

The predictive value of low heart rate and heart rate variability during stress for reoffending in delinquent male adolescents

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Abstract

Low autonomic (re)activity is a consistent correlate of antisocial behavior in juveniles. However, longitudinal research relating autonomic measures to persistent antisocial behavior has remained scarce. Therefore, in the present study we examined the predictive value of heart rate (HR) and heart rate variability (HRV, often studied as respiratory sinus arrhythmia) for reoffending in delinquent male adolescents. At initial assessment, HR and HRV were measured at rest and in response to a public speaking task. Registered reoffending was assessed after 5-year follow-up. Attenuated HR response and stronger HRV response to stress predicted higher reoffending rates. Results provide evidence that HR/HRV reactivity are neurobiological markers for persistent juvenile antisocial behavior. Although effect sizes were small to moderate, our findings underscore the consistency of the relationship between autonomic markers and antisocial behavior.

Descriptors: Heart rate, Behavioral medicine, Children/infants

Because juveniles who display antisocial behavior are at risk for a series of negative outcomes later in life (Maughan & Rutter, 2001), an extensive body of research has focused on factors predicting future deviancy. Over the past decades, interest in neurobiological markers has increased substantially, for example, by examining the main human stress-regulating systems such as the autonomic nervous system (ANS). One dominant paradigm of stress regulation in relation to antisocial behavior is the *low arousal theory*. In this theory, it is argued that individuals seek out stimulation, for example, by displaying antisocial behavior, to optimize their low arousal, which represents an unpleasant physiological state (sensation seeking theory; Zuckerman, 1979). According to this theory, it is expected that the ANS functions at a low basal level. An alternative theory postulates that antisocial individuals do not fear the negative consequences of their actions and therefore are more likely to engage in antisocial behaviors (fearlessness theory; Raine, 1993). From both perspectives it is thus expected that basal ANS activity and ANS responsivity to

psychosocial stress would be attenuated in antisocial juveniles and may put them at risk for further antisocial behavior.

ANS (re)activity can be measured using heart rate. Although a substantial body of cross-sectional studies on associations between heart rate and antisocial behavior exists, longitudinal studies on the subject are scarce. In line with theory, cross-sectional studies have shown low resting heart rate to be a consistent biological correlate of antisocial behavior in juveniles as shown in a meta-analysis by Ortiz and Raine (2004). Resting heart rate is studied rather extensively; however, a much smaller number of studies incorporated heart rate during a stressor. Nevertheless, in the mentioned meta-analysis, a strong effect size was revealed for low heart rate during a stressor in relation to antisocial behavior (Ortiz & Raine, 2004). Recent studies confirmed this by showing attenuated heart rate responses during a stressor in disruptive behavior disordered juveniles compared to normal controls (Fairchild et al., 2008; Popma et al., 2006). To date, longitudinal research on the subject has focused on resting heart rate, mainly in general population samples. In a male adolescent sample, low resting heart rate at age 15 was related to criminal status at age 24 (Raine, Venables, & Williams, 1990) as well as to persistent delinquent behavior in an antisocial subgroup from this population (Raine, Venables, & Williams, 1995). Another study demonstrated that low resting heart rate in a population sample of 3-year-olds predicted future aggressive behavior at age 11 (Raine, Venables, & Mednick, 1997). However, some studies did not

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report resting heart rate to predict antisocial behavior prospectively (Baker et al., 2009; van Bokhoven, Matthys, van Goozen, & van Engeland, 2005). These negative results may be the consequence of the use of a composite measure of antisocial behavior as an outcome measure (Baker et al., 2009) or specificity of the studied population, that is, disruptive behavior disorder children (van Bokhoven et al., 2005). Up till now, only heart rate at rest, but not during stress, has been used as a predictor of antisocial outcome.

