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Chapter 6

Longitudinal relationships between proactive and reactive aggression and autonomic nervous system reactivity in delinquent male adolescents: the moderating effect of posttraumatic stress symptoms

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ABSTRACT

Background: In this study the longitudinal relationships between proactive / reactive aggression and autonomic nervous system (ANS) (re)activity were studied, as well as the moderating effect of posttraumatic stress disorder (PTSD) symptoms.

Methods: Delinquent males were assessed twice over a 5-year period in adolescence. We related self-reported proactive/ reactive aggression and PTSD symptoms to heart rate (HR) and heart rate variability (HRV), measured at rest and in response to a public speaking task.

Results: In line with the polyvagal theory, a longitudinal relationship was observed between attenuated HRV reactivity (decreased vagal withdrawal) and proactive but not reactive aggression. PTSD symptoms was a moderating factor; the relationship between HRV reactivity and proactive aggression was only present at low levels of PTSD symptoms.

Conclusions: This study adds to the advancing knowledge on distinctive neurobiological profiles underlying subtypes of aggression.

INTRODUCTION

There are indications that subtypes of juvenile aggressive behavior, like proactive and reactive aggression, are related to differential profiles of autonomic nervous system (ANS) (re)activity. Proactive ('cold-blooded') aggression has been related to high resting heart rate variability (HRV) and attenuated reactivity of heart rate and HRV, whereas reactive ('emotional') aggression has been related to decreased resting HRV (De Vries-Bouw et al., 2012; Murray-Close & Rellini, 2011; Scarpa et al., 2009). When studying these relationships longitudinally, more insight can be obtained in the dynamics of the relationships over time, as well as in the stability of ANS and aggression parameters. Furthermore, it has been proposed in theoretical models that relationships between ANS (re)activity and aggression are moderated by (early) adverse or traumatic experiences (Raine, 2002b; Susman, 2006; van Goozen et al., 2007), which can lead to symptoms of posttraumatic stress disorder (PTSD) (Brosky & Lally, 2004). Therefore, the present study investigated the longitudinal relationships between ANS (re)activity and proactive and reactive aggression in delinquent male adolescents, and incorporated the moderating effect of PTSD symptoms.

Increasing evidence suggests that general measures of disruptive behavior in juveniles are associated with decreased activity of stress-related neurobiological systems, such as the autonomic nervous system (ANS, represented by heart rate and heart rate variability (HRV), Beauchaine, 2001; Ortiz & Raine, 2004; Raine, 2002a). Findings from a small number of studies on specific types of behavior such as proactive and reactive aggression show a more differentiated view (De Vries-Bouw et al., 2012; Murray-Close & Rellini, 2011; Scarpa et al., 2009). Proactive aggression has been characterized as goal-oriented, planned and unprovoked. Proactive responses are fueled by reward contingencies with the aim to achieve a goal such as possessions or the domination of others (i.e., cold-blooded or instrumental, Dodge et al., 1997; Vitaro et al., 2006). It is thus expected that proactive aggression is related to parameters that reflect low arousal (Raine, 1993; Raine, 2002a; van Goozen et al., 2007; Zuckerman, 1979), such as attenuated heart rate (re)activity. In light of the polyvagal theory (Beauchaine, 2001; Porges, 1995; Porges, 2007), proactive aggression is expected to relate to high resting HRV and decreased HRV reactivity. Previous studies indeed related proactive aggression to attenuated heart rate responsivity (De Vries-Bouw et al., 2012; Murray-Close & Rellini, 2011) and also high resting HRV and attenuated HRV responsivity (Murray-Close & Rellini, 2011; Scarpa et al., 2009). In contrast, reactive aggression is a rather immediate and impulsive response to a source of provocation or threat, and is usually accompanied by the expression of anger (i.e., hot-blooded

