Neuroticism and extraversion in relation to physiological stress reactivity during adolescence

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ABSTRACT

The current study examined mean level and change in extraversion and neuroticism across adolescence in relation to physiological stress reactivity to social evaluation. Adolescents ($n = 327$) from the Dutch general population reported on personality measures at five annual assessments. At age 17 years, adolescents participated in a psychosocial stress procedure characterized by social evaluation during which cortisol, heart rate, pre-ejection period (PEP) and heart rate variability were assessed. Dual latent growth curve models were fitted in which the intercepts (mean level) and slopes (change) of personality across adolescence predicted the intercepts (baseline) and slopes (reactivity) of the physiological stress measures. Most comparisons revealed no relation between personality and stress reactivity. Adolescents with higher mean level scores on extraversion did show lower cortisol reactivity. Adolescents with higher mean level neuroticism scores showed higher PEP reactivity. Our findings lend partial support for a relation between personality and physiological stress reactivity.

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1. Introduction

Hans Selye's and Walter Cannon's pivotal work showed that the physiological stress response is the body's adaptive manner of handling internal and environmental imbalance (Cannon, 1929; Selye, 1950). When confronted with a stressor, autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis activation prepare the individual for and facilitate an appropriate response (i.e. stress reactivity). It is widely understood that individuals vary in their responses to stress, and many factors contribute to this variation. For instance, specific genes (e.g. Mueller, Strahler, Armbruster, Lesch, Brocke, & Kirschbaum, 2012), gender (e.g. Ordaz & Luna, 2012), body mass index (BMI; Carroll, Phillips, & Der, 2008), socioeconomic status (SES; Miller et al., 2009) and pubertal stage

(Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; van den Bos, de Rooij, Miers, Bokhorst, & Westenberg, 2014) have all been shown to influence the magnitude of the physiological and/or perceived stress response. Furthermore, stress responses and the influence of these factors on stress responses may not be uniform across different stages of development (e.g. Evans, Greaves-Lord, Euser, Tulen, Franken, & Huizink, 2013; Gunnar et al., 2009). Despite the recognized influence of these factors, a large portion of the variance in individual stress responses remains unexplained. One potential factor that may further add to clarification of this variance is personality or trait differences, most notably neuroticism and extraversion due to the wealth of theory and previous research on these particular constructs.

The human stress response systems, the ANS and the HPA axis, are phylogenetically old and complex. The ANS consists of two branches: the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). The vagus nerve of the PNS is responsible for maintaining homeostasis and supporting

social engagement during rest and plays a central role in emotion regulation (Porges, 1995, 2007; Porges, Doussard-Roosevelt, & Maiti, 1994). When an organism is confronted with a stressor, the most immediate response involves vagal withdrawal, indicating the organism's preparedness to respond to an anticipated stressor. Vagal activation is often indexed by heart rate variability (HRV), which is high during rest (PNS activation) and low during stress (vagal withdrawal). If the response of vagal withdrawal is insufficient, the SNS is activated, entailing the fight-or-flight response which, among other functions, increases heart rate, blood pressure, and secretion of adrenaline and noradrenaline. This hierarchy of responses is consistent with the Jacksonion principle of dissolution (Jackson, 1958; Porges, 2007). Pre-ejection period (PEP) is frequently utilized as an index of SNS activation and marks the time interval in milliseconds between the beginning of the ventricular depolarization and the beginning of the ventricular ejection. Shorter intervals thus indicate stronger sympathetic activation. Furthermore, PEP is thought to be an index of the dopaminergic response to reward (Beauchaine, 2012). The heart is innervated by both the PNS and SNS. These branches operate independently of one another, and usually exert reciprocal influences on the heart (Berntson, Cacioppo, & Quigley, 1991). Heart rate thus indexes combined PNS and SNS (de) activation.

The response of the HPA axis entails the production of corticotropin-releasing hormone by neurons in the paraventricular nucleus (PVN) of the hypothalamus. This stimulates the secretion of adrenocorticotrophic hormone in the pituitary which in turn stimulates the secretion of cortisol in the outer cortex of the adrenal gland. On normal days, the cortisol levels of healthy individuals follow a diurnal curve with an acrophase approximately 30 min after awakening and a subsequent gradual decrease (Schmidt-Reinwald et al., 1999). When confronted with a stressful situation, the adaptive response of a healthy individual is a temporary increase in the secretion of cortisol which is observable in saliva about 20 min following the onset of the stressor (Sapolsky, Romero, & Munck, 2000). Ultimately, the human stress response is an intricately complex series of events, with activation of the ANS and HPA axis beginning in the PVN (Benarroch, 2005; Joels & Baram, 2009). This is followed immediately by observable physiological changes in ANS functioning (i.e. vagal withdrawal followed by increased heart rate and blood pressure, a shortening of the PEP) and subsequent increases in salivary cortisol levels after some time (Joels & Baram, 2009; Sapolsky et al., 2000).

In an influential biopsychological theory of personality, Hans J. Eysenck proposed that extraversion and neuroticism are independent constructs characterized by differential physiological responses and that distinct biological substrates underlie these responses (Eysenck, 1967; Eysenck & Eysenck, 1985). He hypothesized that neuroticism is related to hyper-arousability of the limbic system, which is implicated in the processing of emotions related to the SNS fight-or-flight response. Therefore it might be expected that individuals scoring high on neuroticism scales would respond physiologically strongly to a stressor, and perhaps in particular when measuring SNS responses. In contrast, extraversion was hypothesized to be associated with hypo-arousability of principally the reticulo-cortical loop and other arousal systems such as the monoamine oxidase system or the pituitary-adrenocortical system (Eysenck, 1967; Eysenck & Eysenck, 1985; Hagemann, Naumann, Lurken, Becker, Maier, & Bartussek, 1999). Individuals scoring high on extraversion may thus be less physiologically responsive to stress than those scoring high on introversion. Especially when the stressor contains social-evaluative elements, extraverts may respond less strongly to the stressor because of their tendency to experience positive affect and to be less inhibited in social situations (Eysenck & Eysenck, 1985).

Emotion regulation may play a key role in the relation between stress reactivity and personality. The ANS (e.g. Porges, 2007; Porges et al., 1994) and the HPA axis (e.g. Stansbury & Gunnar, 1994) are both implicated in emotion regulation capabilities. Extraversion and neuroticism are also characterized by emotion regulation abilities: extraverts tend to be able to effectively regulate their emotions (Jonassaint et al., 2009) while individuals scoring higher on neuroticism are not (e.g. Ormel et al., 2013). Physiological stress reactivity, personality, and emotion regulation capabilities may thus be interrelated, with bidirectional relations proposed between each relation (e.g. Koole, 2009; Porges, 2007; Porges et al., 1994; Stansbury & Gunnar, 1994).

Numerous empirical examinations of a relation between physiological reactivity and personality have been executed since the late 1960s, however yielding inconclusive results (Ormel et al., 2013; Pearson & Freeman, 1991). For example, neuroticism has been linked to physiological hyper-reactivity (e.g. Habra, Linden, Anderson, & Weinberg, 2003), but also to hypo-reactivity (e.g. Chida & Hamer, 2008; Phillips, Carroll, Burns, & Drayson, 2005). Extraversion, too, has been linked to physiological hypo-reactivity (e.g. Gange, Geen, & Harkins, 1979; Jonassaint et al., 2009; Kirschbaum et al., 1995) as well as hyper-reactivity (e.g. Oswald, Zandi, Nestadt, Potash, Kalaydjian, & Wand, 2006). However, the majority of studies failed to find an association between physiological reactivity and neuroticism and/or extraversion (e.g. Arnetz & Fjellner, 1986; Garcia-Banda et al., 2011; Glass, Lake, Contrada, Kehoe, & Erlanger, 1983; Kirschbaum, Bartussek, & Strasburger, 1992; Schommer, Kudielka, Hellhammer, & Kirschbaum, 1999).