Heart rate is an indicator of activity of both sympathetic and parasympathetic nervous systems (respectively, SNS and PNS), which have generally opposing actions on organ systems. The SNS engages the organism for fight or flight and involves increased heart rate and respiration. The PNS corresponds to slower heart rate and respiration and lower overall emotional arousal (Berntson, Cacioppo, & Quigley, 1991). The specific role of the PNS on cardiac activity can be illuminated by measuring respiratory sinus arrhythmia, or high frequency heart rate variability (further mentioned as heart rate variability; Berntson, Cacioppo, & Quigley, 1993; Grossman & Taylor, 2007). Heart rate variability is the variation in the interval between consecutive heart beats in the respiration frequency range due to the influence of the vagus nerve on the sinoatrial node (Beauchaine, 2001). The *polyvagal theory* provides a perspective relating autonomic function and, in specific, parasympathetic function to (antisocial) behavior (Porges, 1995, 2007). This theory specifies two distinct branches of the vagus nerve influencing the heart. The first branch, originating from the dorsal motor nucleus, functions to suppress metabolic demands under conditions of danger. The second, phylogenetically newer branch originates from the nucleus ambiguus. Deployment of this branch suppresses the robust emotional reactions that characterize fight or flight responding by inhibiting acceleratory SNS input to the heart. When such vagally mediated social affiliative behaviors are ineffective in coping with a stimulus, response shifts to the fight or flight response by withdrawal of the inhibitory vagal influence. Functional deficiencies of this branch might place individuals at risk for emotional lability, a hallmark for psychopathology (Beauchaine, 2001).

Heart rate variability appears to be a sensitive index of vagal pathways originating in the nucleus ambiguus (Porges, 2007). With respect to the relationship between heart rate variability and antisocial behavior, several studies showed reduced baseline PNS activity in antisocial juveniles (Beauchaine, Hong, & Marsh, 2008; Beauchaine, Katkin, Strassbert, & Snarr, 2001; Gordis, Feres, Oleski, Rabkin, & Trickett, 2010; Mezzacappa et al., 1997; Pine et al., 1998), although opposite results were found in an early adolescent population sample (Dietrich et al., 2007). With respect to response to physical and psychological stress, no relationships were found between PNS reactivity and antisocial behavior, whereas decreases in PNS activity were reported in both population and antisocial samples (Beauchaine et al., 2001, 2008; Dietrich et al., 2007; Mezzacappa et al., 1997). However, longitudinal studies relating heart rate variability (re)activity to persistent or future antisocial behavior are lacking.

When one studies the predictive value of ANS (re)activity for persistence of delinquent behavior, it is substantially relevant to study delinquent adolescents in particular. It is well known that crime rates peak during the adolescent years, and most delinquents experience their first arrest in adolescence (Agnew, 2003). Improving outcome prediction at an early stage can stimulate the development of effective prevention and intervention strategies,

which carries advantages for society as well as the delinquent juveniles themselves. Therefore, the aim of the current study is to examine the predictive value of heart rate and heart rate variability, in resting and stressful conditions, for reoffending during a 5-year follow-up period in a sample of delinquent male adolescents.

Methods

Participants

Participants were 68 male adolescents (mean age = 13.9 years, $SD = 0.8$) attending a delinquency diversion program after having committed a minor offense. The sample was derived from an original sample of 112 delinquent male adolescents who initially participated between 2002 and 2004 in a study on neurobiological factors of antisocial behavior (Popma et al., 2006). The subsample for this article consisted of the 68 participants from the original sample who performed a public speaking task to assess ANS reactivity to stress. Participants in this subsample were significantly older than nonparticipants (mean age of participants = 13.9 years, $SD = 0.8$; mean age of nonparticipants = 13.4 years, $SD = 0.6$; $t = -3.10$, $p = .002$). There were no differences between groups in reoffending rate, number of reoffences, IQ, ethnicity or socioeconomic status (SES; all $p > .50$). Participants were included after having attended a delinquency diversion program in the area of Amsterdam, The Netherlands, after having committed a minor offense such as shoplifting, property damage, or minor forms of aggression. Exclusion criteria for participation were a history of any neurological or endocrinological disorders and use of steroid medication. For more detailed information, see Popma et al. (2006). The study was approved by the Medical Ethics Committee of the VU University Medical Center Amsterdam, and parents as well as subjects gave written informed consent.

The mean IQ in the sample was 93.9, $SD = 12.4$. Forty-seven percent had a low SES, 28% a middle SES, and 25% a high SES. Thirty-four percent were Caucasian, 29% Surinam/Antillean, 27% Mediterranean, and 10% of other ethnicity.

Reoffending Data

A follow-up on police registrations of reoffending was conducted for all participants in 2009 (mean follow-up time = 5.5 years, $SD = 0.8$). Data on reoffending were obtained from the official Dutch police registration system for criminal behavior (*Herkenningssdiensysteem*, HKS) by means of a computerized search. In this national system, the police register the type of offense a person is suspected of as well as personal data. The minimum age for being registered is 12 years, which is the lowest age of criminal responsibility in The Netherlands (Eggers & Kalidien, 2008).