or emotional, Dodge et al., 1997; Vitaro et al., 2006). This type of aggression is thus expected to relate to parameters that reflect emotional dysregulation, like low resting HRV and increased HRV reactivity (Beauchaine, 2001; Porges, 1995; Porges, 2007). In light of the theories on arousal, reactive aggression is expected to relate to high arousal, as reflected by increased heart rate (re)activity. Previous studies showed that reactive aggression was related to decreased resting HRV (De Vries-Bouw et al., 2012; Scarpa et al., 2009). Because only a small amount of studies so far focused on the distinct neurobiological correlates of proactive and reactive aggression specifically, and most of the studies were cross-sectional in nature, it is important to replicate and extend these findings in longitudinal designs.

Longitudinal studies on the development of ANS parameters have shown moderate to strong correlations between consecutive assessments (El-Sheikh, 2005; Matthews et al., 1990; Matthews et al., 2002), although findings on stability of ANS reactivity to stress are mixed and appear to depend on the type of stressor used (El-Sheikh, 2005; Kudielka et al., 2004a; Matthews et al., 1990). Aggression shows moderate stability during adolescence (Barker et al., 2006; McAuliffe et al., 2006; Tuvblad et al., 2009), whereas decreases in aggression have been described during the transition into adulthood (Monahan et al., 2009; Xie et al., 2011). However, because stability has mainly been studied in general population samples, it remains unclear to what extent ANS parameters and aggression are stable in delinquent samples. Furthermore, to date only few studies have investigated the relationship between ANS parameters and disruptive or aggressive behavior in longitudinal designs. Most of these studies focused on the predictive value of ANS parameters for future and persistent disruptive behavior (Baker et al., 2009; De Vries-Bouw et al., 2011; Raine et al., 1990; Raine et al., 1995). One single study examined repeatedly measured HRV in relation to the development of externalizing behavior in a general population sample at ages 8, 9, 10, and 11 (El-Sheikh & Hinnant, 2011). It was shown that in boys, low initial resting HRV activity, but not development of resting HRV over time, was related to increasing externalizing symptoms over time. The absence of a relationship with development of HRV over time was explained by the developmental time frame assessed in the study not being wide enough (El-Sheikh & Hinnant, 2011). These results can be extended by incorporating samples with broader age ranges in other developmental stages and by studying specific types of behavior such as proactive and reactive aggression.

It has been proposed in theoretical models that the etiology and development of disruptive behavior are related to complex interplays between neurobiological systems like the ANS and factors like (early) adverse or traumatic experiences (Raine, 2002b; Susman, 2006; van Goozen et al., 2007). These relationships were investigated

in a number of studies, it was found that disruptive / aggressive behavior was only related to ANS (re)activity in the presence of a history of traumatic or adverse events (El-Sheikh et al., 2011; Gordis et al., 2010; Murray-Close & Rellini, 2011). However, only part of the juveniles who experience adverse or traumatic events develop symptoms of posttraumatic stress disorder (PTSD) (Brosky & Lally, 2004). There are indications that the relation between experienced events and aggression is mediated by PTSD symptoms (Marsee, 2008; Ruchkin et al., 2007). It is, therefore, particularly important to study the PTSD symptoms in specific rather than traumatic events, when investigating the relationship between ANS parameters and aggression. It was shown that PTSD symptoms are related to high resting heart rate, low resting HRV and to increased heart rate and HRV responsivity to trauma scripts (Blechert et al., 2007; Kirsch et al., 2011; Scheeringa et al., 2004). This increased ANS reactivity appears to be specific for PTSD as compared to traumatized controls without PTSD (Lindauer et al., 2006). Additionally, PTSD symptoms have been related to higher levels of aggression, with reactive aggression in particular (Kivisto et al., 2009). However, a single study on adult women with and without a history of sexual abuse found no moderating effect of PTSD symptoms on the relation between ANS reactivity and relational aggression (Murray-Close & Rellini, 2011). Because these findings cannot be generalized to other samples, further studies are needed, particularly in antisocial or delinquent samples, since in these samples high rates of PTSD symptoms were found (Colins et al., 2010; Marsee, 2008; Ruchkin et al., 2002; Vermeiren et al., 2006).

