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Long-term alteration of heart rate variability following childhood maltreatment: Results of a general population study

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Research Article

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Abstract

Background. Childhood maltreatment (CM) is a risk factor for mental and physical health problems in adulthood, potentially mediated by long-term autonomic nervous system (ANS) dysregulation. To explore this link, the association between CM and vagal-sensitive heart rate variability (HRV) metrics in adults was examined, accounting for biopsychosocial factors.

Methods. Data from 4,420 participants in the Study of Health in Pomerania were analyzed, with CM assessed using the Childhood Trauma Questionnaire. HRV was derived from 10-second electrocardiograms and 5-minute pre-sleep polysomnographic recordings. Post hoc analyses examined abuse and neglect.

Results. CM was associated with reduced HRV (logRMSSD: $\beta = -0.20$ [95%-CI: −0.28, −0.12], $p = 1.2e{-}06$), driven by neglect ($\beta = -0.27$ [−0.35, −0.18], $p = 1.9e{-}09$) rather than abuse ($\beta = 0.01$ [−0.12, 0.14], $p = 1$). Adjustments for age, sex, and medication attenuated these effects, which remained robust after additionally controlling for socioeconomic, lifestyle, body mass index, and depressive symptoms (fully adjusted model: CM $\beta = -0.08$ [−0.15, −0.001], $p = .047$; neglect $\beta = -0.11$ [−0.19, −0.03], $p = .009$; abuse $\beta = -0.08$ [−0.20, −0.04], $p = .174$). Age-related differences were found, with reduced HRV in both young and older participants but not in middle-aged participants (fully adjusted: $F(2,743) = 6.75$, $p = .001$).

Conclusions. This study highlights long-term ANS dysregulation following CM, particularly neglect, indicated by altered vagal-sensitive HRV metrics. Although small in magnitude, the effect on the ANS was independent of adult biopsychosocial factors. This long-term dysregulation may contribute to an increased risk of adverse health outcomes in adulthood.

Introduction

Childhood maltreatment (CM), encompassing abuse (physical, emotional, sexual) and neglect (physical, emotional) [1], affects up to 60% of adults, with 30% experiencing at least moderate CM [2–4]. In general, CM emerges as a prominent risk factor for physical and mental health problems during adulthood [5–9].

CM has been linked to various medical conditions, including cardiovascular, respiratory, and liver diseases, cancer [9–11], as well as mental health problems encompassing depression and anxiety disorders, addiction, borderline personality disorder, antisocial behavior, and alexithymic personality traits [4, 12–18].

The pathogenic role of CM in this broad range of mental and physical conditions might be mediated by an increased stress vulnerability [19], marked by a chronic hyperarousal of the sympathetic nervous system [20]. This alteration of the autonomic nervous system (ANS) might impair the individual's flexibility to adapt adequately to changing environmental demands [21]. This chronic imbalance between the parasympathetic and sympathetic branches of the ANS can be investigated using vagal-sensitive metrics of heart rate variability (HRV) [22–24], which is defined as the timely variance between consecutive heartbeats [25]. Interestingly, higher HRV is associated with better coping skills, including emotion regulation, impulse control [26],

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and self-regulation [27], while lower HRV is linked with short-term [28], chronic [29], and social stress [30] as well as stress-associated mental disorders [31–33].

While the association of HRV and mental disorders has been extensively studied [31–34], the potential long-term effects of CM on ANS dysregulation have primarily been investigated in studies with small sample sizes ($N < 100$) or specific subgroups (e.g., only women), limiting the generalizability of these findings [35–39]. Notably, Bakema et al. [40] found a negative association between HRV and cumulative exposure to CM in a large population-based cohort. However, this association disappeared after adjusting for socioeconomic and demographic covariates. Meta-analyses provide further insights into this relationship. Sigrist et al. [41] found in their meta-analysis of 32 studies examining the effects of early life maltreatment on ANS dysregulation that the association varied as a function of age and the presence of psychopathology. In contrast, Wesarg et al. [42], focusing on the association of childhood adversities and vagal regulation, found altered HRV specifically in individuals exposed to direct childhood adversities. This specificity may arise as adverse childhood experiences encompass a broader range of negative life events compared to CM [43], suggesting potential long-term ANS dysregulation uniquely associated with CM exposure. Moreover, as these heterogeneous findings may stem from the characteristics of the included samples [44] or differences in the research construct, further investigation is needed to clarify the specific relationship between CM and HRV, including the potential influence of covariates.

Therefore, we aimed to investigate this association in a large population-based sample. However, HRV measurements with at least 5 minutes of rest, as recommended by guidelines [22, 45], are rarely applied in epidemiological studies. Nevertheless, 10-second electrocardiograms (ECG) are widely available in population-based studies, and their validity has been previously demonstrated [46]. Based on this, we investigated the association of CM and HRV using baseline data from the TREND cohort of the Study of Health in Pomerania (SHIP-TREND-0). Our analyses accounted for age, sex, HRV-altering medications, body weight, socioeconomic factors, lifestyle, and current depressive symptoms, while additionally considering the recency of CM.

Methods

Study population

Data were used from SHIP-TREND-0 (2008–2012; $N = 4{,}420$), the baseline from a population-based cohort study in northeastern Germany [47]. Participants were randomly selected from local registries in northeastern Mecklenburg-Western Pomerania, with 99.5% ($n = 4{,}396$) undergoing a standardized cardiological examination (10-second 12-lead ECG). Of these, 1,264 participants (28.6% of the cohort) underwent overnight polysomnography (PSG).

The study, which was conducted by trained staff, adhered to the principles of the Declaration of Helsinki, with written informed consent and approval from the Institutional Review Board of the University of Greifswald, Germany.

Data assessment

Participants underwent a standardized computer-assisted personal interview to assess sociodemographic factors, lifestyle, and medical history, followed by subsequent physical examinations, including anthropometric markers. Medication intake of the past week was registered and categorized according to the Anatomical Therapeutic Chemical (ATC) classification [48].

Socioeconomic factors encompassed education level and equivalized disposable income. The level of education was measured by considering the number of years of schooling and vocational training. The equivalized household income was quantified by dividing the household income (nine categories, ranging from 333€ to 5,067€) by the square root of the number of household members.

Lifestyle factors encompassed smoking status, alcohol consumption, and physical activity. Smoking status was categorized as never smoker, former smoker, or current smoker. Average alcohol consumption in grams per day (g/d) during the last 30 days was estimated based on self-reports of consumed alcoholic beverages. Regular physical activity for at least 1 hour per week throughout the year (no/yes) was determined by self-reported physical activity during summer and winter (four categories: none, less than 1 hour, 1–2 hours, more than 2 hours per week).