Time at risk is the period between initial assessment and follow-up, taking into account periods of incarceration, in which participants are assumed to be unavailable to commit offenses. To determine the time at risk, incarceration time during follow-up was subtracted from total follow-up time. To determine incarceration time, the national registry of all persons who are imprisoned was used (named TULP). The mean stay in closed settings during the follow-up period was 60 days ($SD = 164$ days, range: 0 days–2.9 years).

Physiological Assessment

At initial assessment, heart rate and heart rate variability were measured in resting conditions and in response to a standardized psychosocial stress task. This test consisted of a public speaking task (PST) in front of a one-way screen with video recording (Jansen et al., 1999). The PST was embedded in a 2-hour test session. There was a 50-min resting period prior to the PST, which participants spent as relaxed as possible and filling in questionnaires. After the resting period, an unfamiliar test assistant explained the PST itself, which consisted of a 5-min speech on a topic of choice preceded by 10 min 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. The PST was followed by another resting period of 60 min. For a detailed description of the PST, see Popma et al. (2006). This task embodies all the major criteria of an anxiety-arousing situation, for it is perceived as a threat, only partially under participant control, and implies uncertainty with regard to the outcome and consequences (Dickerson & Kemeny, 2004). In healthy participants, similar psychosocial stress tests elicited an increase in heart rate and a decrease in heart rate variability during the task (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Strahler, Mueller, Rosenlocher, Kirschbaum, & Rohleder, 2010).

Heart rate and heart rate variability were continuously measured during the entire test session as an index of autonomic activity, using the VU-Ambulatory Monitoring System (AMS; Klaver, de Geus, & de Vries, 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 inter-beat 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. We performed spectral analyses using Kubios HRV software, developed by the Biosignal Analysis and Medical Imaging Group, Department of Physics, 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). High-frequency power provides a frequency-domain index of parasympathetic activity (Berntson et al., 1997).

The mean heart rate/heart rate variability during the second half of the initial resting period (after participants had adjusted to the setting) was used as the measure of resting state. The 5-min speech performance was divided into 1-min intervals, because dividing the task into short intervals can provide information on

differences between groups in the course of autonomic measures during stress. The first 1-min interval was used as a measure of heart rate/heart rate variability during stress. We used this specific interval because autonomic responses were most prominent during the first minute, and differences between groups may thus be better identified (Kudielka & Wust, 2010). As a specific measure of heart rate/heart rate variability responsivity to stress with correction for baseline values, we computed the differences between the values at rest and during stress and used these as a measure of responsivity to stress.

Recording of Negative Affect

Participants filled out the Von Zerssen (1986) scale, modified for children, to measure affect changes. The scale was filled out at the following five time points: (a) halfway through the initial resting period, (b) before preparing the PST talk, (c) before the talk, (d) immediately after the talk, and (e) 20 min after finishing the talk. Participants were asked to report their feelings from a list of positive and negative affect labels. Items could be scored as follows: 0 = *positive affect label*, for example, "good" or "calm"; 2 = *negative affect label*, for example, "bad" or "nervous"; or 1 = *none of those*. A total negative affect score per time point was calculated by adding scores of the nine items.

Statistical Analyses

Analyses were performed using SPSS version 17.0 and SAS version 9.2. For all analyses, values of $p < .05$ were considered statistically significant. Heart rate variability values were positively skewed; therefore a log transformation was applied after which all values were normally distributed. Heart rate values were normally distributed. For the survival analyses (which are described later), we used the raw heart rate variability values. Because the upper range of these values was high, we divided all values by 1000 in order to obtain interpretable hazard ratios.

Based on the police registration data, participants were characterized as persisters (one or more reoffenses during follow-up) or desisters (no reoffending). Differences between groups in levels of heart rate and heart rate variability at single time periods were analyzed using Student's t test. To assess differences between groups in heart rate and heart rate variability during the five 1-min intervals of the stress task as well as negative affect before, during, and after the task, repeated measures analyses of variance (ANOVAs) were conducted with group as a between-subjects factor and time as a within-subjects factor. Greenhouse–Geisser corrections were applied when the assumption of sphericity was violated. Uncorrected degrees of freedom and the epsilon were reported. Difference contrasts were performed to further assess main effects of time as well as Group \times Time interactions. For negative affect, a series of post hoc analyses was performed, using Kruskal–Wallis tests to examine differences between groups in individual items of the negative affect scale.