For reasons mentioned above, the aim of the present study is threefold. The first aim is to examine to what extent ANS parameters, at baseline and in response to stress, and proactive and reactive aggression are stable over five years in delinquent adolescents. The second aim is to examine longitudinal relationships between ANS parameters and proactive and reactive aggression over the same period during adolescence. The third aim is to examine whether these longitudinal associations are moderated by PTSD symptoms.

METHODS

Participants and procedures

Our study was designed as a prospective longitudinal study on male adolescents who were assessed twice across a period of five years in adolescence. The baseline assessment was conducted in 2002 – 2004, the follow-up assessment in 2006 – 2009.

At baseline, 112 participants were included (mean age 13.7 years, SD 0.7) in

the area of Amsterdam, The Netherlands. Participants were included after they had committed a minor offense, for which they were referred to a delinquency diversion program by the police. Participants underwent behavioral, psychosocial and biological assessment. A subsample of 68 participants (60.7%) was approached to perform a psychosocial stress task during which ANS parameters were recorded. Of the total sample, 85 participants (75.9%) participated at follow-up, which included the same assessment protocol. Re-assessment was refused by 20.5% of the participants ($n = 23$), 1.8% did not live in the Netherlands at the time of approach ($n = 2$) and 1.8% was untraceable ($n = 2$). There were no significant differences between participants and non-participants in age, neurobiological and behavioral parameters at baseline. The entire follow-up assessment including ANS recording was completed by 59 participants (69.4%). The mean IQ in the sample was 93.3, SD 11.9. Forty-nine percent had a low SES, 25.9% a middle SES and 25.0% a high SES. Thirty-eight percent was of Caucasian ethnicity, 26.8% of Surinam / Antillean, 26.8% of Mediterranean and 8.9% of other ethnicity. The study was approved by the Medical Ethical Committee of the VU University medical center Amsterdam, and participants and their parents gave written informed consent for both baseline and follow-up assessment.

Behavioral assessment

To obtain detailed information on specific types of aggression, participants filled out the Reactive-Proactive Aggression Questionnaire (RPQ, Raine et al., 2006). This 23-item self-report questionnaire contains 11 items on reactive aggression and 12 items on proactive aggression. Each item was rated as 0 (never), 1 (sometimes), or 2 (often) for frequency of occurrence. The RPQ has shown good reliability and validity (Raine et al., 2006).

Psychosocial stress task procedure

The participants performed a psychosocial stress test procedure in the laboratory, consisting of a public speaking task (PST) in front of a one-way screen with video recording (Jansen et al., 2000), which is an effective stressor in both children and adults (Dickerson & Kemeny, 2004). The procedure is described in detail elsewhere (Popma et al., 2006). In summary, there was a 50 minute resting period prior to the PST. After the resting period, an unfamiliar test assistant explained the PST itself, which consisted of a 5 minute speech on a topic of choice preceded by 10 minutes of preparation. It was suggested that a 'jury' of three psychologists was behind a one-way screen, judging the participants' performance. This judgment was always positive, thereby ending the stressful situation.