Electrocardiography

Cardiological examination

A 10-second 12-lead ECG was recorded using a CardioPerfect ECG system (Welch Allyn Inc., Auburn, NY, USA) with a sampling rate of 500 Hz, following standard clinical procedures [49]. The examiner tried to establish a calm and relaxed atmosphere, and participants rested in a supine position in a quiet room (temperature 23 ± 2 °C). Optimal signal quality was visually checked and, if necessary, optimized.

Polysomnography

A single-night PSG was conducted as outlined by Stubbe et al. [50], according to the American Academy of Sleep Medicine standards [51] using ALICE 5 PSG devices (Philips Respironics, Eindhoven, The Netherlands). Electrodes were placed carefully, and, among others, a one-channel ECG (Einthoven II) was recorded with a sampling rate of 200 Hz. All signals were checked and, if necessary, optimized. Bedtime and wake-up time were individually chosen by the participants, while a total bedtime of 8 hours was requested.

Questionnaires

Childhood trauma questionnaire

CM was assessed using the Childhood Trauma Questionnaire (CTQ [52]). According to the manual, exposure to CM is defined by reporting at least one moderate exposure in any subtype of CM. In addition, childhood neglect is defined as individuals encountering at least one moderate exposure to physical or emotional neglect, while childhood abuse is characterized by at least one moderate exposure to emotional, physical, or sexual abuse.

PHQ-9

Current depressive symptoms were assessed using the PHQ-9 questionnaire [53, 54]. For further details of the used questionnaires, see the supplement.

Data preparation

Heart rate variability

R-wave detection and calculation of HRV parameters, including the Root Mean Square of Successive Differences (RMSSD), Standard Deviation of the NN Intervals (SDNN), and low- (LF-HRV) and high-frequency HRV (HF-HRV), were performed using the HRVTool

toolbox [55] with an in-house script, as outlined by Krause et al. [46]. Following R-wave detection, a lead-specific template of the QRS complex was generated for each subject to validate detected R-peaks, helping to exclude ectopic beats and false detections (e.g., movement artifacts). Then, a filter was applied to remove implausible RR intervals. For multichannel ECGs, R-peaks detected on each lead were cross-validated across channels (further details in the supplement).

Three different HRV values of RMSSD were derived from the ECG sources. For all participants, ultra-short heart-rate variability (*usHRV*; i.e., HRV derived from less than 5 minutes) was obtained from 10-second ECG recordings. *HRV* was derived from a 5-minute segment of rest before sleep for those participating in PSG (see the Supplement for details). To minimize situation-specific variance, and as the HRV measurements were not directly recorded according to guidelines [22, 45], the obtained HRV values were averaged to yield a more trait-like HRV [56, 57] for conducting sensitivity analyses to validate and support the statistical findings using *usHRV*. Additionally, SDNN, LF-HRV, and HF-HRV were derived only from the 5-minute ECG, as their validity obtained from a 10-second ECG is questionable [58, 59].

Samples ECG data from SHIP-TREND-0 (N = 4,420) included 10-second ECGs for most participants (n = 4,396). PSG data were available for 1,264 participants. Individuals with specific heart problems (i.e., heart attack, pacemaker, atrial fibrillation, and heart surgery); stroke history; or pregnant women were excluded. Thus, two final samples were analyzed: a complete sample (n = 3,438) with one 10-second ECG recording and a PSG-subsample (n = 797) with both a 10-second ECG from the cardiological examination and a 5-minute ECG of wakeful rest from PSG (flowchart in Figure 1).

Statistical analysis

Multivariable linear regression models were used to estimate the effects of CM (*any CM*, *neglect*, and *abuse*) on HRV (logRMSSD) in the complete sample (*usHRV*) and the PSG-subsample (logRMSSD: *HRV, usHRV*, and trait-like HRV). The base model included *age* (nonlinear, three cubic splines – knots at the 10th, 50th, and 90th percentiles), sex, and their interaction as covariates. HRV declines with age [60], while its association with RMSSD remains inconsistent [61]. Antihypertensive (ATC codes: C02, C03, C07, C08, C09) and psychopharmacological medications (clozapine: N05AH02; tricyclics: N06AA, N06AX14), which may alter HRV [32, 62, 63], were added as covariates in the base model. Daytime was added as a covariate in analyses using *usHRV* or *HRV*.

We explored whether the effects of CM on HRV were mediated by body weight, lifestyle, socioeconomic factors, and current depressive symptoms. Body weight was assessed using the body mass index (BMI), encompassing underweight to overweight. Lifestyle factors included smoking, physical activity, and alcohol consumption interacted with sex. Socioeconomic factors included education level and equivalized disposable income. Current depressive symptoms were assessed using the log-transformed PHQ-9 sum score, exploring its influence on the relationship between CM and HRV, given the link between CM and increased depression risk and severity [4, 9, 18]. All continuous variables, except age, were modeled linearly.

RMSSD and the *PHQ-9* sum score were log-transformed to reduce skewness. Regression coefficients were considered significant at $p < .05$, while both p-values from the post hoc analyses of neglect and abuse were FDR-corrected [64]. All analyses were additionally conducted for log-transformed SDNN, HF-HRV, and LF-HRV in the PSG-subsample based on available 5-minute ECG recordings.

All models for logRMSSD were refitted with two modifications: (1) interaction term between CM and age to explore how HRV changes with CM recency, indicated by timing since childhood, and (2) heart rate (HR) as an outcome to investigate if effects were specific to HRV.

All statistical analyses were conducted in *R* (4.2.1) [65] and using the *rms* package [66]. A significance level of $p < .05$ was used for all analyses, and two-tailed tests were applied. Sample sizes varied