Yet, methodological considerations of previous studies preclude dismissing this potential association. Most included small samples and utilized few physiological measurements. The type of stressor used varies greatly; early studies tested the response to visual vigilance tasks (e.g. Gange et al., 1979) or non-laboratory stressors such as lecturing (e.g. Houtman & Bakker, 1991), and many utilized mental arithmetic tasks (e.g. Pearson & Freeman, 1991). Given that tasks containing social-evaluative threat and uncontrollability are most likely to induce a physiological response (Dickerson & Kemeny, 2004), other types of stressors may be less effective herein. Psychosocial stress tasks such as the Trier Social Stress Task (Kirschbaum, Pirke, & Hellhammer, 1993) and the Leiden Public Speaking Task (PST; Westenberg et al., 2009), in which social evaluation is focal, may be more ecologically valid as well as more suited to examining the relation between physiological reactivity and personality (Kamarck & Lovallo, 2003). Only a few studies have utilized such tasks to investigate the physiological correlates of personality (e.g. Garcia-Banda et al., 2011; Kirschbaum et al., 1992), though yielded inconclusive findings, and further research of this line is warranted.

Furthermore, the majority of this research has been performed in adults, whereas the relation between stress reactivity and personality may be especially paramount during adolescence (Charmandari, Kino, Souvatzoglou, & Chrousos, 2003). Indeed, adolescence is considered a sensitive period, critical in the development of both personality traits (Klimstra, Hale, Raaijmakers, Branje, Meeus, 2009) and physiological stress reactivity (Klein and Romeo, 2013; Romeo, 2010). To our knowledge, only two studies examined physiological stress reactivity and personality in adolescent samples. Laceulle and colleagues (Laceulle, Nederhof, van Aken, & Ormel, 2015) found that neuroticism and extraversion were not related to cortisol reactivity to a psychosocial stressor, but were related to basal cortisol levels. Similarly, Zahn, Kruesi, Leonard, & Rapoport, 1994 found that extraversion scores were unrelated to heart rate responses to a reaction time task. Considering that social evaluation becomes an increasingly prominent concern in adolescence (Westenberg et al., 2004), examining the physiologi-

cal response to *psychosocial* stressors in relation to personality traits during adolescence is pertinent.

When studying the link between personality traits and stress in adolescence, it may be necessary to take into account possible changes in personality across the adolescent years. Indeed, personality traits may not be as stable (i.e. mean-level stability) as previously thought (Groothuis & Trillmich, 2011), and may shift during adolescence (Branje, Van Lieshout, & Gerris, 2007; Klimstra et al., 2009; McAdams & Olson, 2010) and early adulthood before becoming gradually more stable across middle and later adulthood (Roberts, Walton, & Viechtbauer, 2006). The prefrontal cortex plays a major part in regulating behavioral inhibition, which is implicated in personality (Koolhaas, de Boer, Buwalda, & van Reenen, 2007). As the prefrontal cortex continues to undergo maturation throughout adolescence (Giedd et al., 1999), we can expect personality to continue to develop during this period as well (Groothuis & Trillmich, 2011). Therefore, in the present study we considered both mean level and change in personality scores across adolescence.

This study aimed to examine the personality traits extraversion and neuroticism in relation to physiological stress reactivity during a psychosocial stressor, characterized by social evaluation, in a large sample of adolescents from the general population. General consensus currently points to five personality traits (i.e. the Big Five; Costa & McCrae, 1988). However, in the current study we chose to focus on extraversion and neuroticism in particular because our research questions and hypotheses are rooted in Eysenck's theory of these two constructs, and given the wealth of previous research that has been done on the relation between these two constructs and physiological stress reactivity. As personality traits may be differentially related to ANS and HPA axis responding, we included measures of both systems. In order to index ANS reactivity, we measured heart rate (HR), PEP and HRV. We included HR because we were mainly interested in overall ANS responding, and HR indexes combined sympathetic and parasympathetic activation. We included PEP and HRV in order to gain more insight into sympathetic and parasympathetic activation specifically. In order to index HPA axis reactivity, we collected salivary cortisol samples. In a series of dual process growth curve models, we examined to what extent individual differences in mean level and change in personality scores across adolescence predicted individual differences in physiological stress levels at baseline and during the psychosocial stressor, which took place in late adolescence. Following Eysenck's (1967) theory, we hypothesized that neuroticism would be related to higher stress reactivity and that extraversion would be related to lower stress reactivity.

2. Materials and methods

2.1. Participants

This study is part of the ongoing longitudinal project for Research on Adolescent Development And Relationships, Younger cohort (RADAR-Y). A cohort of 523 Dutch adolescents (56% male; age at first assessment: $M = 13.02$ years, $SD = 0.51$) was followed from the beginning of secondary education and participated subsequently in an ongoing longitudinal study. Because one of the specific aims of the study was to focus on the development of delinquency, the cohort was oversampled for youth at risk for developing such behavior. About 41% of the original sample was selected based on a higher prevalence of symptoms of externalizing problems (based on a T score at or above the borderline clinical range on the Teacher Report Form, see Achenbach, 1991). Each year, the adolescent participated in a home-based assessment during which, among others, personality characteristics were assessed. The present study used the first five annual assessments.

Approximately three months before the fifth annual assessment, adolescents were invited to participate in a laboratory session. Of the original 523 adolescents, 40 had declined further participation prior to the lab assessment and were therefore not invited to the lab. Of the remaining 483 adolescents, 327 (68%) participated in the Public Speaking Task (58% male; M age = 17.30 years, $SD = 0.45$). Thirty-nine percent of the final sample was at risk for developing delinquency problems. Adolescents in the final sample did not differ from those who declined participation ($n = 156$) with regard to gender, $\chi^2 = 1.47$, $p = 0.225$; risk group, $\chi^2 = 0.00$, $p = 0.961$; age, $t(1, 480) = 0.58$, $p = 0.565$; and average neuroticism scores across ages 13–17 years, $t(1, 480) = 0.87$, $p = 0.386$. However, decliners were more likely to come from a low SES background (18%) compared to participants (6%), $\chi^2 = 16.36$, $p < 0.001$; and average extraversion scores across ages 13–17 years were higher among participants ($M = 4.17$, $SD = 0.88$) than among decliners ($M = 3.90$, $SD = 0.94$), $t(1, 480) = -3.11$, $p = 0.002$.

2.2. Measures

2.2.1. Personality

Extraversion and *neuroticism* were assessed annually for five years using a Dutch version of Big Five personality markers (see Branje et al., 2007), selected from Goldberg (1992). On a 30-item, seven-point Likert-scale (ranging from 1: *very untrue of me* to 4: *sometimes untrue, sometimes true of me* to 7: *very true of me*), adolescents indicated to which degree they possessed personality attributes such as *talkative* (extraversion) and *worried* (neuroticism). Each of the five personality scales consisted of six items. Scores on the subscales *extraversion* and *neuroticism* were used in the present study. The *neuroticism* subscale was the result of recoding the *emotional stability* scale; higher scores represent a greater degree of neuroticism. Internal consistency at the five assessments was good for extraversion (M Cronbach's alpha = 0.84, range 0.80–0.87) and neuroticism (M Cronbach's alpha = 0.86, range 0.81–0.90). The Big Five Personality Inventory is generally regarded as a reliable and valid measure of personality across adolescence (De Fruyt, Mervielde, Hoekstra, & Rolland, 2000).