Procedure for recording of autonomic measures

Heart rate and heart rate variability (HRV) were measured continuously during the stress task procedure as an index of autonomic / parasympathetic activity, using the VU-Ambulatory Monitoring System (AMS, Klaver et al., 1994). Three disposable Ag/AgCl electrodes filled with conducting paste were placed on the chest; they were connected with lead wires to the AMS device. To detect R tops in the electrocardiogram (ECG), one active electrode was placed at the jugular notch of the sternum and the other on the left breast, 1.5 in. below the nipple, between two ribs. The third (ground) electrode was placed at the right chest between the lower two ribs. The ECG signal was led into a differential amplifier, with an input impedance higher than 1 M Ω and a common-mode rejection ratio of 96 dB. The amplified ECG was passed through a bandpass filter of 17 Hz. The R top was recognized with a level detector with automatic level adjustment. The output of the level detector was connected to an interrupt request line of the microprocessor in the device. At each R peak, a millisecond counter was read and reset, yielding the raw interbeat interval (IBI). The R-R time accuracy was 1 ms; the sample rate was set at 1 kHz. From the ECG we obtained the IBI time series. For the analysis of HRV, we performed spectral analyses using Kubios HRV software, developed by the Biosignal Analysis and Medical Imaging Group, University of Kuopio, Finland. The IBI time series were decomposed into component heart rate variability frequencies by using Fourier transformations. The resulting components are expressed in terms of a spectral density function, or the amount of spectral power within a given frequency band. For the purpose of this study, we used high-frequency heart rate variability (0.15 – 0.40 Hz), which provides a frequency-domain index of parasympathetic activity (Berntson et al., 1997).

The mean heart rate / HRV during the second half of the initial resting period (after participants had adjusted to the setting) was used as basal value. To compute a measure of responsivity to stress, we subtracted the basal value of the mean value during the first minute of the speech. Since heart rate generally increases in response to stress, a positive value for heart rate reactivity reflects an increase in physiological arousal to stress. In contrast, because HRV generally decreases in response to stress (vagal withdrawal), a positive value for HRV reactivity reflects relatively attenuated HRV reactivity (decreased vagal withdrawal), whereas a negative value for HRV reactivity reflects increased HRV reactivity.

Assessment of posttraumatic stress symptoms, and experienced traumatic events

The presence of posttraumatic stress symptoms was assessed using the Child Post Traumatic Stress Disorder – Reactivity Index (CPTSD-RI, Frederick et al., 1992). This is

a 20-item self-report questionnaire, reporting on the past 30 days. Items were scored on a five-point likert scale, ranging from 1 (none) to 5 (most of the time). A total score was based on the sum of all items. A total score lower than 12 indicates no PTSD, 12-24 mild PTSD, 25-39 moderate PTSD, 40-59 severe PTSD, and 60 or higher indicates very severe PTSD. The CPTSD-RI has shown good reliability and validity (Pynoos et al., 1987; Pynoos et al., 1993).

To obtain information on the history of experienced traumatic events and the presence of a PTSD diagnosis, the National Institute of Mental Health (NIMH) Diagnostic Interview Schedule for Children (DISC), version IV (Shaffer et al., 2000) was used, which is an extensive structured psychiatric interview. The section on posttraumatic stress disorder (PTSD) was administered from both participants and their parents by trained interviewers. The first eight questions concern traumatic events that a participant may ever have experienced. Participants were scored as having experienced an event, when an event was scored in either of the separate interviews.

Statistical analyses

Analyses were performed using SPSS version 19.0. We considered p -values $< .05$ statistically significant. HRV values were positively skewed. Because a log transformation resulted in negatively skewed values, we applied a square root transformation, after which HRV values were normally distributed. Square root transformations were also applied to proactive aggression at both assessments.

Stability of ANS and aggression parameters was investigated for the total group, as well as within individuals. Group stability was studied by examining changes in mean levels of a parameter over time, by means of paired samples T-tests. Individual stability was studied by computing Pearson's correlations between the measurements at baseline and follow-up, to indicate the maintenance of a participants' relative position within the studied population over time (also referred to as tracking, Twisk, 2003)

The longitudinal relationships between ANS parameters and proactive and reactive aggression were studied by means of generalized estimating equations (GEE). In this method, data of a participant at a particular time point is entered in the analysis when all dependent and independent variables are available at that time point. Consequently, the number of time points can differ between participants. GEE accounts for dependence of repeated measures within one participant by using a working correlation structure. An exchangeable correlation structure was used for all analyses. In separate linear models, proactive or reactive aggression were used as dependents variables, and each separate ANS parameters was used as independent

variable. Also applying GEE analyses, PTSD symptoms were studied in relation to proactive or reactive aggression, and to each of the ANS parameters. In each of these analyses, an aggression or ANS parameter was entered as dependent variable, and PTSD symptoms as independent variable.