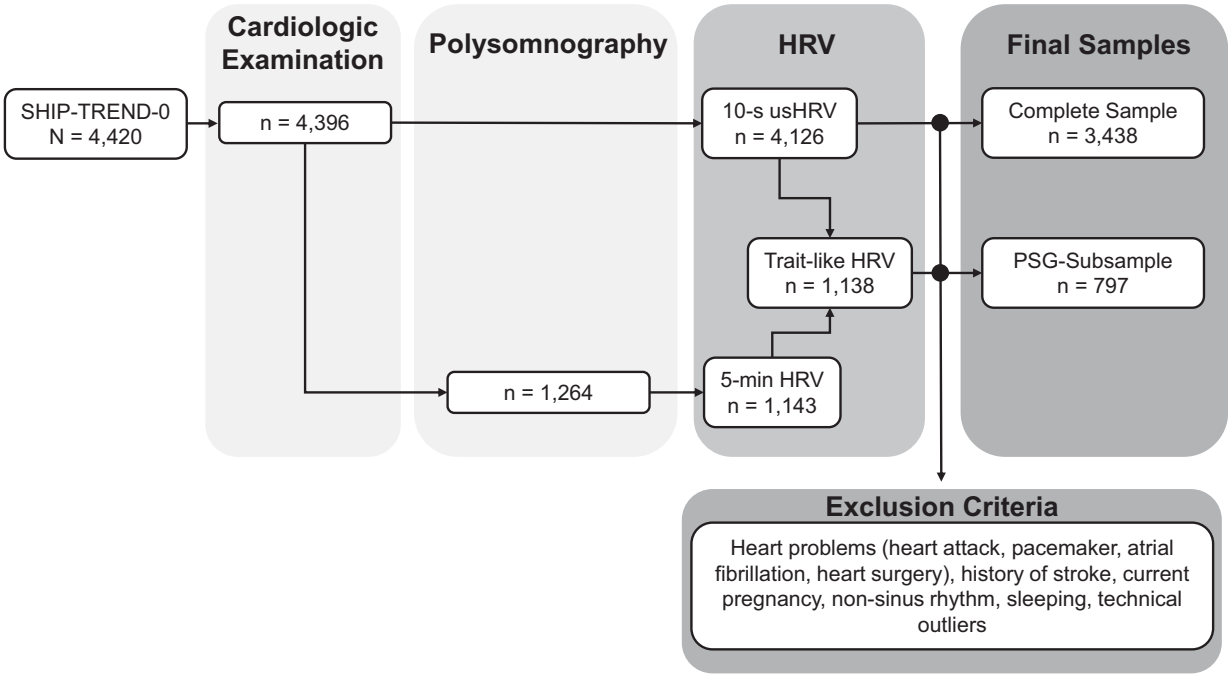

Figure 1. Flowchart of the samples and generated HRV parameters based on the ECG recordings in SHIP-TREND-0.

between models. Continuous variables were z-transformed within each model; thus, the reported regression coefficients are standardized. Visualizations are conditioned on median (continuous) and mode (categorical) values.

Results

Characteristics of the samples

The complete sample encompassed 3,438 participants, with an average age of 49.7 years (*SD* = 14.8), and 54.7% were women (see Table 1 for details; Supplementary Table S1 for the sex-stratified sample in the supplement). The PSG-subsample (*N* = 797 participants, 52.1% women) was largely comparable. However, the mean age was slightly higher (M[SD] = 51.7[13.4]), as were the BMI, educational status, equivalized disposable income, PHQ-9 sum score, and intake of antihypertensives. In contrast, more participants in the complete sample were currently smoking, were less physically active, and had a slightly higher resting HR.

In total, 22.3% of the participants reported any CM, whereas neglect was more prevalent (19.6%) than abuse (7.5%). These characteristics were similar in the PSG-subsample (any CM 23.0%; neglect 19.5%; abuse 8.0%).

Individuals exposed to CM had lower usHRV across both samples, as well as lower HRV and trait-like HRV in the PSG-subsample, with no differences in HR. In the complete sample, participants reporting CM were older, more obese, used antihypertensive medication more often, were less physically active, had fewer years of education, lower equivalized disposable income, and more current depressive symptoms. In the PSG-subsample, these trends were similar but mostly nonsignificant.

Association of HRV and CM

Overall, exposure to CM was found to be associated with lower HRV in the complete sample (usHRV: $\beta = -0.20$ [95%-CI: −0.28, −0.12], p = 1.2e-06) and the PSG-subsample (trait-like HRV: $\beta = -0.33$ [95%-CI: −0.49, −0.16], p = 1.1e-04; Table 2; Supplementary Table S2 for results of usHRV and HRV of the PSG-subsample).

Base model

Adjusting for age, sex, and antihypertensive and psychopharmacological medications, the negative association between HRV and CM was attenuated but remained significant in the complete sample (usHRV: $\beta = -0.09$ [95%-CI: −0.16, −0.02], p = .015) and in the PSG-subsample (trait-like HRV: $\beta = -0.27$ [95%-CI: −0.41, −0.12], p = 2.4e-04).

Adjusted models

Additional adjustments for socioeconomic factors, BMI, lifestyle, and depressive symptoms did not explain the association between CM and HRV. None of these factors, either individually or in combination, accounted for the observed effects.

Association of HRV and neglect

HRV was lower in individuals exposed to childhood neglect in the complete sample (usHRV: $\beta = -0.27$ [95%-CI: −0.35, −0.18], p = 1.9e-09) and the PSG-subsample (trait-like HRV: $\beta = -0.36$ [95%-CI: −0.53, −0.18], p = 1.8e-04).

Base model

Adjusting for age, sex, and HRV-altering medication attenuated the negative association between HRV and neglect but remained significant in the complete sample (usHRV: $\beta = -0.12$ [95%-CI: −0.19, −0.04], p = .008) and the PSG-subsample (trait-like HRV: $\beta = -.25$ [95%-CI: −0.40, −0.10], p = .004).

Adjusted models

Again, additional adjustments did not explain these effects.

Association of HRV and abuse

No association between HRV and childhood abuse was found in the complete sample (usHRV: $\beta = 0.01$ [95%-CI: −0.12, 0.14], p = 1) or in the PSG-subsample (trait-like HRV: $\beta = 0.01$ [95%-CI: −0.24, 0.27], p = 1), in either model.

Association of HR, SDNN, HF-HRV, and LF-HRV with CM

Exposure to CM showed no association with HR in either the complete sample or the PSG-subsample (Supplementary Table S3). In the PSG-subsample, no consistent associations were found between CM and SDNN, HF-HRV, or LF-HRV (Supplementary Tables S4–S6).

Association between HRV and the recency of CM

Exploring the interaction between CM and age at assessment, indicating CM recency, revealed no significant association in the complete sample (all $p > .05$; Table 3 and Supplementary Table S7). However, in the PSG-subsample, the interaction trended toward significance in the unadjusted model (trait-like HRV: p = .065) and reached significance when adjusting for covariates (base model: p = .029, BMI: p = .014; lifestyle factors: p = .024; socioeconomic factors: p = .004; depressive symptoms: p = .03, fully adjusted: p = .001). In the PSG-subsample, CM exposure was associated with reduced trait-like HRV in younger participants, an association that diminished at midlife but seemed to reemerge in older participants (Figure 2).

Similar, although less pronounced, patterns were observed for neglect and abuse in the PSG-subsample, but p-values survived FDR correction only in the fully adjusted model of neglect (HRV: p = .014; Supplementary Figure S1) and abuse (trait-like HRV: p = .036; Supplementary Figure S2).