Survival analysis using the Cox proportional hazard regression analysis was used to examine values of heart rate and heart rate variability as predictors for reoffending. First, univariate single event models were performed, each with a different heart rate or heart rate variability measure (resting or stress response) as the predictor. The outcome (survival time) was the time between initial measurement (T0) and an event (defined as the first reoffense during follow-up) or censoring. Subjects who did not

Table 1. Descriptive Statistics for Heart Rate and Heart Rate Variability

Measure	<i>M</i>	<i>SD</i>	Min	Max
Heart rate				
Resting	78.41	9.65	60.64	106.95
During stress	88.04	12.18	56.87	121.34
Reactivity	9.64	10.50	-11.16	37.47
Log(heart rate variability)				
Resting	6.85	0.97	4.32	8.44
During stress	6.55	1.03	4.04	8.17
Reactivity	-0.31	0.91	-2.82	1.98

Note. Heart rate is expressed in beats per minute; log(heart rate variability) is expressed in milliseconds squared.

reoffend during the follow-up period were considered to be censored on the last day of the follow-up. Second, univariate Andersen–Gill proportional means models for recurrent events were fitted to the data (Andersen, Borgan, Gill, & Keiding, 1993). The proportional means model is a generalization of the Cox proportional hazards model, which allows for multiple events per subject.

The proportional hazards assumption was verified by including a time-dependent variable in the model. Another assumption of the statistical methods used is that the total time that a participant is at risk should not depend on his rate of reoffending. As participants were not considered to be at risk during the time they were in prison after reoffending, there may have been a small violation of this assumption. However, sensitivity analyses showed the impact of such a violation on the estimated hazard ratios to be negligible.

Results

Differences in Heart Rate Levels between Persisters and Desisters

At follow-up, 47 participants (69%) had reoffended (persisters), with a median of two reoffenses per participant (range 1–16). Descriptive statistics for heart rate levels are presented in Table 1. A graphic representation of heart rate levels for persisters and desisters in resting conditions and during the stress task (five 1-min intervals) is given in Figure 1a. Heart rates in resting conditions did not differ between the two groups, $t = 1.36, p = .179$, Cohen’s $d = 0.36$. As shown in this figure, the increase of heart

rate during the stress task was limited to the first minute of the task. A repeated measures ANOVA on the five intervals showed a significant effect of time for all time points, $F(4,260) = 25.70, p < .001, \eta^2 = .278, \epsilon = .852$, and a trend toward an effect of group, $F(1,65) = 3.45, p = .068, \eta^2 = .05$, but no Group \times Time interaction, $F(4,260) = 1.69, p = .154, \eta^2 = .018$, showing that the course over time of heart rate during the stress task is similar for both groups. Although the persisters showed a significantly lower heart rate during the first minute of the task compared to the desisters, $t = -2.35, p = .022$, Cohen’s $d = -0.62$, there were no differences in the heart rate response between the two groups, $t = 1.41, p = .163$, Cohen’s $d = 0.38$.

Differences in Heart Rate Variability between Persisters and Desisters

Descriptive statistics for heart rate variability levels are presented in Table 1. A graphic representation of heart rate variability for persisters and desisters in resting conditions and during the stress task is given in Figure 1b. There were no differences between the groups in heart rate variability during resting conditions, $t = -1.52, p = .135$, Cohen’s $d = -0.41$. A repeated measures ANOVA showed a trend toward a main effect of group, $F(1,63) = 3.94, p = .052, \eta^2 = .06$, showing slightly higher heart rate variability during stress for persisters compared to desisters, but no main effect of time, $F(4,252) = 1.91, p = .109, \eta^2 = .03$, or a Group \times Time interaction, $F(4,252) = 0.73, p = .575, \eta^2 = .01$. There was no significant difference between groups in the response of heart rate variability, $t = 0.12, p = .905$, Cohen’s $d = 0.04$.