Next, the moderating effect of PTSD symptoms was examined. For that purpose, GEE analyses were applied with proactive or reactive aggression as dependent variables, and each separate ANS parameter, PTSD symptoms and their interaction as independent variables. In all GEE analyses, we used standardized variables. The interaction variables were computed as the product of the standardized variables and were not standardized themselves.

RESULTS

Stability of study variables over time

Table 1 presents descriptive statistics at baseline and follow-up as well as stability over time for proactive and reactive aggression, ANS parameters and PTSD symptoms. They reflect original scores before transformation and standardization.

The mean levels of proactive and reactive aggression were stable from baseline to follow-up assessment, as analyzed with paired samples T-tests. Both types of aggression showed significant although weak correlations between baseline and follow-up assessment, indicating stability within individuals.

The mean resting heart rate showed a significant decline of 7 bpm over time, whereas the mean heart rate reactivity significantly increased circa 14 bpm. Both parameters showed significant moderate correlations between baseline and follow-up assessments. Mean levels of resting HRV and HRV reactivity were stable over time. Resting HRV showed a significant moderate correlation between baseline and follow-up, whereas HRV reactivity showed no correlation.

The mean level of PTSD symptoms was stable over time, whereas PTSD symptoms at baseline and follow-up showed no correlation. This indicates that, although for the total group the mean level of PTSD symptoms did not change, within the group the individual participants showed no stability in PTSD symptom level. None of the participants scored a formal PTSD diagnosis. With respect to the experienced traumatic events, at baseline assessment, 75.2% reported one or more events, with a mean number of 1.6, SD 1.4, range 0 – 7. At follow-up, 76.5% reported one or more events, with an increase in the mean number to 2.4, SD 1.9, range 0 – 7.

Table 1. Descriptive statistics, correlations and paired t-tests of aggression, autonomic parameters and posttraumatic stress symptoms at baseline and follow-up

	Baseline (T0)			Follow-up (T1)			Correlation T0 – T1			Paired T-test	
	N	Mean (SD)	Range	N	Mean (SD)	Range	N	R	p	T	p
Proactive aggression	60	4.6 (4.0)	0 – 16	68	4.2 (3.4)	0 – 14	35	.346	.042	0.726	.473
Reactive aggression	60	9.7 (4.4)	1 – 21	68	9.3 (4.3)	0 – 18	34	.348	.044	-0.732	.469
HR resting	68	78.4 (9.6)	60.6 – 107.0	53	71.4 (10.6)	51.0 – 95.0	31	.640	.000	4.62	.000
HR reactivity	67	9.6 (10.6)	-11.1 – 37.5	53	24.0 (12.2)	3.9 – 47.9	31	.596	.000	-8.12	.000
HRV resting	66	1428 (1173)	75 – 4636	53	1358 (1632)	12 – 7013	31	.450	.011	-0.04	.972
HRV reactivity	66	-359 (1047)	-3197 – 1638	49	-541 (957)	-3527 – 1355	29	.121	.530	1.39	.177
PTSD symptoms	84	15.0 (8.1)	0 – 34	71	15.0 (9.1)	1 – 40	53	-.056	.691	0.21	.834

HR: heart rate; HRV: heart rate variability; PTSD symptoms: posttraumatic stress symptoms

Longitudinal relationships between ANS parameters and proactive and reactive aggression

Applying GEE analyses, the longitudinal relationships between ANS parameters and proactive and reactive aggression were examined (see Table 2). There was a significant longitudinal relationship between attenuated HRV reactivity (smaller decrease of HRV in response to stress) in relation to proactive aggression. There was a trend toward a longitudinal relationship between low resting HRV and proactive aggression. There were no longitudinal relationships between heart rate (re)activity and proactive and reactive aggression, and between HRV (re)activity and reactive aggression.