Discussion

This study examined the long-term impact of CM on ANS dysregulation by exploring its relationship with vagal-sensitive metrics of HRV in a population-based sample. We revealed reduced HRV in adults exposed to CM, mainly driven by neglect. After adjusting for age, sex, and HRV-altering medication, the association was attenuated but remained stable when further accounting for BMI, lifestyle, socioeconomic factors, and current depressive symptoms. Notably, this is the first time these associations have been identified in a population-based sample using ultra-short HRV obtained from a 10-second multichannel ECG.

In detail, CM was associated with reduced 10-second usHRV in the complete sample and trait-like HRV in a subsample, indicating tonic disinhibition of the sympathetic branch of the ANS. This finding is in line with previous studies reporting reduced HRV in

Table 1. Sample characteristics

	Complete sample				PSG-subsample				ΔSamples
Sample	Complete	No CM	Any CM	*p*-value	Complete	No CM	Any CM	*p*-value	*p*-value
Sample size	3,438	2,670	768		797	614	183		
Age M[SD]	49.7 [14.8]	48.9 [14.65]	52.5 [14.7]	<.001	51.7 [13.4]	51.2 [13.3]	53.0 [13.5]	.112	<.001
Range	20–83	20–83	20–82		20–81	21–81	20–79		
Sex – Women [%]	1,882 [54.7]	1,465 [54.9]	417 [54.3]	.779	424 [52.1]	312 [50.8]	99 [54.1]	.839	.105
HRV – logRMSSD									
usHRV M[SD]	3.1 [0.7]	3.2 [0.7]	3.0 [0.7]	<.001	3.1 [0.7]	3.2 [0.7]	3.0 [0.6]	<.001	.276
HRV M[SD]					3.3 [0.6]	3.4 [0.6]	3.2 [0.6]	.003	
Trait-like HRV M[SD]					3.2 [0.5]	3.3 [0.5]	3.1 [0.5]	<.001	
Δdays ECG 10-s vs. PSG Mdn[range]					7 [0–83]	7 [0–83]	7 [0–76]	.381	
RMSSD									
usHRV M[SD]	28.2 [22.4]	29.0 [22.8]	25.5 [20.5]	<.001	27.0 [21.2]	28.2 [21.4]	22.8 [20.0]	.003	.153
HRV M[SD]					31.3 [18.8]	32.3 [19.3]	28.0 [16.6]	.007	
Trait-like HRV M[SD]					29.1 [17.3]	30.3 [17.8]	25.4 [15.0]	<.001	
Valid RR-intervals									
ECG 10-s M[SD]	10.0 [1.6]	10.0 [1.6]	10.0 [1.7]	.637	9.8 [1.5]	9.8 [1.5]	10.0 [1.6]	.076	.002
Range	5–16	5–16	6–15		6–15	6–15	7–15		
PSG 5-min M[SD]					323.2 [42.4]	322.3 [42.1]	326.1 [43.1]	.278	
PSG 5-min range					203–443	212–443	203–426		
Heart rate – BPM									
HR 10-s M[SD]	66.7 [9.4]	66.7 [9.4]	66.9 [9.5]	.657	65.5 [8.9]	65.2 [8.8]	66.3 [9.1]	.144	<.001
HR 5-min M[SD]					66.1 [8.4]	65.9 [8.4]	67.0 [8.4]	.111	
HR averaged M[SD]					65.8 [7.7]	65.6 [7.6]	66.7 [7.8]	.085	
Body weight kg M[SD]	80.6 [16.8]	80.2 [16.5]	81.8 [17.6]	.025	82.0 [15.7]	81.9 [15.2]	82.7 [17.5]	.569	.015
Body height cm M[SD]	170.0 [9.4]	170.3 [9.4]	169.1 [9.1]	.002	170.3 [9.1]	170.6 [9.0]	169.9 [9.1]	.387	.253
Body mass index, kg/m^2 M[SD]	27.8 [5.2]	27.60 [5.0]	28.6 [5.7]	<.001	28.2 [4.9]	28.1 [4.7]	28.6 [5.5]	.267	.034
Antihypertensives [%]	987 [28.7]	726 [27.2]	261 [34.0]	<.001	264 [32.4]	193 [31.4]	61 [33.3]	.072	.077
TCAs or Clozapine [%]	50 [1.5]	36 [1.4]	14 [1.8]	.333	11 [1.4]	8 [1.3]	3 [1.6]	.743	.874
Smoking status				<.001				.124	<.001
Never [%]	1,289 [37.5]	1,061 [39.7]	228 [29.7]		344 [42.3]	273 [44.5]	62 [33.9]		
Former [%]	1,188 [34.6]	882 [33.0]	306 [39.8]		312 [38.3]	218 [35.5]	88 [48.1]		
Current [%]	957 [27.8]	725 [27.2]	232 [30.2]		157 [19.3]	123 [20.0]	32 [17.5]		
Current Smoking [%]	957 [27.8]	725 [27.2]	232 [30.2]	.090	157 [19.3]	123 [20.0]	32 [17.5]	.005	<.001
Alcohol consumption, g/d M[SD]	8.4 [12.9]	8.4 [12.8]	8.2 [13.2]	.690	8.7 [12.1]	9.1 [12.5]	7.6 [10.5]	.155	.419
Physically active [%]	2,384 [69.3]	1,889 [70.8]	495 [64.5]	.001	600 [73.7]	463 [75.4]	122 [66.7]	.280	.024
Education years M[SD]	12.3 [2.5]	12.5 [2.5]	11.8 [2.4]	<.001	12.7 [2.4]	12.7 [2.3]	12.9 [2.3]	<.001	<.001
Education years Mdn[range]	11 [0–17]	11 [0–17]	11 [0–17]	<.001	13 [0–17]	13 [0–17]	13 [0–17]	<.001	<.001
Equivalized disposable income, Euro M[SD]	1,386 [722]	1,419 [725]	1,274[702]	<.001	1,411[734]	1,446 [735]	1,298 [725]	.019	.370
PHQ–9 sum score M[SD]	3.9 [3.5]	3.5 [3.1]	5.2 [4.4]	<.001	4.4 [3.7]	4.0 [3.4]	5.8 [4.3]	<.001	<.001
Childhood maltreatment									
Any CM – yes [%]	768 [22.3]	0	768 [100]	<.001	183 [23.0]	0	183 [100]	<.001	.704
Neglect – yes [%]	672 [19.6]	0	672 [87.5]	<.001	155 [19.5]	0	155 [84.7]	<.001	.938
Abuse – yes [%]	259 [7.5]	0	259 [33.7]	<.001	64 [8.0]	0	64 [35.0]	<.001	.645
CTQ Sum score M[SD]	34.2 [9.2]	30.8 [3.6]	46.7 [12.3]	<.001	34.3 [8.5]	31.0 [3.6]	45.7 [10.7]	<.001	.712