Self-reported Negative Affect

With respect to negative affect before, during, and after the stress task, a repeated measures ANOVA on five time points revealed a significant effect of time, $F(4,264) = 23.73, p < .001, \eta^2 = .26, \epsilon = .753$, attributable to Time Points 2, 3, and 5. There were no effect of group, $F(1,66) = 0.045, p = .833, \eta^2 = .00$, or a Group \times Time interaction, $F(4,264) = 0.783, p = .505, \eta^2 = .01$. A series of post hoc analyses showed no differences between persisters and desisters on the individual items of the negative affect scale, all $p > .11$.

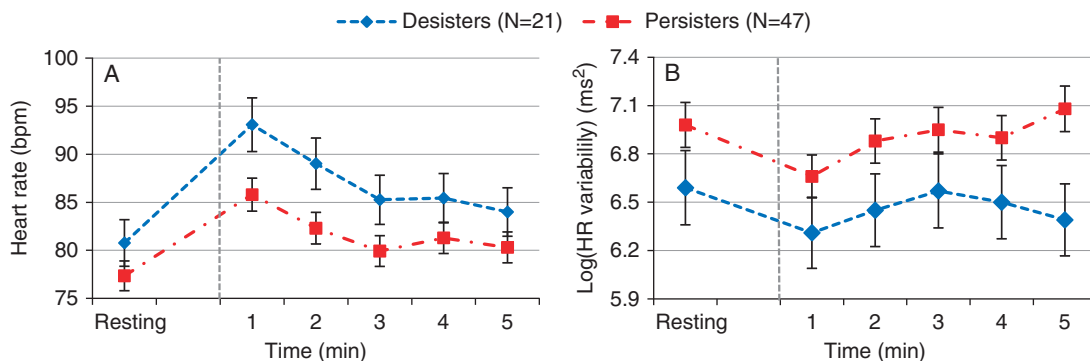


Figure 1. Heart rate (a) and Heart rate variability (b). Differences between persisters and desisters in resting conditions and during the five 1-min intervals during the stress task. Means and SEM are displayed. The vertical line in both figures indicates the start of the 5-min stress task. In the first minute of the task, persisters showed significantly lower heart rate compared to desisters. Furthermore, persisters showed a slightly lower heart rate and a slightly higher heart rate variability during the 5-min task compared to desisters.

Table 2. *The Predictive Value of Heart Rate and Heart Rate Variability for Time until First Reoffense*

Predictors	Outcomes		
	Hazard ratio	95% CI	<i>p</i>
Heart rate			
Resting	0.99	[0.96, 1.01]	.305
Reactivity	0.97	[0.95, 1.00]	.086
Heart rate variability			
Resting	1.18	[0.92, 1.52]	.202
Reactivity	0.81	[0.61, 1.09]	.161

Note. CI = confidence interval.

The Predictive Value of Heart Rate and Heart Rate Variability for Reoffending

To determine the predictive value of heart rate and heart rate variability for reoffending, we conducted univariate single-event survival analyses. Separate Cox regression models with the time between the initial measurement and the first reoffense as the outcome were fitted, each with a different heart rate or heart rate variability measure as the predictor. Results are presented in Table 2. An attenuated heart rate response to stress predicted time until the first reoffense; this finding showed a trend toward statistical significance. The hazard ratio is 0.97, meaning that when the heart rate response to stress is reduced by 5 bpm, the risk of reoffending enhances by a factor of 1.16. Resting heart rate did not predict time until first reoffense.

With respect to heart rate variability, we did not find a predictive value for time until first reoffense either at rest or in response to stress.

The Predictive Value of Heart Rate and Heart Rate Variability for the Number of Reoffenses

Next, we applied the Andersen–Gill model for recurrent events, which allows one to take into account all offenses committed during follow-up. Results are presented in Table 3. An attenuated heart rate response significantly predicted a higher rate of reoffending. The hazard ratio of the heart rate response is 0.969, meaning that when the heart rate response is reduced by 5 bpm, the mean number of reoffenses during the follow-up increases by a factor of 1.17 (Figure 2). Although we observed the same pattern for resting heart rate, this finding was not significant.