2.2.2. Cortisol

Salivary cortisol was assessed eight times during the laboratory assessment by collecting saliva in a test tube. Saliva was collected at the beginning (sample 1) and directly after (sample 2) the baseline, after the speech delivery (sample 3), and at five-minute intervals starting ten minutes into the recovery period (samples 4–8). Taking into account the approximate 20-min delay between activity in the hypothalamus and observable changes in salivary cortisol levels (Sapolsky et al., 2000), each sample corresponded to cortisol levels earlier in the procedure. The first corresponded to cortisol levels during the computer tasks prior to the PST and was thus excluded from the analysis. The second and third corresponded to cortisol levels during the first and final five minutes of the baseline period of the PST (baseline 1 and baseline 2 in Fig. 1). The fourth through sixth measurements corresponded to cortisol levels during the three phases of the speech task (i.e. fourth: anticipation, fifth: preparation, sixth: speech). The seventh and eighth measurements corresponded to cortisol levels during the post-task period. We chose to focus on reactivity in the present study, therefore only cortisol samples 2 through 6 were utilized in this study. Samples were stored uncentrifuged at −20 °C (Aardal & Holm, 1995) and analyzed using electrochemiluminescence immunoassay ECLIA (E170 Roche, Switzerland) at the laboratory of the Leiden University Medical Center (further details available upon request). Outliers greater than 3 SD above the mean were removed from the analyses due to possible contamination (e.g. blood, medicine). All cortisol val-
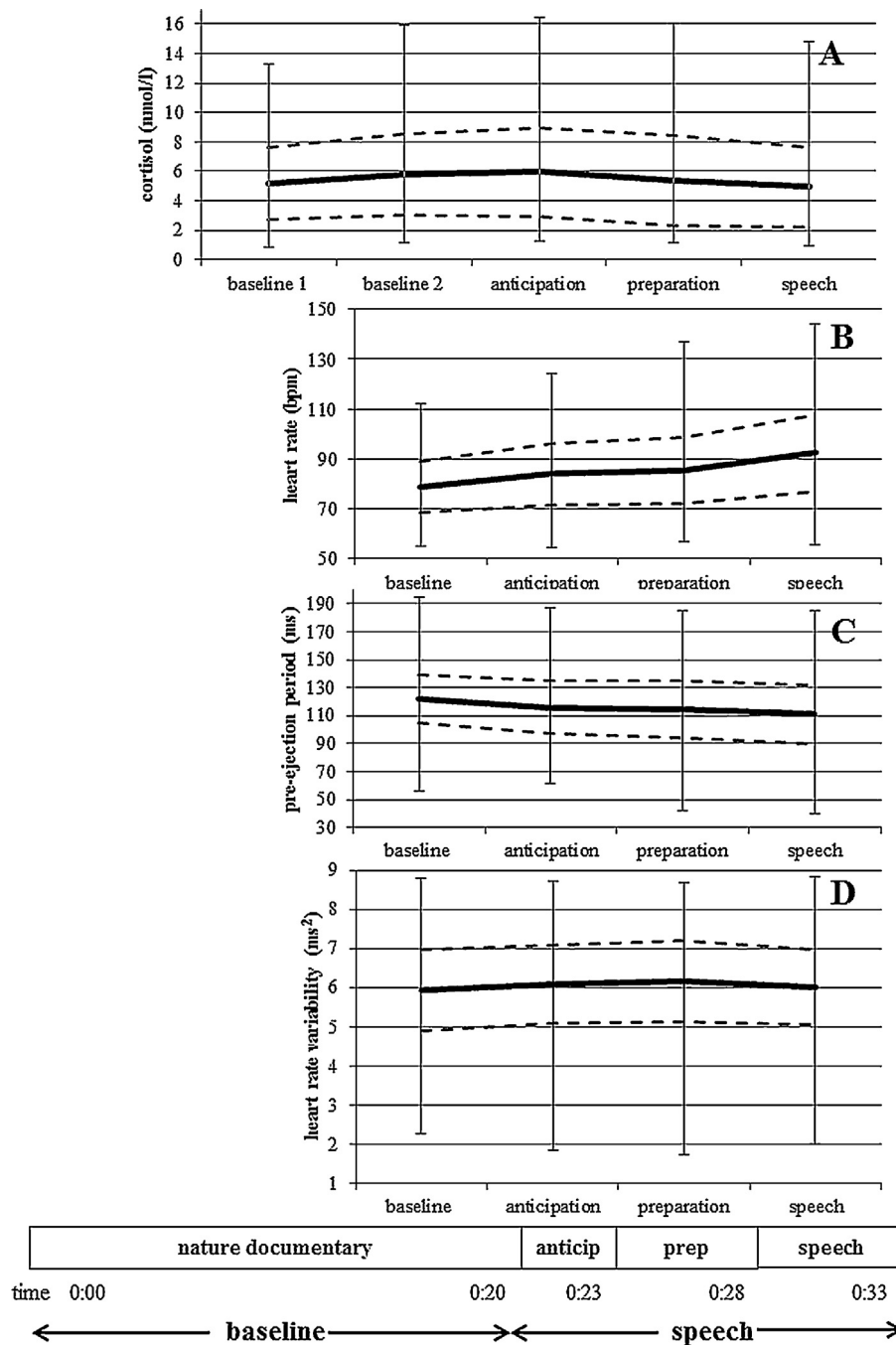
Fig. 1. Illustration of descriptive statistics for physiological stress measures and the psychosocial stress procedure.
Caption: average (solid line), standard deviation (dotted lines) and range (bars) statistics are depicted for cortisol (A), heart rate (B), pre-ejection period (C) and heart rate variability (D) across the psychosocial stressor, including baseline and speech (anticipation, preparation and speech delivery) periods.
Note: Cortisol and heart rate variability values are natural log transformed; anticip = anticipation; prep = preparation.

ues were natural log transformed in order to approximate normal distributions.

2.2.3. Heart rate (HR), pre-ejection period (PEP) and heart rate variability (HRV)

The VU Ambulatory Monitoring System 5 fs device (VU-AMS; De Geus & Van Doornen, 1996) was used to record the electrocardiogram (ECG) and changes in the impedance cardiogram (ICG). This device allows continuous recording of ECG and ICG signals at a 1000 Hz sampling rate (van Lien, 2014). The system uses a seven-electrode configuration of which two for the ECG and

one for the ground.[1] Serial markers were automatically sent to the device. The PEP is the time interval in milliseconds between the beginning of the ventricular depolarization (Q-wave onset in

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[1] The electrodes were placed as follows: **1**: slightly below the right collar bone, four centimeters to the right of the sternum; **2** (ground): on the right side, between the lower two ribs; **3**: at the apex of the heart on the left lateral margin of the chest approximately at the level of the processus xiphodius; **4**: on the spine at least three centimeters above electrode 6; **5**: on the spine at least three centimeters below electrode 7; **6**: at the suprasternal notch above the top of the sternum; **7**: at the processus xiphodius at the bottom of the sternum. For more information and a diagram, see http://www.psy.vu.nl/vu-ams.

ECG) and the beginning of the ventricular ejection (B-point in ICG). For HR, R-to-R distances were recorded, automatically scored (with VU-DAMS software, http://www.psy.vu.nl/vu-ams) and visually corrected, resulting in an interbeat-interval (IBI) time series. PEP was automatically scored (using the same software), visually inspected and adapted when necessary. Power spectral analysis was used to calculate HRV. For this calculation, the IBI time series was convoluted with a smoothness prior matrix to yield a stationary signal on which a discrete Fourier analysis was performed. This yielded a power spectra of the rhythmic oscillations over a frequency range of 0.0001 Hz to 0.40 Hz. The power in the high frequency band (0.15–0.40 Hz) was calculated as an index of HRV (Akselrod, Gordon, Ubel, Shannon, Barger, & Cohen, 1981). Mean HR (in beats per minute), mean PEP (in ms) and mean HRV (in ms^2) were calculated for each of the phases of the PST: the baseline (only the final five minutes during which participants were standing), anticipation (three minutes), preparation (five minutes) and speech delivery (five minutes). The final part of the baseline was performed standing in order to avoid confounding effects of posture as the anticipation, preparation and speech delivery parts of the PST were also performed in a standing position. HRV measurements were natural log transformed in order to approximate normal distributions. Post-task measurements were not used due to our focus on reactivity only. Due to technical issues, 7% of the raw ANS data were lost or unreliable and were therefore excluded from the analyses.

2.2.4. Potential control variables

We adjusted all stress response models for the following control variables: risk group, sex and SES. We furthermore tested whether any of the following control variables correlated significantly with the slope and/or intercept of each of the stress response models: age; BMI; having practiced sport or had lunch on the day of the lab session; having drunk tea, coffee, coca-cola or energy drinks in the past 24 h; having used pills for concentration or sleeping in the past 24 h; having used alcohol, cigarettes, marijuana or other drugs in the past 24 h; having had a headache, tiredness, a cold, been ill or had an infection in the past week; having allergies; whether vitamins or other dietary supplements were currently used; whether following a diet; whether medicine was used; and for girls, the menstrual cycle phase (i.e. luteal or follicular) and whether oral contraceptives were currently being used. For the cortisol response model only, we additionally tested whether it was necessary to control for having consumed milk or yogurt in the past 24 h; and having had a toothache, bleeding gums or a wound in the mouth.

2.3. Procedure

Home-based assessments took place annually during five consecutive springs, starting in March 2006, during which participants filled in a set of questionnaires, including an assessment of personality characteristics. A well-trained research assistant was present to answer questions during the home assessment, which lasted approximately three hours in total.