Continued

Table 1. *Continued*

Sample	Complete sample				PSG-subsample				ΔSamples
	Complete	No CM	Any CM	*p*-value	Complete	No CM	Any CM	*p*-value	*p*-value
Abuse									
Physical – yes [%]	135 [3.9]	0	135 [17.6]	<.001	32 [4.0]	0	32 [17.5]	<.001	.901
Emotional – yes [%]	138 [4.0]	0	138 [18.0]	<.001	30 [3.8]	0	30 [16.4]	<.001	.744
Sexual – yes [%]	98 [2.9]	0	98 [12.8]	<.001	27 [3.4]	0	27 [14.8]	<.001	.422
Neglect									
Physical – yes [%]	530 [15.4]	0	530 [69.0]	<.001	114 [14.3]	0	114 [62.3]	<.001	.417
Emotional – yes [%]	385 [11.2]	0	385 [50.1]	<.001	95 [11.9]	0	95 [51.9]	<.001	.557

Abbreviations: BPM, beats per minute; CM, Childhood Maltreatment; HR, heart rate; HRV, heart rate variability derived from polysomnography before falling asleep (5 min); logRMSSD, logarithmized root mean square of successive differences between heartbeats; M, mean; Mdn, median; PHQ-9, Patient health questionnaire; TCAs, tricyclic antidepressants; trait-like HRV, averaged usHRV and HRV; usHRV, HRV derived from 10-second ECG.

Table 2. Association of childhood maltreatment with heart rate variability

Regression model	Sample	HRV	Any CM		Neglect		Abuse	
			β (95% CI)	*p*-Value	*β* (95% CI)	p_{FDR}	*β* (95% CI)	p_{FDR}
Unadjusted	Complete sample	usHRV	−0.20 [−0.28, −0.12]	1.2e–06	−0.27 [−0.35, −0.18]	1.9e–09	0.01 [−0.12, 0.14]	1
	PSG-subsample	Trait-like HRV	−0.33 [−0.49, −0.16]	1.1e–04	−0.36 [−0.53, −0.18]	1.8e–04	0.01 [−0.24, 0.27]	1
Base model	Complete sample	usHRV	−0.09 [−0.16, −0.02]	.015	−0.12 [−0.19, −0.04]	.008	−0.10 [−0.22, 0.01]	.110
	PSG-subsample	Trait-like HRV	−0.27 [−0.41, −0.12]	2.4e–04	−0.25 [−0.40, −0.10]	.004	−0.20 [−0.43, 0.02]	.115
Base model + BMI	Complete sample	usHRV	−0.08 [−0.15, −0.01]	.025	−0.11 [−0.18, −0.03]	.008	−0.09 [−0.20, 0.03]	.110
	PSG-subsample	Trait-like HRV	−0.26 [−0.40, −0.12]	3.5e–04	−0.24 [−0.39, −0.09]	.004	−0.18 [−0.41, 0.04]	.115
Base model + lifestyle factors	Complete sample	usHRV	−0.09 [−0.16, −0.01]	.019	−0.11 [−0.19, −0.04]	.013	−0.10 [−0.22, 0.01]	.191
	PSG-subsample	Trait-like HRV	−0.26 [−0.40, −0.12]	3.7e–04	−0.24 [−0.39, −0.09]	.005	−0.20 [−0.43, 0.03]	.164
Base model + socioeconomic factors	Complete sample	usHRV	−0.08 [−0.16, −0.01]	.028	−0.11 [−0.19, −0.03]	.013	−0.10 [−0.22, 0.01]	.120
	PSG-subsample	Trait-like HRV	−0.26 [−0.41, −0.12]	4.5e–04	−0.24 [−0.39, −0.08]	.006	−0.20 [−0.43, 0.03]	.123
Base model + depressive symptoms	Complete sample	usHRV	−0.09 [−0.16, −0.01]	.019	−0.12 [−0.19, −0.04]	.018	−0.09 [−0.21, 0.02]	.132
	PSG-subsample	Trait-like HRV	−0.25 [−0.40, −0.11]	7.4e–04	−0.24 [−0.39, −0.09]	.009	−0.16 [−0.39, 0.08]	.142
Fully adjusted	Complete sample	usHRV	−0.08 [−0.15, −0.001]	.047	−0.11 [−0.19, −0.03]	.009	−0.08 [−0.20, 0.04]	.174
	PSG-subsample	Trait-like HRV	−0.25 [−0.40, −0.10]	.001	−0.24 [−0.39, −0.08]	.007	−0.14 [−0.38, 0.10]	.291

Note: Base model adjusted for age, sex, antihypertensive, and psychopharmacological medication (tricyclic antidepressants and clozapine), the interaction of sex with age and daytime in the case of usHRV; BMI, body mass index; lifestyle factors included smoking status (never, former, current), regular physical activity (less/at least 1 hour per week), alcohol consumption (in g/d) and its interaction with sex; socioeconomic factors included education and equivalized disposable income; depressive symptoms were assessed using the sum score of the PHQ-9 questionnaire; CI, confidence interval; FDR, false discovery rate to adjust for multiple testing; usHRV, HRV derived from 10-second ECG; trait-like HRV, HRV based on the average of usHRV and HRV.

individuals exposed to CM [35, 38–40]. However, our findings differ from those reported in an earlier population-based study [67] and in two meta-analyses [41, 42], likely due to methodological differences (e.g., assessment of HRV, HRV-parameters), sample characteristics, and the construct of CM in contrast to adverse childhood experiences. For instance, van Ockenburg et al. [67] investigated adverse childhood experiences rather than specifically CM, encompassing a broader spectrum of negative life events, some of which might be more easily buffered, for example, by sensitive and responsive caregiving. Similarly, the meta-analysis by Wesarg