With respect to heart rate variability, we found that a stronger heart rate variability response (increased vagal withdrawal) significantly predicted a higher rate of reoffending (Table 3). The

Table 3. *The Predictive Value of Heart Rate and Heart Rate Variability for the Number of Reoffenses*

Predictors	Outcomes		
	Hazard ratio	95% CI	<i>p</i>
Heart rate			
Resting	0.99	[0.96, 1.03]	.586
Reactivity	0.97	[0.95, 0.99]	.017
Heart rate variability			
Resting	1.23	[0.94, 1.62]	.128
Reactivity	0.76	[0.59, 0.97]	.026

Note. CI = confidence interval.

hazard ratio of 0.757 means that when the heart rate variability response (a decrease) further decreases by 500 ms² (e.g., from –200 to –700 ms²), the mean number of reoffenses increases by a factor of 1.15 (Figure 2). Heart rate variability at rest did not significantly predict the number of reoffenses.

Discussion

The objective of our study was to examine the predictive value of heart rate and heart rate variability, in resting and in stressful conditions, for reoffending during a 5-year follow-up period in a sample of delinquent male adolescents. At follow-up, two thirds of the delinquents had reoffended. An attenuated heart rate response to stress as well as a stronger heart rate variability response to stress significantly predicted a higher reoffending rate.

The results of our study add to the existing literature in several ways. With respect to heart rate, we found attenuated heart rate responsivity in relation to reoffending. In previous studies, a low resting heart rate has been related to future antisocial behavior (Raine et al., 1990, 1995, 1997), although this was not confirmed in some other studies (Baker et al., 2009; van Bokhoven et al., 2005). Our findings add that attenuated heart rate responsivity predicts persistent antisocial behavior in a delinquent sample, an issue that had not previously been studied longitudinally. With respect to heart rate variability, both persisters and desisters displayed a similar decrease in response to stress, which is in line with previous cross-sectional studies on antisocial juveniles (Beauchaine et al., 2001, 2008; Mezzacappa et al., 1997). However, when extending this finding by using survival analyses as a more linear approach instead of comparing the two subgroups, a stronger heart rate variability response (increased vagal withdrawal) did predict a higher reoffending rate. This finding adds that heart rate variability responsivity has a predictive value for persistent delinquent behavior. From a clinical perspective, it is of importance that relationships were found in an adolescent delinquent sample. As such, the current study provides evidence for both heart rate and heart rate variability responsivity to stress to be neurobiological markers for persistence of antisocial behavior during adolescence. Hence, if confirmed in other studies, markers of ANS responsivity may be of additive value in the early prediction of persistent antisocial behavior.

When the results of our study are viewed in the perspective of the low arousal theory, the observed association between attenuated heart rate responsivity and persistent antisocial behavior fits in with this theory (Raine, 1993, 2002). In this view, attenuated heart rate responsivity reflects low responsivity to social cues in general, for example, punishment, and thus predisposes to antisocial development. Furthermore, the observed stronger heart rate variability response in relation to reoffending fits in with the polyvagal theory (Porges, 1995, 2007). Increased vagal withdrawal is thought to reduce a person's ability to inhibit impulses, causing fight or flight responding (Beauchaine, 2001; Porges, 2007). Although our findings may thus fit well in these two theoretical perspectives, low heart rate responsivity is thought to relate to fearlessness, whereas increased heart rate variability responsivity is thought to relate to fearfulness. In our study, however, differences in experienced fear did not seem to explain our findings, as persisters and desisters did not show differences in self-reported subjective well-being during the stress task. This finding of alterations in physiological but not emotional parameters in persistent delinquents is in line with previous