For the laboratory session, the adolescents and their parents were first sent invitation letters. In the letters, the laboratory session was introduced and adolescents were invited to participate in the session that took place at one of the two participating faculties (the Faculty of Social and Behavioural Sciences, Utrecht University, and the Faculty of Psychology and Education, VU University Amsterdam), depending on the residence of the participant. Moreover, the letters contained information about the aim and duration of the session, the planned tasks, and practical issues, including the full refunding of travel expenses and the compensation for participation. Next, the adolescents were contacted by telephone to confirm or decline participation and to schedule an appointment. On average two weeks prior to the planned laboratory session, con-

firmation letters were sent to the adolescents and their parents. Both parents and adolescents were asked to give their informed consent and assent, respectively.

The laboratory session was conducted by a trained experimenter, and took place in a specially equipped room that was kept at room temperature ($M = 20.43°$ Celsius, $SD = 1.00$) with an average air humidity of 54% ($SD = 8.19$). The session always took place in the afternoon and lasted approximately three hours in total. During the session, participants performed three computer tasks and started subsequently, between 3 and 4 pm, with the PST protocol. Directly after completing the laboratory session, adolescents received a gift certificate of 50 Euros. The RADAR study was approved by the medical ethics committee of Utrecht University.

2.3.1. Public speaking task

During the Leiden PST (Westenberg et al., 2009), participants delivered a speech in front of a pre-recorded audience consisting of age-matched peers and a teacher. A camera was installed and participants were told that they were being recorded and would be judged later by an age-matched class.

In the confirmation letter that was sent two weeks prior to the laboratory visit, the adolescents were informed about the details of the PST and were asked to prepare a five-minute speech about types of movies they liked, a subject that adolescents from various backgrounds were expected to relate to. The adolescents thus knew in advance that they were to perform a speech and were encouraged to prepare for it. This was done to avoid confounding task effects of unpredictability of the test situation, because we aimed to measure reactivity to social evaluation specifically (see also Dickerson & Kemeny, 2004; Erdmann & Baumann, 1996).

The task contained five phases: baseline (20 min, of which the final five standing), anticipation (three minutes standing), preparation (five minutes standing), speech delivery (five minutes standing), and post-task (30 min; see Fig. 1). During the baseline phase, participants watched a nature documentary. The anticipation phase consisted of the experimenter reminding participants of the upcoming task and detailed instructions were repeated. Subsequently, participants were given time to prepare the speech (preparation phase) and were then asked to deliver the speech (speech delivery phase). Directly after the speech, during the first 10 min of the post-task phase, participants filled in a questionnaire on potential control variables, e.g., caffeine, medication intake or drug use, night rest, and illnesses. Finally, participants watched another nature documentary. A small adaptation was made to the Westenberg protocol: whereas in the original protocol the experimenter entered the test room for an interview on potential control variables directly after the speech, in the current protocol participants filled in a questionnaire on these variables so that participants were alone in the test room during the entire session after installation of the equipment and some practical instructions (such as where to sit and stand, and that further communication would take place via an intercom). From the start of the baseline documentary, the experimenter communicated at predefined moments with participants through an intercom. Validity of the Leiden PST has been examined in Dutch adolescents (Westenberg et al., 2009).

2.4. Data analysis

We first examined descriptive statistics of and correlations between all variables. For the main analyses, we estimated eight dual process growth curve models. We began by estimating a single basic growth curve model for each of the stress response indices (i.e. cortisol, HR, PEP and HRV). The intercept was set at the first measurement point (prior to the stress-inducing part of the PST) as an index of baseline (resting) stress levels. We then corrected the stress response growth curves for the covariates risk, sex and SES,
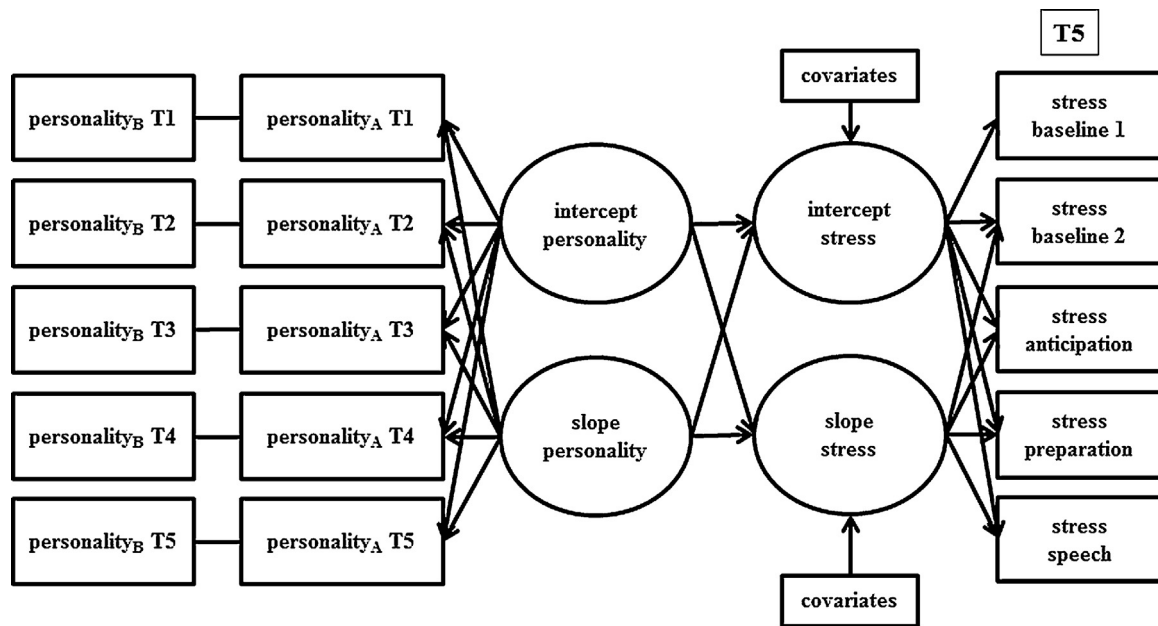
Fig. 2. Depiction of the model tested.
Caption: Eight dual latent growth curve models were tested in which the intercept (mean level) and slope (change) of personality traits across five years of adolescence (T1–T5) predicted the intercept (baseline) and slope (reactivity) of physiological stress levels. Separate models were tested for personality trait A (e.g. extraversion), controlling for the time-varying covariate personality trait B (e.g. neuroticism) predicting cortisol, heart rate, pre-ejection period and heart rate variability at baseline and reactivity. Identical models were tested with neuroticism as personality trait A and extraversion as personality trait B. Covariates differed per model (see Table 2).

and tested whether it was necessary to correct for additional covariates (see *potential control variables* above) by regressing the growth curve parameters on the covariates. In order to check whether menstrual cycle phase or oral contraceptive use significantly influenced the outcomes we reran all final models in girls only. The growth curve corrected for covariates was then used in the further analyses.

In order to examine the relation between personality (mean level and change across adolescence) and stress reactivity, we added a second growth curve (estimating personality) to the model. The intercept for the personality growth curve was set at the third (middle) measurement point as an indication of mean level personality during adolescence because the change in personality scores was approximately linear. We regressed the intercept and slope of the stress response growth curve on the intercept and slope of the personality growth curve. Separate models were fitted for extraversion and neuroticism on each of the four stress reactivity indices. To account for potential overlap between extraversion and neuroticism, the model of extraversion was adjusted for neuroticism by including the neuroticism scores as time-varying covariates (similar procedure for neuroticism, with extraversion scores serving as time-varying covariates). See Fig. 2 for a depiction of the model tested.

All models used maximum likelihood estimation. To assess model fit, we used the Comparative Fit Index (CFI) and Tucker Lewis Index (TLI; both with values >0.90 indicating acceptable and >0.95 indicating good fit) and the Standardized Root Mean Squared Residual (SRMR; with values <0.08 indicating good fit; Hu & Bentler, 1999). Results reported were considered statistically significant at $p < 0.05$. All analyses were performed in Mplus version 6.11 (Muthén & Muthén, 1998–2011).