Table 3. Association of HRV and childhood maltreatment with interaction of age

			Any CM			Neglect			Abuse		
Regression model	Sample	HRV	Any CM p	Age p	Any CM × Age p	Neglect p_{FDR}	Age p_{FDR}	Neglect × Age p_{FDR}	Abuse p_{FDR}	Age p_{FDR}	Abuse × Age p_{FDR}
Unadjusted	Complete sample	usHRV	1.2e–06			1.9e–09			1		
	PSG-subsample	Trait-like HRV	1.1e–04			1.8e–04			1		
Age	Complete sample	usHRV	.125	2.4e–164	.829	.041	3.5e–163	.639	.230	8.5e–169	.093
	PSG-subsample	Trait-like HRV	.653	9.0e–51	.065	1	9.8e–50	.357	.597	8.5e–51	.357
Base model	Complete sample	usHRV	.163	1.3e–136	.739	.065	3.5e–136	.618	.327	6.4e–140	.104
	PSG-subsample	Trait-like HRV	.559	4.9e–42	.029	1	8.8e–41	.321	.393	2.1e–42	.099
Base model + BMI	Complete sample	usHRV	.221	7.3e–125	.774	.095	1.5e–124	.695	.276	1.5e–127	.121
	PSG-subsample	Trait-like HRV	.395	3.8e–37	.014	1	6.6e–36	.267	.257	2.6e–37	.054
Base model + lifestyle factors	Complete sample	usHRV	.165	1.2e–130	.820	.077	2.9e–130	.711	.348	3.9e–134	.098
	PSG-subsample	Trait-like HRV	.490	8.0e–43	.024	1	1.0e–41	.332	.369	2.9e–43	.081
Base model + socioeconomic factors	Complete sample	usHRV	.334	2.9e–132	.590	.238	6.5e–132	.651	.317	1.1e–135	.136
	PSG-subsample	Trait-like HRV	.261	8.0e–40	.004	.741	1.3e–38	.084	.327	1.8e–40	.073
Base model + depressive symptoms	Complete sample	usHRV	.146	6.0e–136	.700	.055	1.9e–135	.581	.309	1.2e–139	.131
	PSG-subsample	Trait-like HRV	.533	1.1e–41	.030	1	1.5e–40	.296	.474	3.8e–42	.149
Fully adjusted	Complete sample	usHRV	.313	1.5e–114	.679	.226	4.0e–114	.755	.300	1.0e–117	.227
	PSG-subsample	Trait-like HRV	.140	3.5e–35	.001	.572	5.2e–34	.054	.276	1.5e–35	.036

Note: Base model adjusted for age, sex, antihypertensive, and psychopharmacological medication (tricyclic antidepressants and clozapine), the interaction of sex with age and daytime in the case of usHRV; BMI, body mass index; lifestyle factors included smoking status (never, former, current), regular physical activity (less/at least 1 hour per week), alcohol consumption (in g/d) and its interaction with sex; socioeconomic factors included education and equivalized disposable income; depressive symptoms were assessed using the total score of the PHQ-9 questionnaire; FDR, false discovery rate; usHRV, HRV derived from 10-second ECG; trait-like HRV, HRV based on the average of usHRV and HRV.

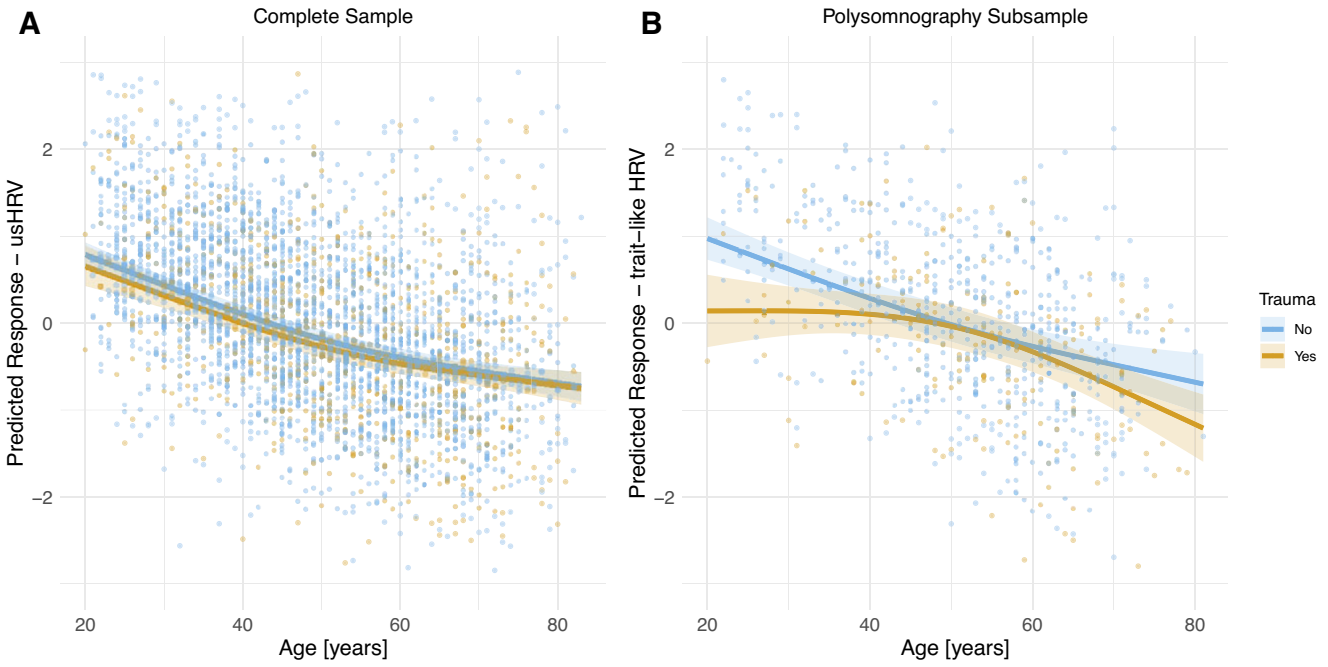

Figure 2. Predicted HRV (z-transformed logRMSSD) as a function of age and exposure to childhood maltreatment in the complete sample (A) and the PSG-subsample (B), both based on the fully adjusted model.

et al. [42] found no association with broader adverse childhood experiences but with CM.

The association between HRV and CM may be mediated by socioeconomic disadvantages, poor lifestyle, and mental health problems, which are more likely to emerge following exposure to CM [4, 9, 68], as well as by HRV-altering factors such as age, sex, physical fitness, and medication. HRV declines with age [46, 60] and is altered by medications such as antihypertensives, clozapine, and tricyclics [32, 62, 63]. Adjusting for these factors attenuated the association between HRV and CM but remained stable after further adjustments for socioeconomic status, lifestyle, current depressive symptoms, and BMI. This is interesting as CM is associated with unhealthy lifestyle factors (e.g., alcohol use, smoking, physical inactivity), depressive symptoms, and low socioeconomic status [4, 68, 69], all of which can alter HRV [61, 70–74]. Thus, our findings suggest small but substantial long-term ANS dysregulation in those exposed to CM, even after decades.