Table 2. Longitudinal association between ANS parameters and proactive – reactive aggression

	N	Proactive aggression		Reactive aggression	
		Beta	p	Beta	p
HR resting	78	.085	.247	-.013	.854
HR reactivity	78	-.128	.152	-.067	.491
HRV resting	76	-.162	.089 [†]	.065	.532
HRV reactivity	74	.201	.029 [*]	.160	.122

Note. HR: heart rate; HRV: heart rate variability. Because HRV generally decreases in response to stress, an association for HRV reactivity in the positive direction indicates attenuated HRV reactivity (decreased vagal withdrawal) in relation to aggression.

[†] p < .10

^{*} p < .05

PTSD symptoms in relation to ANS parameters and aggression

Applying GEE analyses, PTSD symptoms were found to significantly relate to reactive aggression (Beta = .251; p = .003), not to proactive aggression (Beta = .156; p = .110). Furthermore, PTSD symptoms were related to an attenuated HRV reactivity (less decrease in response to stress, Beta = .214; p = .022), but not to resting HRV (Beta = .005; p = .962), resting heart rate (Beta = -.133; p = .124) or heart rate reactivity (Beta = -.015; p = .890).

Interactions between PTSD symptoms and ANS parameters in relation to proactive and reactive aggression

To assess the moderating effect of PTSD symptoms on associations between ANS parameters and proactive and reactive aggression, GEE analyses were applied. There was a trend toward a significant interaction between HRV reactivity and PTSD symptoms in relation to proactive aggression (Beta = -.170, p = .083). A graphic representation of this interaction is presented in Figure 1. Single slope analyses reveal that attenuated



HRV reactivity is significantly related to higher levels of proactive aggression, when PTSD symptoms are low. When PTSD levels are high, this relationship diminishes.

Although there were trends toward interactions between PTSD symptoms and heart rate reactivity in relation to proactive aggression and between PTSD symptoms (Beta = $-.131$, $p = .098$) and resting HRV in relation to reactive aggression (Beta = $-.135$, $p = .099$), follow-up simple slope analyses were not significant. No other interactions were found.

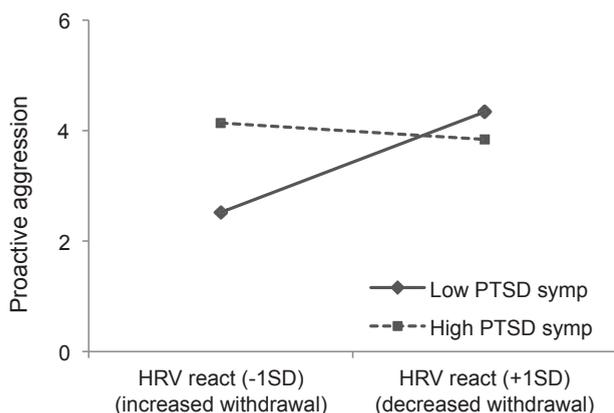


Figure 1. Interaction between PTSD symptoms and HRV reactivity in relation to proactive aggression. The relationship between HRV reactivity and proactive aggression is presented by two single slopes which represent high (+1 SD) and low (-1 SD) values of PTSD symptoms. Because HRV generally decreases in response to stress, an association for HRV reactivity in the positive direction indicates attenuated HRV reactivity (decreased vagal withdrawal) in relation to proactive aggression.

DISCUSSION

The first aim of the present study was to investigate stability of ANS parameters and proactive and reactive aggression over five years in delinquent male adolescents. Second, we aimed to study longitudinal relationships between ANS parameters and proactive and reactive aggression. Finally, we investigated the moderating effect of PTSD symptoms on these relationships.