3. Results

3.1. Preliminary analyses

The means, standard deviations and ranges of the stress and personality variables are depicted in Figs. 1 and 3, respectively.
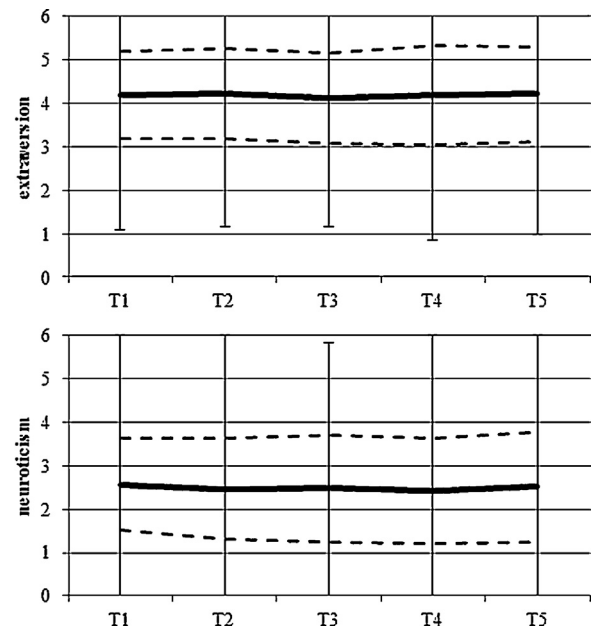
Fig. 3. Illustration of descriptive statistics for personality variables across adolescence.
Caption: average (solid line), standard deviation (dotted lines) and range (bars) statistics are depicted for extraversion (top) and neuroticism (bottom) across the five measurement points during adolescence (T1–T5). Participants were aged 13 years on average at T1 and 17 years on average at T5.

Zero-order correlations of all study variables are shown in Table 1. Extraversion and neuroticism scores were strongly and negatively correlated and remained relatively stable across adolescence.

3.2. Preliminary models and model fit

The preliminary stress growth curve models indicated that the slopes for cortisol and heart rate were positive and the slope for PEP

Table 1

Pearson correlations between study variables.

	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
1. C-B1	**0.77**	**0.64**	**0.52**	**0.54**	0.00	−0.03	−0.05	−0.07	−0.01	0.03	0.03	0.05	−0.04	−0.01	−0.03	−0.03	0.06	0.04	0.08	0.01	0.06	−0.02	−0.01	−0.13	−0.04	−0.11
2. C-B2	–	**0.80**	**0.70**	**0.71**	0.07	0.11	0.07	0.09	−0.03	−0.07	−0.09	−0.09	0.02	0.01	0.03	0.03	0.03	−0.09	−0.03	−0.07	−0.04	−0.07	−0.01	−*0.13*	−0.03	−0.12
3. C-A		–	**0.86**	**0.83**	0.08	*0.14*	0.10	0.11	−0.11	**−0.16**	**−0.17**	−*0.13*	−0.02	0.01	0.00	0.00	0.02	−0.07	0.06	−0.03	−0.05	−0.08	−0.03	−*0.12*	−0.04	−0.08
4. C-Pr			–	**0.91**	0.10	**0.17**	*0.12*	**0.16**	−0.08	−*0.14*	−*0.15*	−0.11	−0.09	−0.05	−0.08	−0.08	0.03	−0.02	0.08	0.02	0.03	−0.09	−0.02	−0.10	−0.03	−0.10
5. C-S				–	0.08	*0.13*	0.10	0.11	−0.05	−0.09	-0.10	−0.08	−0.06	−0.04	−0.07	−0.07	0.02	−0.06	0.03	−0.04	−0.01	−0.08	−0.05	−*0.13*	−0.03	−0.12
6. H-B					–	**0.78**	**0.79**	**0.70**	**−0.17**	**−0.19**	**−0.18**	**−0.21**	0.06	0.11	−0.02	−0.02	−0.09	−0.06	−0.08	−0.11	−0.08	0.06	0.06	0.01	0.04	0.06
7. H-A						–	**0.95**	**0.88**	**−0.20**	**−0.35**	**−0.36**	**−0.37**	−0.02	0.06	−0.01	−0.01	−*0.12*	−*0.13*	**−0.16**	**−0.15**	**−0.17**	0.11	0.08	0.07	0.08	*0.12*
8. H-Pr							–	**0.90**	**−0.20**	**−0.33**	**−0.36**	**−0.36**	−0.01	0.07	−0.03	−0.03	−*0.15*	−*0.12*	**−0.17**	**−0.17**	**−0.17**	*0.12*	0.09	0.09	0.08	0.11
9. H-S								–	**−0.20**	**−0.37**	**−0.36**	**−0.44**	−0.04	0.04	−0.01	−0.01	−*0.12*	−*0.12*	**−0.15**	−*0.12*	−*0.13*	*0.14*	0.08	0.09	0.08	0.10
10. P-B									–	**0.84**	**0.78**	**0.67**	0.01	−0.06	−0.08	−0.08	0.08	−0.03	0.00	0.08	0.08	−0.05	0.11	0.09	0.04	0.00
11. P-A										–	**0.93**	**0.83**	0.02	−0.06	−0.07	−0.07	*0.14*	0.01	0.02	0.08	0.12	−0.05	0.11	0.06	0.04	−0.02
12. P-Pr											–	**0.87**	0.00	−0.07	−0.02	−0.02	0.11	0.06	0.07	*0.14*	0.11	−0.11	0.02	−0.01	−0.03	−0.08
13. P-S												–	0.03	−0.04	−0.02	−0.02	0.10	0.04	0.05	0.12	0.07	−0.11	0.04	0.01	−0.02	−0.06
14. V-B													–	**0.80**	**0.86**	**0.86**	−0.02	−0.01	−0.06	−0.01	−0.03	0.10	0.09	0.01	0.03	0.02
15. V-A														–	**0.80**	**0.80**	−0.02	−0.02	−0.10	−0.05	−0.06	0.03	0.09	0.04	0.05	0.04
16. V-Pr															–	**1.00**	−0.02	0.01	−0.07	−0.03	−0.07	0.09	0.08	0.04	0.06	0.04
17. V-S																–	−0.02	0.01	−0.07	−0.03	−0.07	0.09	0.08	0.04	0.06	0.04
18. E1																	–	**0.56**	**0.51**	**0.51**	**0.47**	**−0.47**	**−0.23**	**−0.17**	**−0.23**	**−0.22**
19. E2																		–	**0.66**	**0.59**	**0.55**	**−0.25**	**-0.40**	**−0.25**	**−0.27**	**−0.24**
20. E3																			–	**0.66**	**0.64**	**−0.28**	**-0.30**	**−0.43**	**−0.38**	**−0.33**
21. E4																				–	**0.74**	**−0.27**	**−0.27**	**−0.29**	**−0.45**	**−0.34**
22. E5																					–	**−0.24**	**−0.17**	**−0.23**	**−0.33**	**−0.41**
23. N1																						–	**0.55**	**0.50**	**0.45**	**0.46**
24. N2																							–	**0.68**	**0.60**	**0.56**
25. N3																								–	**0.74**	**0.71**
26. N4																									–	**0.73**
27. N5																										–

Note: **Bold** values indicate statistical significance at *p* < 0.01; values in *italics* indicate statistical significance at *p* < 0.05. C = cortisol; H = heart rate; P = pre-ejection period; V = heart rate variability; B = baseline; A = anticipation; Pr = preparation; S = speech; E = extraversion; N = neuroticism.

Table 2
Mean and variance estimates for the stress growth curves and model fit statistics for all models.