The observed association was particularly driven by neglect rather than abuse. Although abuse is generally considered more harmful and has been associated with altered HRV [35, 37], its relatively low prevalence in our sample (7.5%, n = 259) may have reduced the statistical power. Additionally, the characteristics of abuse may vary in timing, whereas neglect, defined as the experience of deprivation, is inherently chronic [75]. Thus, chronic stress induced by neglect might cause tonic sympathetic disinhibition, especially if it occurs during vulnerable developmental periods in childhood [76], leading to ANS dysregulation rooted in the prefrontal-amygdala axis in the brain [24]. As this may manifest as a disruption in a child's development, it can impair homeostasis and adaptive behavioral control [24], as well as emotional processing and executive functioning [35, 77]. This might have reduced stress resilience [78], facilitating the emergence of mental and physical health impairments in adulthood.

Our analysis revealed an age-dependent effect of CM on ANS dysregulation within the PSG-subsample. HRV was reduced in young adults exposed to CM, diminished by midlife, only to reemerge in older participants. Interestingly, this pattern was not observed in the complete sample, likely due to differences in sample characteristics, such as the PSG-subsample having fewer smokers, being more physically active, and possessing higher education. These factors might mediate a selection bias, particularly given the time-intensive nature of the additional PSG examination. Furthermore, the age effects may have been obscured in the complete sample, as only usHRV derived from 10-second ECGs was available as a surrogate.

Moreover, our PSG-subsample findings differ from two meta-analyses [41, 42] that found the association between adverse childhood experiences and ANS dysregulation to increase with age. Unlike these studies, which used mean ages across samples, we examined CM exposure within one large sample. Therefore, our results suggest that CM-related ANS dysregulation peaks in early adulthood, when exposure to CM is still recent, potentially affecting crucial personal, professional, and social development. However, this effect diminishes by midlife, perhaps due to compensatory factors or benefits from more stable life circumstances, before re-emerging in old age, possibly as CM-induced early life ANS dysregulation exerts a toll on the individual's health [79]. This later-life effect may be underestimated due to selection bias, as severely affected individuals might be less likely to participate in epidemiological studies or even due to survival bias.

Moreover, post hoc analyses indicated that this effect might stem from abuse and neglect. While our main analyses found ANS dysregulation primarily associated with neglect, these findings highlight the need for further research.

Limitations

We used ECGs recorded during rest to derive HRV, although they were only approximately recorded according to current guidelines [22, 45]. This might raise concerns about the validity of our results.

However, we previously demonstrated that a 10-second HRV can be a valid surrogate in large cohorts [46]. Additionally, we replicated our results using the average of two HRV values obtained from two different ECGs within a subgroup, aiming to derive a more reliable measure of HRV as a trait and minimizing situation-specific variance [56, 57]. In this regard, it is worth noting that our sample was phenotyped using identical assessments under continuous quality control procedures, resulting in a homogeneous sample, in contrast, for example, to a meta-analysis.

Our null finding on abuse should be interpreted cautiously, as previous studies found ANS dysregulation and adverse childhood experiences to vary as a function of psychopathology [41] and to be associated with abuse [35, 80]. In this regard, post-traumatic symptoms should be considered in future studies.

Moreover, our results should be replicated in other population-based cohorts, such as the German National Cohort, as previous findings on the association between HRV and CM were heterogeneous, possibly due to differences in HRV parameters, sample characteristics, and definitions of CM versus childhood adversity. Since HRV measures besides RMSSD in this study showed no substantial association, future research should focus on HRV metrics that provide insights into the overall autonomic regulation of cardiac function.

Additionally, investigating HRV's mediating effect on somatic outcomes like cardiovascular disease and mental health outcomes such as anxiety and depression, especially related to childhood neglect, could clarify how CM translates into long-term health risks.

CM was assessed using the self-report questionnaire CTQ, a valid and reliable tool, although retrospective reports of CM can be biased [81]. Replicating our findings with cross-validated data, such as interviews, is therefore advisable. For variables like alcohol consumption, medication, BMI, depressive symptoms, and income, only current data were available, limiting insights over the lifespan.

Summary and conclusion

Our results suggest long-lasting effects of ANS dysregulation following exposure to CM, especially neglect, based on HRV data derived from 10-second multichannel ECGs in a population-based sample. These effects remained robust, unaffected by socioeconomic, lifestyle factors, BMI, and current depressive symptoms, highlighting the need for prevention and intervention to reduce individual suffering and socioeconomic costs.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1192/j.eurpsy.2025.10040.

Data availability statement. The data from the SHIP study cannot be made publicly available due to the informed consent of the study participants; however, it can be accessed through a data application form available at https://fvcm.med.uni-greifswald.de/ for researchers who meet the criteria for access to confidential data.

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Author contribution. M.S., E.K., and H.J.G. designed the project. All coauthors contributed to the acquisition, analysis, or interpretation of the data. M.S., E.K., and H.J.G. drafted the manuscript, while all other coauthors critically revised it. MS and EK conceived and conducted the statistical analyses with support from J.K.K., M.V., S.F., and A.W., M.D., L.K., S.B.F., B.S., R.E., H.V., and H.J.G. were involved in data curation, administrative, technical, or material support, or funding acquisition. H.J.G. and E.K. supervised the project. All authors have read and approved the submitted version of the manuscript.

