hypothesis (H1c) and previous research on neuroticism in adult samples (Jeronimus et al., 2014; Riese et al., 2014). The observation that adolescents who are prone to frustration at age 16 tend to select themselves into environments that enhance this disposition is known as the corresponsive principle (Caspi et al., 2005; Laceulle et al., 2015). Nonetheless, frustration proneness at age 16 predicted 1–5% of the variance in SLE occurrences over the following 3 years, which is negligible from a prevention perspective but still meaningful, given all possible person–environment transactions, especially because such effects tend to accumulate over time (Jeronimus et al., 2014; Vinkers et al., 2014).

Important as well is our observation that this stress generation effect of baseline frustration was confounded by concurrent symptoms of psychopathology (Figure 2). This conclusion puts the many previous observations of frustration-driven stress generation in a different light (e.g. Laceulle, Jeronimus et al., 2015). Such observations may be confounded by concomitant psychopathology, which would dismiss SLEs as an important mediating pathway between high temperamental frustration and later psychopathology. This conclusion aligns with a previous observation in adult samples that most of the stress generation effect of high neuroticism on SLEs disappears after adjustment for baseline symptoms of anxiety and depression (e.g. Jeronimus, Ormel, Aleman, Penninx, & Riese, 2013). Nonetheless, this control also takes legitimate variance out of neuroticism, especially from the facet traits anxiety and depression (Riese, Ormel, Aleman, Servaas, & Jeronimus, 2016), and this analogy probably extends to temperamental frustration (we elaborate upon this trait vs state problem in the succeeding texts).

Important as well is our observation that this stress evocation effects (of baseline and concurrent psychopathology) from causes common to both frustration and psychopathology. Because the total overlap between frustration and psychopathology at baseline was not even 10% (as elaborated upon in the succeeding texts), the observation that this overlap explains over half of the prospective association suggests that frustration is close to the origin of the causal pathways towards psychopathology not only via (i) overlap that may reflect state effects, common causes, measurement overlap, or symptoms intervening between frustration and diagnosis (Frustration $\rightarrow$ Sx $\rightarrow$ Dx) but also via (ii) an independent adjusted ‘direct’ vulnerability effect of frustration for the development of psychopathology (Frustration $\rightarrow$ Sx/Dx).
The frustration vulnerability effect

About 45% of the prospective effect of high frustration (at t1) on psychopathology (at t2) was independent from overlap at baseline and unmediated by SLEs. This ‘unique’ frustration-driven effect on change in psychopathology is medium-sized ($d = ~0.35$). The mechanisms underlying this ‘direct’ effect of frustration remain largely unknown but arguably tap into processes that frustration sets in motion—such as a negative bias in attention, interpretation and recall of information, increased reactivity, and ineffective coping, which may render these adolescents more vulnerable to develop psychopathology (Caspi et al., 2005; Chan et al., 2007; Laceulle, Jeronimus et al., 2015; Ormel et al., 2013). Our results confirmed that adolescents high on frustration are more sensitive to stress (H2b, moderation), as they were more likely to develop psychological problems after endogenous SLEs, in line with the vulnerability model (Jeronimus et al., 2016; Laceulle et al., 2015; Ormel et al., 2013).

Unexpectedly, high baseline frustration weakened the impact of exogenous SLEs on subsequent psychopathology (for both genders). This superficially appears to indicate a protective effect, in which greater frustration after exogenous SLEs protects against psychopathology. However, careful investigation of the form of the interaction suggests a ceiling effect, in which adolescents high on frustration (at t1) were already so prone to psychopathology that exogenous SLEs did not measurably increase their risk for psychopathology (at t2), while their peers low on frustration were unhindered by this restriction of range (Figure 5) and responded to exogenous SLEs. Hence, this alleged ‘protective effect’ of frustration, which, to the best of our knowledge, has never been reported before, requires replication with a different design to gain substance. Overall, our results provide robust support for the perspective that adolescents predisposed to frustration at age 16 are more vulnerable to stress and more likely to develop psychopathology over the following years.

The structure of psychopathology

Conventional models outline that high frustration predicts the development of both internalizing and externalizing problems (Eisenberg et al., 2001; Laceulle, Ormel et al., 2014; Lengua, 2003; Muris & Ollendick, 2005; Oldehinkel et al., 2004; Ormel et al., 2005). The three-factor model in the present paper, however, suggests that high frustration at age 16 is only predictive for change in the externalizing factor (next to the general distress factor) and not for the internalizing factor (cf. Khan, Jacobson, Gardner, Prescott, & Kendler, 2005). This difference illustrates how our understanding of the structure of psychopathology can shape our perspective on the effects of temperament (Lahey et al., 2012; Tackett et al., 2013). Our procedure to derive an internalizing and externalizing factor was analogous to an adjustment for confounders, for example, age, gender, SES, baseline psychopathology, or the general factor. Both at ages 16 and 19, frustration showed overlap with about 6% of the variance in the general factor, 4% of the externalizing factor, and 1% of the internalizing factor (roughly 7% in total).