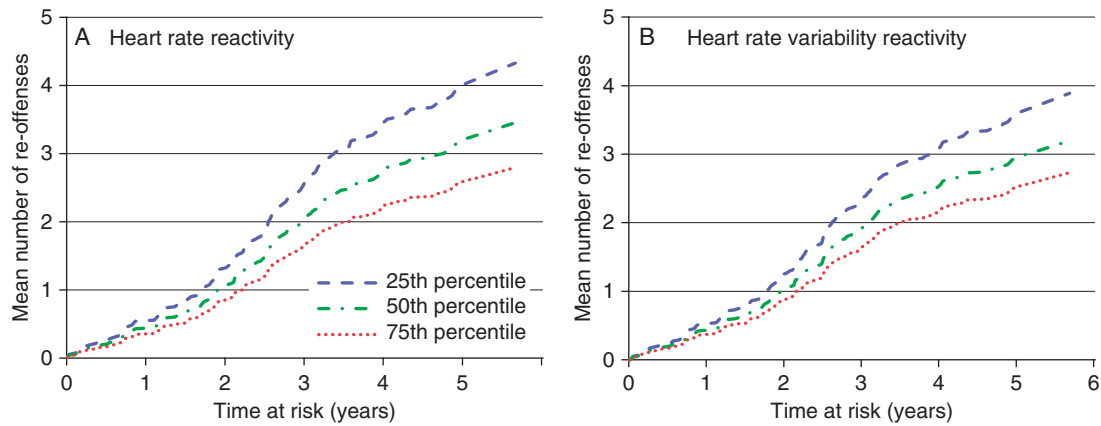


Figure 2. The predicted mean number of reoffenses of heart rate response (a) and heart rate variability (b) response to stress as a function of the time at risk. For the purpose of graphic representation, we display cumulative mean functions for three different levels of heart rate/heart rate variability response, corresponding to 25th, 50th, and 75th percentiles (heart rate, respectively, 2.3, 8.9, and 15.3 bpm increase; heart rate variability, respectively, -867 , -159 , and 405 ms² increase). Predictions are based on the Andersen–Gill model for recurrent events that was fitted to the data, analyses being performed with heart rate/heart rate variability as a continuous measure. An attenuated response to stress of both heart rate and heart rate variability was significantly associated with a higher reoffending rate.

studies (Fairchild et al., 2008; van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000).

Because SNS and PNS have been assumed to display coupled, reciprocal action on organ systems, increased vagal withdrawal was expected to be associated with an enhanced SNS response, producing a significant increase in heart rate and thereby stimulating fight or flight behaviors (Porges, 1995). However, we observed increased vagal withdrawal in combination with a decreased heart rate responsivity, the latter suggesting decreased SNS reactivity, although we did not measure SNS reactivity directly. This finding may be explained by research showing that the coupled activity of the two ANS branches may be either reciprocal or nonreciprocal, the latter entailing concurrent increases (coactivation) or decreases (coinhibition) in SNS and PNS activity (Berntson et al., 1991). Our finding of concurrent attenuated heart rate reactivity and increased vagal withdrawal, suggesting coinhibition, is in line with previous studies in which concurrent low levels of both PNS and SNS were found in relation to juvenile antisocial behavior (Beauchaine, Gatzke-Kopp, & Mead, 2007; Boyce et al., 2001; El-Sheikh et al., 2009).

The present findings are thus consistent with the view that disturbed ANS (re)activity may play a role in the development of delinquent behavior. Our effect sizes for differences between groups were moderate, and effect sizes for the predictive value of ANS markers for persistent delinquent behavior were small. When comparing our effect sizes to the overall effect sizes for heart rate reactivity reported in a meta-analysis by Ortiz and Raine (2004), we found that our effect sizes were in the same direction but smaller. One explanation may be found in the longitudinal design of our study, as Ortiz and Raine only used cross-sectional studies in their meta-analysis on heart rate reactivity. Another explanation may be found in the stressor we used in our study. The psychosocial stressor we used has been proven to elicit an increase in heart rate and a decrease in heart rate variability in healthy participants (Kudielka et al., 2004; Strahler et al., 2010). However, our stressor differs from stressors used in other studies that, for example, involved ANS reactivity to reward (Beauchaine et al., 2008). Moreover, some researchers have suggested that stronger associations of ANS measures and antisocial behavior may be observed when stressors are used that are more

intrinsically related to antisocial behavior, for example, frustration provocation tasks (van Goozen, Fairchild, Snoek, & Harold, 2007). Therefore, effect sizes (and results in general) may have differed when another task was used. Our results should therefore be interpreted with caution. They indicate that psychophysiological measures like ANS (re)activity alone only predict part of the variance of persistent delinquent behavior. Hence, ANS (re)activity is only one of many factors involved in the etiology of antisocial behavior. Other factors include social factors like dysfunctional family influences or childhood adversity (Moffitt & Caspi, 2001). There appear to be complex interplays between neurobiological and social factors (Raine, 2002). For example, although resting heart rate is often found to be lower in antisocial individuals, it may be a particularly strong characteristic of antisocial individuals from higher social classes (Raine & Venables, 1984). Moreover, the presence of both biological and social risk factors appears to exponentially increase rates of antisocial and violent behavior (Raine, 2002). More recently, van Goozen et al. (2007) proposed a comprehensive theoretical model in which neurobiological factors like attenuated ANS (re)activity are placed as possible mediators between childhood adversity and juvenile antisocial behavior. Recent work by El-Sheikh et al. (2009) indeed showed an interaction between SNS and PNS activity and marital conflict in relation to children's externalizing behavior. Coinhibition of SNS and PNS exacerbated the effect of marital conflict, whereas reciprocal activation buffered the effects. More (longitudinal) studies are needed to further unravel the role of neurobiological factors like attenuated autonomic stress responsivity in the etiology of antisocial behavior. The directions of the relationships then can be studied in more detail: Are they bidirectional, reciprocal, or transactional in nature? This will provide possibilities to make statements on causality as well (van Goozen et al., 2007).