Regarding the first aim of our study, we found significant although weak stability of proactive and reactive aggression in our sample of delinquent male adolescents, which is in line with previous results (McAuliffe et al., 2006; Tuvblad et al., 2009). As expected in such a sample, the mean levels of aggression were higher than previously found in an adolescent general population sample (Raine et al., 2006). With respect to stability of ANS parameters, the observed decline in mean resting heart rate and

increase in mean heart rate reactivity over time were consistent with findings in the general population (Iliff & Lee, 1952; Matthews et al., 1990; Matthews et al., 2002). At individual level, both heart rate parameters as well as resting HRV showed considerable stability between early and late adolescence, referring to the maintenance of a participants' relative position within the studied population over time. Taken together with findings from the literature on relationships between these ANS parameters and disruptive behavior, resting heart rate / HRV and heart rate reactivity can be regarded as stable risk-factors for disruptive behavior. In contrast, HRV reactivity showed no stability. To our knowledge, ours is the first study that examined stability of HRV in response to a public speaking task with a relatively long follow-up period. To determine whether lack of stability is typical for a sample of delinquent adolescents, these results need to be replicated in other samples, including from the general population.

With respect to the second aim of our study, we found a longitudinal relationship between attenuated HRV reactivity (decreased vagal withdrawal) and proactive aggression. To our knowledge, our current study is the first to longitudinally investigate HRV reactivity in relation to proactive (and reactive) aggression. Previous studies on HRV reactivity and disruptive behavior showed conflicting and often negative results (Beauchaine et al., 2001; Beauchaine et al., 2008; De Vries-Bouw et al., 2011; Dietrich et al., 2007; El-Sheikh et al., 2011; Mezzacappa et al., 1997). Several methodological issues may underlie these inconsistencies, such as the different, often heterogeneous, measures of disruptive behavior that were studied. We studied specific types of disruptive behavior, i.e. proactive and reactive aggression, because of the assumed differential neurobiological profiles underlying these subtypes of aggression (De Vries-Bouw et al., 2012; Murray-Close & Rellini, 2011; Scarpa et al., 2009). Our result that attenuated HRV reactivity is related to proactive but not reactive aggression indeed provides support for this idea.

The observed association between attenuated HRV reactivity and proactive aggression fits in with the polyvagal theory (Porges, 1995; Porges, 2007). This theory postulates that deployment of the vagus nerve in response to stress (as reflected by low vagal withdrawal or even vagal augmentation), inhibits the acceleratory SNS input to the heart and thereby suppresses the robust emotional reactions that characterize fight or flight responding. This efficient regulation of emotion facilitates social engagement. In contrast, when vagal withdrawal is increased, response shifts to the fight/flight response that is accompanied by rage and panic. Such emotional dysregulation is regarded as hallmark for psychopathology, including internalizing as well as externalizing problems (Beauchaine, 2001). Proactive aggression is characterized as goal-oriented, planned and unprovoked (Dodge et al., 1997; Vitaro et al., 2006) and

is thus expected to require an adequate level of emotion regulation, represented by low vagal withdrawal. Our results confirm this by showing a relationship between decreased vagal withdrawal and proactive aggression.