Even if the reader does not agree with this three-factor approach (e.g., because one thinks it is an ‘evaluation bias’, Pettersson, Turkmeyer, Horn, & Menatti, 2012; Ye, 2009), this is no free lunch, as the way you conceptualize the structure of psychopathology determines whether one observes gender differences in the externalizing factor at ages 16 and 19. Our results support the hypothesis that prevalent forms of psychopathology have both important common (general factor) and unique features (Laceulle et al., 2014; Lahey et al., 2012). Frustration is closest to the externalizing factor (both at ages 16 and 19), which also shows unique links with risk taking and lack of empathy (Khan et al., 2005; Tackett et al., 2013), whereas the internalizing factor predicts educational outcomes (Ormel, 2015), among others. In the present study, only the externalizing factor at age 16 predicted endogenous SLEs over the following 3 years (‘selection effects’), whereas both higher internalizing and externalizing factor scores at age 16 predicted subsequent exogenous SLEs.

Today personality science leads research and practice in clinical psychology and remains the only evidence-based approach to a comprehensive classification of psychopathology (Krueger, Hopwood, Wright, & Markon, 2014; Tackett et al., 2013). It remains a core question what distinguishes temperamental frustration (or personality) from psychopathology (Durbin & Hicks, 2014; Ormel, Laceulle, & Jeronimus, 2014). Theorists argue that measures for psychopathology tap into more extreme behaviours (the pathological deviations from normality, cf. Kendler, Zachar, & Craver, 2011; Weiss, Süsser, & Catron, 1998), which are thought to manifest in some impairment of functioning for the adolescent (Tackett, 2006) and to have a more episodic nature (Ormel et al., 2014). Measures for temperament (or personality) are thought to capture a much broader and more normative set of behaviours, which are more stable over time (Durbin & Hicks, 2014; Ormel et al., 2013; Snyder et al., 2015). However, these are rather general notions, and it remains largely unclear how both concepts relate. Further research on the construct of temperament in adolescence is required for a comprehensive understanding of adolescent mental health, and such research may have important implications for our understanding of adolescent functioning in academic and social contexts (Snyder et al., 2015).

Gender effects

Boys and girls did not differ in frustration levels at age 16 in our study, which is new to the literature. Furthermore, frustration proneness at age 16 did not manifest in a higher probability of internalizing problems in girls than in boys at age 19 [in discord with H4a, G $\times$ F(t1)$\rightarrow$($\Delta$Int[t1-2] $>$ $\Delta$Ext[t1-2])] and neither in a higher probability of developing externalizing problems in boys than in girls [in discord with H4b, B $\times$ F(t1)$\rightarrow$($\Delta$Ext[t1-2] $>$ $\Delta$Int[t1-2])]. Post hoc, however, we observed that frustration-prone 16-year-olds were in general less likely to respond with internalizing problems to exogenous SLEs (in discord with H3b), and this effect was moderated by gender (stronger in boys, in line with a display rule, H4b).
Overall, high frustration at age 16 predicted increases in the externalizing factor after endogenous SLEs over the following 3 years (in line with H3a) but a lower probability of externalizing symptoms after exogenous SLEs (in line with H3b). Additionally, a three-way interaction indicated that only frustration-prone boys (at age 16) reacted with internalizing problems to exogenous SLEs ($\beta = 0.16$, Figure 6), in line with a vulnerability effect. This observation cannot be explained as a ceiling effect, as frustration-prone boys reported already more internalizing symptoms at baseline (Figure 4). In sum, our results suggest that there is merit to the idea that frustration-prone adolescents tend to react with externalizing responses to endogenous SLEs (which they appraise as controllable).

Our observation of a display rule that enhances the effect of frustration in boys (in line with Sell et al., 2009) requires refinement in future work because not all results supported our gender hypotheses, as there was no indication of stronger internalizing responses to SLEs in girls (in discord with H4a). Moreover, the few gender effects we observed were modest at best, in line with a meta-analysis of gender differences, which indicated that these are typically small (mean $r = 0.12$, see Richard et al., 2003). A meta-analysis of emotional expressions suggested that frustration did not differ over infancy, but that boys started to show more externalizing tendencies during toddlerhood/pre-school age ($g = 0.17$) and middle childhood ($g = 0.13$), which arguably reflects a cultural gender difference (given the absence of significant hormonal divergence) that tends to disappear again over adolescence ($g = -0.27$; see Chaplin & Aldao, 2013). Perhaps changing gender roles makes adolescent girls’ expression of anger more acceptable in recent years (Chaplin, 2015), which could explain why most of our observations failed to support the hypothesized cultural display rules. Furthermore, gender differences may also lay hidden in our concepts; for example, depression seems more often manifested in terms of anger (and somatic symptoms) in men and in terms of sadness and anxiety in women (Addis, 2008; Martin, Neighbors, & Griffith, 2013).