Contrary to most of the findings in previous studies, the association between low baseline ANS measures and reoffending did not reach statistical significance in our study, although for resting heart rate the observed relations with antisocial parameters were in the same direction as for heart rate responsivity. In delinquent samples like in our study, there may be less variance in antisocial behavior as well as in baseline autonomic regulation

compared to general population samples, making differences in regulation of a system smaller and therefore harder to detect. By challenging a system, differences in regulation are enlarged and can thus be better identified (Kudielka & Wust, 2010). The use of a psychosocial stressor as the challenge may therefore be a more sensitive method to identify differences in autonomic regulation between groups. Indeed, Ortiz and Raine (2004) already found a larger effect size for stress-induced heart rate than resting heart rate in relation to antisocial behavior in their meta-analysis of cross-sectional studies.

The results of this study point to some recommendations for future research. First, future studies on the etiology and development of juvenile antisocial behavior should focus on multiple factors, neurobiological as well as psychological and social factors. For reasons mentioned above, parameters of both SNS and PNS should be taken into account when one studies ANS. Furthermore, interactions between ANS and the hypothalamic-pituitary-adrenal axis may provide valuable insights as well (Bauer, Quas, & Boyce, 2002). Relationships between all different factors may well be investigated from comprehensive biopsychosocial models. Second, future studies should examine the potential relevance for outcome prediction and intervention purposes. Measures of ANS (re)activity may be an additional tool for the early identification of those individuals within delinquent groups who are at risk of most negative outcome, for example, within groups of individuals who have committed a first offense. Taking into account ANS profiles in diagnostic processes may also help to predict effectiveness of current treatment options for antisocial juveniles. Preliminary evidence for this assumption was provided by Stadler et al. (2008). They studied school-aged children with a disruptive behavior disorder and found that those with low heart rates showed less reduction of disruptive symptoms after treatment than those with high heart rates. This finding clearly needs to be replicated in other samples and for different interventions, incorporating ANS measures at rest and in response to stress.

There are some methodological limitations of the study that should be considered when interpreting the results. First, we studied a specific population of delinquent male adolescents. Although studying such a specific group has evident relevance, results cannot be generalized to other samples like clinic-referred disruptive behavior disorder juveniles, very young offenders, or girls. With respect to the latter, in addition to the clear differences in the prevalence of antisocial behavior between boys and girls (Moffitt & Caspi, 2001), sex differences have been found regarding the ANS–antisocial behavior relationship (Beauchaine et al., 2008; Dietrich et al., 2007). Whereas aggressive boys demonstrated reduced autonomic functioning, aggressive girls displayed greater electrodermal responding and equal cardiovascular reactivity compared to controls. It has been suggested that girls' behaviors are being driven by different etiological mechanisms, perhaps including stronger social–environmental influences (Beauchaine et al., 2008). Second, reoffending was only determined from official registration systems. Offenses unknown to the police were not assessed, for example, by using self-report questionnaires. Both methods are, however, hampered by their own flaws. In self-reports, for instance, there is, in general, an overrepresentation of property offenses and an underrepresentation of serious violent offenses (Wittebrood, 2000). Nevertheless, results may have differed had self-reports had been taken into account.

Despite the limitations of the study, our results are an important contribution toward advancing knowledge on the role of stress-regulating mechanisms in persistence of juvenile antisocial behavior. Although effect sizes are small to moderate, our findings underscore the consistency of the relationship between disrupted stress responsivity and antisocial behavior. Our study shows that measures of ANS (re)activity may help understand the mechanisms that underlie patterns of persistent antisocial behavior. This may ultimately lead to more effective, tailor-made interventions for at-risk individuals to prevent future antisocial behavior at an early stage.

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