Regarding our third aim, the moderating role of PTSD symptoms, we found that PTSD symptoms were related to higher levels of reactive, but not proactive aggression, which is in line with previous findings (Kivisto et al., 2009). Furthermore, we found a moderating effect of PTSD symptoms on the association between HRV reactivity and proactive aggression, this moderating effect showed a trend toward statistical significance. The association between HRV reactivity and aggression was present at low levels of PTSD symptoms, whereas at high levels of PTSD symptoms the association diminished. Previous studies on this topic mostly focused on early adversities (El-Sheikh et al., 2011; Gordis et al., 2010; Murray-Close & Rellini, 2011). It has been argued in theoretical models that stress-related neurobiological systems like the ANS act as a mediator in the relation between (early) adverse or traumatic events and disruptive behavior (Raine, 2002b; van Goozen et al., 2007). Early adversities are proposed to play a role in the attenuation of such physiological systems, which in turn is considered an important mechanism involved in the development and persistence of disruptive behavior (Susman, 2006; van Goozen et al., 2007). We extended previous studies by studying PTSD symptoms, which can be the consequence of adverse or traumatic events. Since PTSD symptoms are expected to increase HRV reactivity, high levels of PTSD symptoms are expected to diminish the association between HRV reactivity and proactive aggression. We found, rather surprisingly, that PTSD symptoms were related to attenuated HRV reactivity, yet the moderating effect of PTSD symptoms on the relationship between HRV reactivity and proactive aggression was in the expected direction. However, our results are not consistent with a previous study on adult women in which no moderating effect of PTSD symptoms on the association between HRV and proactive aggression was found (Murray-Close & Rellini, 2011). This inconsistency between ours and their results may be explained by the difference in sample. Previous studies showed marked differences between boys and girls in the association between HRV reactivity and aggressive or delinquent behavior; most of the associations were found in boys, yet not in girls (Beauchaine et al., 2008; El-Sheikh et al., 2011; El-Sheikh & Hinnant, 2011). Our findings underline the importance of incorporating factors like trauma-related variables when studying relationships between ANS parameters and aggression. Future studies in other samples are needed to further explore the moderating role of PTSD symptoms in the relationship between ANS or other stress-regulated systems and disruptive behavior.

There are some limitations of the study that need to be considered when interpreting the results. First, our longitudinal assessment was limited to two

measurements across adolescence. Although our study provides important information regarding the stability of ANS and aggression parameters and their coherence, we still cannot make any statements regarding causality. We do not know whether a change in ANS causes a change in aggression, whether it is the other way around, or whether it is a reciprocal relation. Future studies on the development of neurobiological and behavioral parameters should include more assessments, for example annually across adolescence, allowing for more advanced statistical techniques such as time lag models, to provide more insight on causality. Furthermore, experimental procedures could be incorporated. It can be studied whether induced changes in ANS parameters, for example by the use of stimulants, lead to a change in aggression.

Second, we studied a relatively small and specific sample of delinquent male adolescents. The small sample size prohibited us to study subgroups within our population. Studying subgroups is relevant, for example to compare stability of neurobiological reactivity between adolescents who show persistent high levels of aggression and those with persistent low or variable levels of aggression. This can provide additional information on whether the relationship between changes in ANS parameters (HRV reactivity) and changes in (proactive) aggression is specific for certain subgroups. This may have relevance for clinical purposes.

Third, our information on experienced traumatic events was limited. Information on the moment when traumatic events had occurred and on the duration of the PTSD symptoms was lacking. Nevertheless, we do have indications that a large part of the participants had experienced one or more different traumatic events, yet none of the participants scored a formal PTSD diagnosis. The range of PTSD symptoms thus was limited, this may have affected the results. Furthermore, although we used a validated instrument to assess PTSD symptoms, this questionnaire provided only information on the level of symptoms in the month prior to the assessment. It has been shown that acute PTSD symptoms are related to increased arousal, whereas chronic PTSD, or prolonged experienced traumatic events, can lead to attenuated arousal (Murali & Chen, 2005; Susman, 2006). It is, therefore, relevant to incorporate detailed information on the moment and the duration of the traumatic event(s) that occurred, as well as the duration of PTSD symptoms.

Despite the limitations, the results of our longitudinal study add to the advancing knowledge on distinctive neurobiological profiles underlying subtypes of aggression, and to insight in the role of associated factors like PTSD symptoms. There was considerable stability within individuals of aggression and ANS parameters, except HRV reactivity. Moreover, we found a relationship between attenuated HRV reactivity, reflecting adequate emotional regulation, and proactive aggression, characterized

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as instrumental and cold-blooded. We found indications that the association between HRV reactivity and proactive aggression was only present at low levels of PTSD symptoms. Consequently, future studies are recommended to incorporate this moderating factor.