Cortisol

Model	Fit			Mean			Variance		
	CFI	TLI	SRMR	Est	SE	p	Est	SE	p
Basic	0.99	0.98	0.03						
Intercept				1.56	0.03	<0.001	0.18	0.02	<0.001
Slope				0.03	0.01	<0.001	0.00	0.00	<0.05
Covariates[a]	0.99	0.99	0.04						
Extraversion	0.99	0.99	0.04						
Neuroticism	0.96	0.97	0.06						
Heart rate									
	Fit			**Mean**			**Variance**		
	CFI	TLI	SRMR	Est	SE	p	Est	SE	p
Basic	0.93	0.91	0.11						
Intercept				79.25	0.61	<0.001	107.49	8.68	<0.001
Slope				3.18	0.20	<0.001	16.50	1.28	<0.001
Covariates[b]	0.93	0.95	0.05						
Extraversion	0.96	0.97	0.05						
Neuroticism	.95	.96	.05						
Pre-ejection period									
	Fit			**Mean**			**Variance**		
	CFI	TLI	SRMR	Est	SE	p	Est	SE	p
Basic	0.96	0.94	0.13						
Intercept				120.65	1.04	<0.001	263.85	24.67	<0.001
Slope				−3.15	0.31	<0.001	20.52	2.86	<0.001
Covariates[c]	0.98	0.98	0.05						
Extraversion	0.98	0.98	0.05						
Neuroticism	0.98	0.98	0.05						
Heart rate variability									
	Fit			**Mean**			**Variance**		
	CFI	TLI	SRMR	Est	SE	p	Est	SE	p
Basic 1	0.94	0.93	0.17						
Intercept				6.05	0.06	<0.001	0.88	0.09	<0.001
Slope				0.03	0.02	<0.05	0.00	0.01	0.74
Basic 2	1.00	1.00	.05						
Intercept				5.91	0.06	<0.001	0.81	0.01	<0.001
Slope				0.28	0.04	<0.001	0.00	0.00	–
Slope^2				−0.08	0.01	<0.001	0.00	0.00	0.46
Covariates[d]	1.00	1.00	0.05						
Extraversion	1.00	1.00	0.04						
Neuroticism	0.99	0.99	0.05						

Note:CFI = Comparative Fit Index; TLI = Tucker Lewis Index; SRMR = Standardized Root Mean Squared Residual; Est = estimate; SE = standard error.

[a] The cortisol growth curve was corrected for risk group, sex, socioeconomic status, body mass index, age, medicine use and having smoked cigarettes in the past 24 h. In girls only, oral contraceptive use had a significant negative effect on the cortisol slope, however, results of the final models were similar when controlling for this effect (statistics available upon request). Menstrual cycle phase did not significantly influence cortisol (intercept or slope).

[b] The heart rate growth curve was corrected for risk group, sex, socioeconomic status, having consumed tea, cola and alcohol in the past 24 h, having smoked cigarettes in the past 24 h, having felt tired or had a cold in the past week, and having allergies. In girls only, oral contraceptive use had a significant and positive effect on the intercept of heart rate, but the results of the final models were similar when controlling for this effect. Menstrual cycle phase did not significantly influence heart rate (intercept or slope).

[c] The pre-ejection period growth curve was corrected for risk group, sex, socioeconomic status, body mass index, having consumed coffee and alcohol in the past 24 h, having had a headache in the past week, and having allergies. In girls only, menstrual cycle phase and oral contraceptive use did not significantly influence pre-ejection period (intercept or slope).

[d] The heart rate variability growth curve was corrected for risk group, sex, socioeconomic status, body mass index, having consumed tea, alcohol and marijuana in the past 24 h and current use of vitamins. In girls only, neither oral contraceptive use nor menstrual cycle phase were significantly related to heart rate variability (intercept or slope).

was negative, as expected. The slope for HRV rose initially and then dropped slightly at the last time point (during the speech delivery part of the PST). Therefore we included a quadratic slope in the HRV models, which significantly improved the fit (see Table 2). The estimates of the variances of the cortisol, heart rate and PEP growth curve intercepts and slopes were statistically significant, indicating significant between-individual differences. For the HRV model, the estimate of the variance of the intercept was statistically significant, but the estimates for the linear and quadratic slopes were not. Therefore, in subsequent steps, we only examined relations between personality and the HRV intercept (HRV at baseline). See Table 2 for the means and variances of the intercepts and slopes of all stress growth curve models, as well as the fit statistics for the basic stress growth curve models, the stress growth curve models corrected for covariates, and the final models includ-

ing the extraversion or neuroticism growth curve. The final models were corrected for neuroticism or extraversion, respectively, as time-varying covariates. Pertaining to the cortisol growth curve, the average maximum response was during the anticipation phase, and cortisol levels decreased thereafter (see Fig. 1). This is consistent with previous studies in adolescents using the Leiden PST (van den Bos et al., 2014). Therefore, we utilized only the first three cortisol samples (i.e. samples 2–4, pertaining to baseline 1, baseline 2 and anticipation) in the cortisol growth curve estimation.

Sex significantly influenced cortisol, HR, PEP and HRV measures. Boys showed higher cortisol (Est = −0.13, SE = 0.06, $p < 0.05$) and higher PEP (Est = −4.65, SE = 2.02, $p < 0.05$) levels at baseline. Boys showed stronger cortisol reactivity (Est = −0.22, SE = 0.01, $p < 0.05$) and stronger HRV reactivity (Est = −0.32, SE = 0.09, $p < 0.001$), and girls showed stronger HR reactivity (Est = 1.80, SE = 0.31, $p < 0.001$).

Table 3
Statistics for the fit, means and variances of the extraversion and neuroticism growth curves.

	Fit			Means			Variances		
	CFI	TLI	SRMR	Est	SE	*p*	Est	SE	*p*
Extraversion	1.00	1.00	0.03						
Intercept				5.13	0.07	<0.001	0.55	0.05	<0.001
Slope				−0.03	0.03	0.33	0.03	0.01	<0.001
Neuroticism	0.98	0.98	0.04						
Intercept				4.41	0.12	<0.001	0.69	0.06	<0.001
Slope				−0.01	0.06	0.82	0.04	0.01	<0.001

Sex was not related to baseline HR levels, baseline HRV levels or PEP reactivity.

The extraversion and neuroticism growth curve slopes were not significantly different from zero. However, both evidenced significant slope variances ($p < 0.001$), indicating significant inter-individual differences which warrants further examination of correlates of these slopes (see e.g. Llabre, Spitzer, Saab, & Schneiderman, 2001; Muthén & Muthén, 1998–2010). For the statistics of the means and variances of the personality growth curves, see Table 3.

Sex was related to the intercept of the extraversion growth curve and to both the intercept and slope of the neuroticism growth curve. Girls were slightly more extraverted (Est = 0.18, SE = 0.09, $p < 0.05$) and scored higher on neuroticism at T3 (Est = 0.49, SE = 0.10, $p < 0.001$). Girls also showed less decline in neuroticism across adolescence (Est = 0.09, SE = 0.03, $p < 0.01$). Sex was not related to the slope of the extraversion growth curve.

3.3. Cortisol

Pertaining to extraversion, the intercept of extraversion (i.e. extraversion scores at the third measurement point, during the middle of adolescence) was not significantly related to the intercept of the cortisol growth curve. It was significantly associated with the slope of the cortisol growth curve, such that more extravert adolescents showed less cortisol reactivity. See Fig. 4A for a depiction of this effect. The slope of extraversion was not significantly related to either the intercept or slope of cortisol reactivity. Regarding neuroticism, mean level and change in neuroticism scores across adolescence were not significantly related to cortisol at baseline or cortisol reactivity. The statistics for all final models are depicted in Table 4.

3.4. Heart rate

Neither extraversion (intercept and slope) nor neuroticism (intercept and slope) were related to HR at baseline or HR reactivity.

3.5. Pre-ejection period

Pertaining to extraversion, neither the intercept nor the slope of the extraversion growth curve was related to the intercept or the slope of the PEP response curve. Regarding neuroticism, the intercept of the neuroticism growth curve (indicating neuroticism scores at the third measurement) was significantly associated with the slope of the PEP response curve such that adolescents who scored higher on neuroticism showed stronger PEP reactivity. An illustration of this effect is depicted in Fig. 4B.

3.6. Heart rate variability

The pattern of HRV reactivity did not show significant inter-individual variance in our sample. Therefore, we only examined the relation between personality and HRV at baseline. Neither extraversion (intercept and slope) nor neuroticism (intercept and slope) were related to HRV at baseline.

4. Discussion

In the current study, we examined individual differences in mean level and change in extraversion and neuroticism across adolescence in relation to individual differences in physiological stress reactivity to social evaluation in a large sample of adolescents from the general population. Most of the comparisons revealed no relation between personality and physiological stress reactivity. We did observe statistically significant relations between mean level extraversion and lower cortisol reactivity and between mean level neuroticism and greater PEP reactivity, both of which were in line with our hypotheses.