**Individual differences**

Although most adolescents reacted with externalizing symptoms to endogenous SLEs, in keeping with our hypotheses and the argument that people tend to externalize blame (Tennen & Affleck, 1990), it remains likely that in some adolescents, the self as the agent of blame may have resulted in internalizing problems. More fine-grained theories are required to explain such individual variability, as the final frustration reaction is undoubtedly influenced by many factors, of which perceived controllability is only one. It has been shown that adolescents’ stress-induced psychophysiological responses reflect complex interactions between the nature of the stressor and other individual characteristics (e.g. Stroud, Salovey, & Epel, 2002), but the mechanisms behind dispositional frustration in terms of states and endocrine reactivity (hormones like cortisol and functioning of the prefrontal cortex) remain poorly understood (Lopez-Duran, Hajal, Olson, Felt, & Vazquez, 2009; Panksepp & Biven, 2012). Future studies using an individual difference approach will probably provide a clearer, more robust, and more accurate understanding of the underlying mechanisms.

**Frustration versus anger**

In the literature on adolescent temperament, it is often argued that negative emotionality (frustration, fear, anger, and sadness) mobilizes avoidance behaviour away from non-reward or punishment, stressing the close relationship to constructs such as neuroticism and Gray’s Behavioral Inhibition System (e.g. Derryberry & Rothbart, 1997; Muris & Ollendick, 2005). Our observation that frustration at age 16 predicted the development of the (three-factor) externalizing but not internalizing factor between ages 16 and 19 (next to general distress) is in discord with this idea but aligns with recent studies that indicate that high frustration is associated with greater approach tendencies, which yield more motivation towards goals and stronger responses to goal thwarting (Carver, 2006; Carver & Harmon-Jones, 2009). This prospective effect of frustration on the externalizing factor might reflect the appraisal of poor approach success combined with the appraisal of the goal as attainable, resulting in anger (Carver & Harmon-Jones, 2009; Ekman, 2007; Sell et al., 2009), while low approach success combined with giving up the initial goal results in sadness/depression (Carver, 2004; Panksepp & Biven, 2012; Sell et al., 2009). Importantly, high adolescent frustration is also manifested as the perception of more frequent hostile intend, rejection, and disapproval in others (Harris, 2009; Hubbard, Mc Cauliffe, Morrow, & Romano, 2010; Laceulle, Jeronimus et al., 2015), which in turn elicits defensive responses.

Our results align with the popular frustration–aggression hypothesis, which holds that frustration leads to aggression (Dollard et al., 1939; Munyo & Rossi, 2013). However, we still argue that this is not necessarily so, and that instrumental aggression can also be learned (like all other instrumental behaviours) because failure to obtain a desired goal is not a necessity for aggression, which it is for frustration (Berkowitz, 1988). Frustration tends to elicit anger (an emotion), which in turn reduces inhibitions and narrows attention to cues for threat, which can lead to aggression (a behaviour, either physical, verbal, or relational, see Warburton & Anderson, 2015; Panksepp & Biven, 2012). The frustration–aggression link can be refined via the dual aggression model that distinguishes reactive from proactive aggression (see for a review Hubbard et al., 2010). Temperamental frustration elicits reactive aggression to a perceived blockage (aroused/hot), resulting in an emotional, impulsive, and defensive or hostile/retributive reaction (Berkowitz, 1962; Warburton & Anderson, 2015). In our study, this effect was stronger after endogenous SLEs (more externalizing problems), but frustration can also elicit anger after exogenous events that are not directed at people personally, such as a traffic jam (Berkowitz, 1988). Proactive aggression, in contrast, tends to be calm and deliberate (unaroused/cold) and instrumental to a desired outcome (gain/dominance) and is associated with adolescent’s popularity, delinquency, and psychopathy but
not frustration (Hubbard et al., 2010; Warburton & Anderson, 2015).