Adolescence is regarded as a critical period in the development of personality traits (i.e. Klimstra et al., 2009). Therefore, we examined both mean level (intercept) and change (slope) in personality across adolescence using growth curve models. Both curves evidenced significant inter-individual variance, which warranted further examination of correlates of the slopes. Interestingly, change in personality was not related to individual differences in physiological stress reactivity. Seemingly, in middle adolescence at least, physiological stress reactivity is only related to mean level personality traits, and not to change in these traits. In addition to genetic influences, personality change is generally considered to be, to a large extent, the result of changing contexts (see e.g. Kogan, 1990; Roberts et al., 2006). Shifts in mean-level personality traits may, for example, be related to increased contact with peers in adolescence or to the transition from school to work in early adulthood. A hallmark of adolescence is that adolescents' attention becomes increasingly focused on social evaluation (Westenberg et al., 2004), therefore we might expect this to coincide with increases in physiological stress reactivity, especially in response to a social evaluative stressor. Indeed, evidence suggests that adolescents show heightened stress reactivity compared to children and adults (e.g. Dahl & Gunnar, 2009; Klein & Romeo, 2013). Thus, while the data from the present study show that change in personality does not seem to be related to stress reactivity in later adolescence, perhaps a more pertinent question would be to ask whether change in personality across adolescence is related to change in stress reactivity *across adolescence*. Instead of measuring stress reactivity at only one point, as we did, it might be more informative to have multiple measurements of stress reactivity during adolescence. Such a design may very well yield relations between change in personality and change in stress reactivity. Since this is the first study to examine the relation between change in personality and any measure of stress reactivity, further research will need to corroborate our findings, and verify whether they hold across longer time spans, for example, from adolescence through middle adulthood.

In contrast to our expectations and results from previous longitudinal studies of personality change across adolescence (e.g. Branje et al., 2007; Klimstra et al., 2009), the slopes of the personality growth curves in our study were not significantly different from zero. This indicated that personality did not change across the years studied. One reason for this may be that the age ranges in the previous studies mentioned were somewhat broader: 11–17 years in the Branje et al. (2007) study and 12–20 years in the Klimstra et al. (2009) study. The total age range in the present study was 13–17 years. Furthermore, both previous studies found fairly small slope effects, especially for neuroticism (i.e. no effect in the Branje et al. (2007) study and a small effect for boys only in the Klimstra et al. (2009) study). As the latter study had a much larger sample than the present ($n = 1313$), our study may have simply
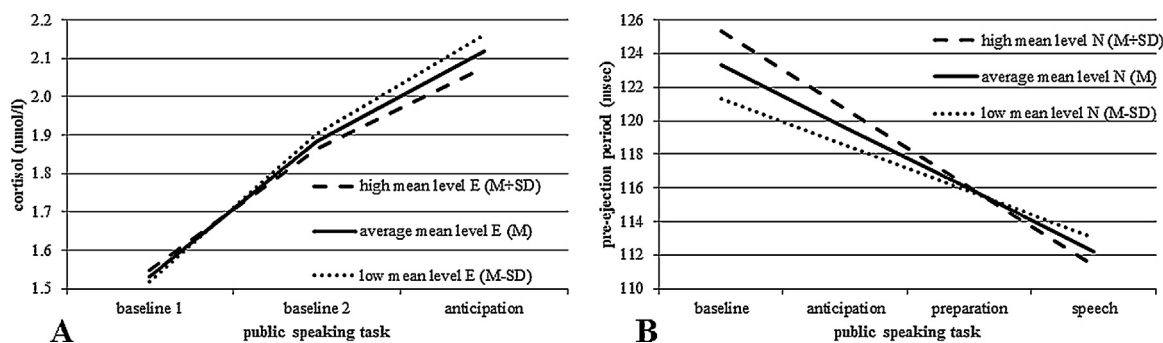
Fig. 4. Effects of mean level extraversion on cortisol reactivity (A) and mean level neuroticism on pre-ejection period reactivity (B).
Caption: A: Graphic depiction of the effect of mean level extraversion on cortisol reactivity. B: Graphic depiction of the effect of mean level neuroticism on pre-ejection period reactivity.
Note: Cortisol values are natural log transformed. E = extraversion; N = neuroticism.

Table 4
Statistics for effects of the intercept and slope of the stress response curves regressed on the intercepts and slopes of the personality growth curves.

Cortisol						Heart rate					
Intercept			Slope			Intercept			Slope		
Est	SE	p	Est	SE	p	Est	SE	p	Est	SE	p
Extraversion											
0.02	0.05	0.69	**−0.02**	**0.01**	**0.04**	−1.75	0.94	0.06	−0.19	0.26	0.47
−0.20	0.23	0.39	0.01	0.04	0.88	2.24	4.89	0.65	−0.31	1.29	0.81
0.09	0.04	0.01	0.17	0.08	0.04	0.04	0.02	0.07	0.09	0.02	<0.001
Neuroticism											
−0.04	0.04	0.29	0.00	0.01	0.97	−0.07	0.92	0.94	0.32	0.26	0.22
−0.26	0.22	0.24	0.06	0.04	0.15	0.98	5.40	0.86	−3.05	1.59	0.05
0.10	0.04	0.01	0.16	0.09	0.06	0.03	0.02	0.13	0.10	0.03	<0.001
Pre-ejection period						**Heart rate variability**					
Intercept			**Slope**			**Intercept**					
Est	SE	p	Est	SE	p	Est	SE	p			
Extraversion											
1.95	1.55	0.21	−0.18	0.44	0.69	−0.04	0.10	0.66			
4.14	8.29	0.62	−0.72	2.38	0.76	0.22	0.47	0.64			
0.08	0.04	0.04	0.14	0.04	0.001	0.08	0.03	0.02			
Neuroticism											
2.42	1.58	0.13	**−1.13**	**0.045**	**0.01**	0.03	0.09	0.77			
−1.64	8.74	0.85	4.17	2.62	0.11	0.27	0.49	0.58			
0.07	0.03	0.04	0.17	0.05	0.001	0.08	0.03	0.02			

Note: I = intercept; S = slope; Est = estimate, SE = standard error; **bold** values indicate statistical significance at $p < 0.05$. Explained variance for the latent constructs of the intercepts and slopes of the stress reactivity growth curves are given in R^2.

lacked the power to detect such small effects. Consistent with this study, preliminary analyses in our study showed that boys showed stronger decreases in neuroticism across adolescence. Pertaining to extraversion, Branje et al. (2007) observed a positive growth curve slope for girls across adolescence and a negative slope for boys. Our findings are in contrast to this in that sex did not significantly influence the slope of the extraversion growth curve.

Personality theory (Eysenck, 1967) predicts that individuals scoring high on extraversion would be less prone to stress, that is, would respond (physiologically) less strongly to a stressor. Individuals scoring high on neuroticism would be more prone to stress, that is, would respond more strongly to a stressor. Our findings are somewhat in line with these expectations. Individuals scoring higher on extraversion showed lower cortisol reactivity to the social evaluative stressor (i.e. had more moderate cortisol slopes). This effect was evident for individuals with higher mean level extraversion scores (i.e. the intercept of the extraversion growth curve, which was set at the middle of adolescence). Regarding neuroticism, individuals scoring higher on this trait showed stronger PEP responses to the stressor. Similar to extraversion, this was evident for mean level neuroticism scores. Contrary to our expectations, extraversion was not related to HRV, PEP or heart rate

reactivity and neuroticism was not related to HRV, heart rate or HPA axis reactivity. Thus, the majority of the relations tested in the present study indicated no relation between personality and stress reactivity. This, coupled with previous findings of no relation between these constructs in adolescents (i.e. Laceulle et al., 2015; Zahn et al., 1994), leads us to conclude that, while there may be some physiological basis to personality, the effects are quite marginal.

It is interesting that the two personality constructs examined in the present study were associated with different stress systems: extraversion with HPA axis reactivity and neuroticism with PEP reactivity. If there is only a weak relation between personality and stress reactivity, why was it that these particular relations reached statistical significance? This may not simply be random. First, it is striking that Eysenck (1967) already hypothesized these particular associations. Specifically, he proposed two sets of neural loops. The first was a cortical-reticular loop implicated in arousal and inhibition and the introversion-extroversion construct. Secondly, he proposed a loop involving the limbic brain areas which are implicated in the processing of emotions and sympathetic activation and thus related to neuroticism/emotionality (Eysenck, 1967). At the time, there was not much of an empirical basis to Eysenck's pro-

posal. Contemporary scientific theories and understanding of the brain, stress systems and personality, however, may also provide some evidence in the same line.

Regarding extraversion, a key component of this construct is that individuals are more comfortable and less inhibited in social situations. People scoring high on extraversion prefer being with others as opposed to being alone, and enjoy interacting with other people and attending social gatherings (Costa & McCrae, 1988; Eysenck & Eysenck, 1985). The functioning of the HPA axis is highly sensitive to social developmental influences: there is fairly robust evidence that social support has a buffering effect on the (developing) HPA axis (Gunnar & Donzella, 2002; Hostinar & Gunnar, 2013). Thus, social support seems to be a common factor in both extraversion and HPA axis functioning, and may underlie the development of both. Another common factor in these constructs is emotion regulation. Previous studies have associated extraversion with the experience and expression of positive emotions (see Jonassaint et al., 2009). Individuals who scored high on extraversion were found to be less likely to use emotion regulation strategies such as suppression because they were not as sensitive to social rejection as those scoring high on introversion. Therefore, these individuals were less likely to use suppression to distance themselves from others who could reject them (John & Gross, 2004). In this way, adolescents who score high on extraversion may be more able to regulate their emotions effectively. HPA axis functioning is also strongly related to emotion regulation: individuals who are better able to regulate their emotions tend to show lower HPA axis reactivity (Stansbury & Gunnar, 1994). As such, higher scores on extraversion and lower HPA axis reactivity have been associated with effective emotion regulation capabilities.

Neuroticism is defined as the tendency to experience negative emotions (Costa & McCrae, 1988; Eysenck, 1967) and therefore individuals who have more neurotic traits may be less able to effectively regulate their emotions. Indeed, physiological and neurobiological studies have confirmed less adequate emotion regulation capabilities in those scoring high on neuroticism (Ormel et al., 2013). Furthermore, neuroimaging studies have provided evidence for reduced connectivity in limbic brain areas in individuals scoring high on neuroticism. This is proposed to underlie hyper-reactivity of the amygdala to stress and in turn manifest as anxiety, high stress reactivity and negative emotionality in these individuals (Depue, 2009; Ormel et al., 2013). Neuroticism may be related to SNS responding in particular because of the following: according to the Jacksonian principle of dissolution there is a hierarchy of ANS response patterns (Jackson, 1958; Porges, 2007). The phylogenetically newest branch of the ANS, the PNS, is immediately deactivated when a stressor is perceived by the individual. If this response is insufficient, the phylogenetically older branch of the ANS, the SNS, is activated, entailing the fight-or-flight response. Emotion regulation and the ability to self-regulate are thought to be controlled by the PNS (Porges, 2007). Because individuals scoring high on neuroticism are thought to not be able to regulate their emotions effectively, it can be hypothesized that their PNS may function less optimally. Indeed, empirical research has provided evidence that individuals with poor emotion regulation capabilities show lower (less-optimally functioning) vagal tone (e.g. Ormel et al., 2013; Porges et al., 1994). Possibly, such diminished PNS functioning sets off an exaggerated SNS response, indexed by the PEP response in the current study. This may also explain why extraversion was not related to SNS functioning in our study, as extraverts are more effective in regulating their emotions. Of course, this possibility needs to be confirmed empirically. In the current study, HRV, an index of PNS functioning, was not related to neuroticism as we hypothesize. However, the inter-individual variances of the slopes of HRV reactivity were not statistically significant, and therefore it was not justifiable to examine relations between personality

and HRV reactivity. We were able to examine relations between personality and HRV functioning at baseline, which were not significant. We would expect, though, that personality would be related to HRV *reactivity*, and therefore our hypothesis remains to be tested in future studies.

The observed relation between PEP hyper-reactivity and neuroticism in the present study is furthermore in line with research by Beauchaine and his colleagues. Beauchaine (2012) proposed that PEP indexes approach behavior and sensitivity to reward. Findings from his group showed that youth with externalizing problems tend to exhibit blunted PEP responses. Because neuroticism is closely linked to internalizing problems (e.g. Ormel et al., 2013), our findings of PEP hyper-reactivity in those scoring higher on neuroticism are in line with this previous research.

Two previous studies examined extraversion and neuroticism in relation to physiological stress reactivity in adolescents, and did not find evidence for a relation between personality and HR reactivity (Zahn et al., 1994) or HPA axis reactivity (Laceulle et al., 2015). Like Zahn et al. (1994), we did not observe any relation between personality and HR reactivity. Our findings are in contrast to the Laceulle and colleagues (2015) study. That study was fairly similar to the present study: both used relatively large samples of adolescents of approximately similar ages (i.e. 17 years on average at the time of the PST in the present study and 16 years on average in the Laceulle et al., 2015 study), and both utilized a psychosocial stressor. However, in the Laceulle and colleagues study, the psychosocial stress procedure was modeled after the Trier Social Stress Test (Kirschbaum et al., 1993), which is characterized by social evaluation and uncontrollability, both of which are pivotal in eliciting a physiological stress response (see Dickerson & Kemeny, 2004). In contrast, in the Leiden PST, implemented in the present study, the element of uncontrollability is eliminated. Adolescents were informed beforehand of the nature of the task they would be asked to perform, and were even encouraged to prepare for it. Consequently, it is plausible that stress elicited by the PST was mainly driven by the threat of social evaluation. Previous research indicates that individuals who are more extravert are less sensitive to social rejection (John & Gross, 2004). Thus, perhaps the fundamentality of social evaluation *only* in the PST explains the observation (in the present study) of more extravert adolescents showing lower cortisol reactivity, and the absence of such an observation using the psychosocial stressor modeled after the TSST (in the Laceulle and colleagues study). Of course, this hypothesis will need to be confirmed in other samples, but may be an interesting possibility, with consequences for both personality and stress physiology research.

The current study should be considered in light of the following. The RADAR adolescents were not randomly drawn from the general Dutch population. Only 6% of the adolescents came from low SES families, and almost all were of Dutch origin. Both physiological stress reactivity and personality have been shown to be related to SES and ethnicity (Jonassaint, Siegler, Barefoot, Edwards, & Williams, 2011; Musante, Treiber, Kapuku, Moore, Davis, & Strong, 2000), however, in our sample this was not the case. As yet, our findings are only generalizable to adolescents from Dutch, middle/high SES backgrounds. Furthermore, the original sample was selected based on a higher prevalence of symptoms of externalizing problems. However, this risk score was controlled in all analyses, and therefore we do not expect that it influenced the present findings. Secondly, non-participation in the laboratory tests was related to personality such that the decliners were slightly less extravert than the participants. Although this selective drop-out may seem logical because adolescents had to travel by public transport to the university where they had never been before, and they knew in advance they had to do a public speaking task, this implicates that the variance in extraversion in our sample may be smaller than in the general population. Third, we chose to focus on physiological stress

reactivity in the present study, and therefore did not take stress recovery into account. Finally, we chose to focus on the personality traits extraversion and neuroticism in the current study because the majority of research on the relation between stress reactivity and personality has been on these constructs only. Furthermore, given our sample size and the number of associations already examined, we did not wish to further increase our chance of finding and reporting Type 1 errors. However, there is substantial theoretical (e.g. Rothbart, Ahadi, & Evans, 2000), as well as beginning empirical evidence for a relation between stress reactivity and conscientiousness or effortful control (Oldehinkel, Hartman, Nederhof, Riese, & Ormel, 2011; Spangler & Friedman, 2015). Therefore it would be interesting for future studies on this topic to include measures of conscientiousness.

In conclusion, the current study provides some evidence for an underlying physiological basis to personality, though the effects of this seem to be quite marginal. Consistent with early theories of personality (Eysenck, 1967), adolescents scoring high on extraversion showed lower cortisol stress reactivity, while adolescents scoring high on neuroticism showed higher PEP stress reactivity in response to a social evaluative stressor. In contrast to our hypotheses, extraversion was not related to HRV, PEP or heart rate reactivity, and neuroticism was not related to HRV, heart rate or HPA axis reactivity. In contrast to our expectations, change in personality across adolescence was not related to stress reactivity. To our knowledge, this study was the first to examine both mean level and change in personality across adolescence in relation to stress reactivity, therefore, replication in other adolescent populations is needed.

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