Sigmund Freud (1912) introduced ‘frustration’ to refer both to external barriers to goal attainment and internal obstacles blocking satisfaction, and we agree that outcomes may be conceptualized as a hybrid of psychological and contextual factors. The internal obstacles may refer to (i) the mentioned negative biases in attention and interpretation (e.g. hostile attributional bias) and recall or coping; (ii) individual deficiencies in knowledge or skill (such as social cue encoding or emotional awareness) and physical abilities (Hubbard et al., 2010; Sell et al., 2009; Shorkey & Crocker, 1981); and most prominently, (iii) executive control abilities when experiencing high emotional arousal (Moran, Lengua, & Zalewski, 2013; Rothbart, 2011; Valiente et al., 2003).

High frustration may not be a risk for externalizing problems when adolescents are able to modulate their emotional reactivity, in which case the adolescent is able to respond in a flexible, adaptive, and socially acceptable way. It is the combination of dispositional frustration with low levels of self-regulation that is associated with the expression of adolescent psychopathology (Caspi & Shiner, 2011; Muris & Ollendick, 2005).

In this paper, we focused upon contextual factors that may moderate the effect of temperamental frustration at age 16 (external barriers), which we operationalized in terms of endogenous and exogenous SLEs, but which captured only a small subcategory of all potential frustration-evoking events. Additionally, other clustering strategies, such as interpersonal stressor versus other stress, may also yield different results or evidence gender differences (Stroud et al., 2002). Moreover, the literature suggests that situational effects are generally similar or slightly larger in magnitude compared with person effects (r = .20, see Richard et al., 2003). Our results support the idea that interactions within the developmental context should be incorporated in the study of temperamental frustration (cf. Jeronimus et al., 2014; Rauthmann, Sherman, & Funder, 2015; Sherman, Rauthmann, Brown, Serfass, & Jones, 2015), if only because adolescents who never encounter triggers to activate this propensity do not show frustration (Harris, 2009; Rothbart, 2011). In practice, however, all people encounter barriers that must be removed to achieve their goals, and in this context, temperamental frustration plays a pivotal role, as this study showed that the tendency to respond with frustration increases the probability to develop psychopathology over adolescence, especially symptoms in the externalizing spectrum.

**Limitations**

The results of our study should be interpreted in light of the following strengths and limitations. The strengths of this study are our sample of almost a thousand adolescents from the general Dutch population in which SLE occurrences were assessed with LSIs. We applied powerful statistical tools to analyse the associations among frustration, SLEs, and psychopathology, which should reliably detect effects from \( r = .10 \) (\( d = 0.20 \)) 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 the Methods section) who rated SLEs independently of the respondent, a procedure that currently forms the gold standard of life stress research (Dohrenwend, 2006; Monroe, 2008).

Another limitation is that we operationalized psychopathology solely with a self-report measure (see the Methods section), and not by multiple raters, or a diagnoses based upon a psychiatric interview. Our measure therefore taps into psychological problems but does not index clinical disorders per se. Furthermore, at baseline, we used the YSR to measure psychopathology, and at follow-up the Young ASR, but as outlined in the Methods section, previous work suggests that these measures are fairly comparable (Ferdinand et al., 1995). Moreover, the association of \( r = .62 \) over 2 years (Table 2) is identical to most test–retest associations for other measures of psychopathology (see for a review Ormel et al., 2013).

The suggestion that our observations regarding the effect of frustration at age 16 on externalizing problems at age 19 follow from higher measurement overlap at baseline is not supported, as i) both measures were based upon different questions and time frames (frustration taps into characteristic irritability and frustration sensitivity, while the externalizing items capture behaviours, including setting fires, lying, stealing from your parents, being truant, using drugs, or lacking guilt), and (ii) the associations between baseline frustration and baseline internalizing and externalizing scales were equal (unadjusted, both \( r = .19 \), Table 2). This suggests that the item overlap between frustration and the conventional externalizing symptom scale was not larger than with the internalizing symptom scale and cannot explain our observations. Additionally, frustration was based upon parent report, and the internalizing and externalizing scales on self-report, which also decreases shared-method problems.

**CONCLUSION**

In this study, three pathways were identified that underlie the prospective association of frustration at age 16 on total psychopathology at age 19: stress generation, a carry-over effect of frustration on future psychopathology via concurrent psychopathology plus past common causes, and an independent direct vulnerability effect of frustration. Our findings suggest that high temperamental frustration and associated internalizing and externalizing tendencies form a transactional ‘vicious’ cycle between adolescents and the environments they navigate and shape, which can give rise to more psychopathology and an even deeper ingrained tendency for negative affect (Jeronimus et al., 2014, 2016). We hope that future studies test additional mechanisms that may explain the prospective association between frustration at age 16 and externalizing psychopathology at age 19 that is not mediated by SLEs, such as social interactions and perceptual alterations.
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