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References

[1] World Health Organization. Global status report on preventing violence against children 2020. Geneva: World Health Organization; 2020.
[2] Schulz A, Schmidt CO, Appel K, Mahler J, Spitzer C, Wingenfeld K, et al. Psychometric functioning, socio-demographic variability of childhood maltreatment in the general population and its effects of depression. Int J Methods Psychiatr Res. 2014;23(3):387–400.
[3] Gilbert R, Widom CS, Browne K, Fergusson D, Webb E, Janson S. Burden and consequences of child maltreatment in high-income countries. Lancet (London, England) 2009;373(9657):68–81.
[4] Klinger-König J, Streit F, Erhardt A, Kleineidam L, Schmiedek F, Schmidt B, et al. The assessment of childhood maltreatment and its associations with affective symptoms in adulthood. World J Biol Psychiatry. 2023;24(10):897–908.
[5] Bengtsson J, Elsenburg LK, Andersen GS, Larsen ML, Rieckmann A, Rod NH. Childhood adversity and cardiovascular disease in early adulthood: A Danish cohort study. Eur Heart J. 2023;44(7):586–93.
[6] Heim CM, Entringer S, Buss C. Translating basic research knowledge on the biological embedding of early-life stress into novel approaches for the developmental programming of lifelong health. Psychoneuroendocrinology. 2019;105:123–37.
[7] Spitzer C, Bouchain M, Winkler LY, Wingenfeld K, Gold SM, Grabe HJ, et al. Childhood trauma in multiple sclerosis: A case-control study. Psychosom Med. 2012;74(3):312–8.
[8] Spitzer C, Wegert S, Wollenhaupt J, Wingenfeld K, Barnow S, Grabe HJ. Gender-specific association between childhood trauma and rheumatoid arthritis: A case-control study. J Psychosom Res. 2013;74(4):296–300.
[9] Klinger-König J, Berger K, Grabe HJ, Erhardt A, Streit F, Völker MP et al. Childhood Trauma and Somatic and Mental Illness in Adulthood—Findings of the NAKO Health Study. Dtsch Arztebl Int. 2024;121(1):1–8.
[10] Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) study. Am J Prev Med. 1998;14(4):245–58.
[11] Kessler RC, McLaughlin KA, Green JG, Gruber MJ, Sampson NA, Zaslavsky AM, et al. Childhood adversities and adult psychopathology in the WHO world mental health surveys. Br J Psychiatry J Mental Sci. 2010;197(5):378–85.
[12] Hovens JGFM, Wiersma JE, Giltay EJ, van Oppen P, Spinhoven P, Penninx BWJH, et al. Childhood life events and childhood trauma in adult patients with depressive, anxiety and comorbid disorders vs. controls. Acta Psychiatr Scand. 2010;122(1):66–74.
[13] MacMillan HL, Fleming JE, Streiner DL, Lin E, Boyle MH, Jamieson E, et al. Childhood abuse and lifetime psychopathology in a community sample. Am J Psychiatry. 2001;158(11):1878–83.
[14] Porter C, Palmier-Claus J, Branitsky A, Mansell W, Warwick H, Varese F. Childhood adversity and borderline personality disorder: A meta-analysis. Acta Psychiatr Scand. 2020;141(1):6–20.
[15] Schulz A, Becker M, van der Auwera S, Barnow S, Appel K, Mahler J, et al. The impact of childhood trauma on depression: Does resilience matter?

Population-based results from the study of health in Pomerania. J Psychosom Res. 2014;77(2):97–103.
[16] Terock J, van der Auwera S, Janowitz D, Wittfeld K, Frenzel S, Klinger-König J, et al. Childhood trauma and adult declarative memory performance in the general population: The mediating effect of alexithymia. Child Abuse Negl. 2020;101:104311.
[17] Terock J, Weihs A, Teumer A, Klinger-König J, Janowitz D, Grabe HJ. Associations and interactions of the serotonin receptor genes 5-HT1A, 5-HT2A, and childhood trauma with alexithymia in two independent general-population samples. Psychiatry Res. 2021;298:113783.
[18] Humphreys KL, LeMoult J, Wear JG, Piersiak HA, Lee A, Gotlib IH. Child maltreatment and depression: A meta-analysis of studies using the childhood trauma questionnaire. Child Abuse Negl. 2020;102:104361.
[19] Simon L, Admon R. From childhood adversity to latent stress vulnerability in adulthood: The mediating roles of sleep disturbances and HPA axis dysfunction. Neuropsychopharmacology. 2023;48(10):1425–35.
[20] Murphy F, Nasa A, Cullinane D, Raajakesary K, Gazzaz A, Sooknarine V, et al. Childhood trauma, the HPA Axis and psychiatric illnesses: A targeted literature synthesis. Front Psych. 2022;13:748372.
[21] Thayer JF, Brosschot JF. Psychosomatics and psychopathology: Looking up and down from the brain. Psychoneuroendocrinology. 2005;30(10):1050–8.
[22] Malik M. Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. Circulation. 1996;93(5):1043–65.
[23] Thayer JF, Lane RD. A model of neurovisceral integration in emotion regulation and dysregulation. J Affect Disord. 2000;61(3):201–16.
[24] Smith R, Thayer JF, Khalsa SS, Lane RD. The hierarchical basis of neurovisceral integration. Neurosci Biobehav Rev. 2017;75:274–96.
[25] Beauchaine TP, Thayer JF. Heart rate variability as a transdiagnostic biomarker of psychopathology. Int J Psychophysiol 2015;98(2 Pt 2):338–50.
[26] Williams DP, Cash C, Rankin C, Bernardi A, Koenig J, Thayer JF. Resting heart rate variability predicts self-reported difficulties in emotion regulation: A focus on different facets of emotion regulation. Front Psychol. 2015;6:261.
[27] Segerstrom SC, Nes LS. Heart rate variability reflects self-regulatory strength, effort, and fatigue. Psychol Sci 2007;18(3):275–81.
[28] Castaldo R, Melillo P, Bracale U, Caserta M, Triassi M, Pecchia L. Acute mental stress assessment via short term HRV analysis in healthy adults: A systematic review with meta-analysis. Biomed Signal Process Control. 2015;18:370–7.
[29] Lampert R, Tuit K, Hong K-I, Donovan T, Lee F, Sinha R. Cumulative stress and autonomic dysregulation in a community sample. Stress. 2016;19(3):269–79.
[30] Lischke A, Jacksteit R, Mau-Moeller A, Pahnke R, Hamm AO, Weippert M. Heart rate variability is associated with psychosocial stress in distinct social domains. J Psychosom Res. 2018;106:56–61.
[31] Koch C, Wilhelm M, Salzmann S, Rief W, Euteneuer F. A meta-analysis of heart rate variability in major depression. Psychol Med. 2019;49(12):1948–57.
[32] Kemp AH, Quintana DS, Gray MA, Felmingham KL, Brown K, Gatt JM. Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. Biol Psychiatry. 2010;67(11):1067–74.
[33] Chalmers JA, Quintana DS, Abbott MJ-A, Kemp AH. Anxiety disorders are associated with reduced heart rate variability: A meta-analysis. Front Psych. 2014;5:80.
[34] Schneider M, Schwerdtfeger A. Autonomic dysfunction in posttraumatic stress disorder indexed by heart rate variability: A meta-analysis. Psychol Med. 2020;50(12):1937–48.
[35] Bussone S, Pesca C, Casetti V, Croce Nanni R, Ottaviani C, Troisi A, et al. The long-term impact of early adverse experience on adaptive functioning: A pilot study integrating measures of mental status, nonverbal communication, and heart rate variability. Eur J Psychotraumatol. 2023;14(1):2181766.
[36] Stone LB, Amole MC, Cyranowski JM, Swartz HA. History of childhood emotional abuse predicts lower resting-state high-frequency heart rate variability in depressed women. Psychiatry Res. 2018;269:681–7.
[37] Thurston RC, Carson MY, Koenen KC, Chang Y, Matthews KA, Känel R von et al. The relationship of trauma exposure to heart rate variability during wake and sleep in midlife women. Psychophysiology 2020;57(4):e13514.
[38] Krause-Utz A, Walther J-C, Kyrgiou AI, Hoogenboom W, Alampanou M, Bohus M, et al. Severity of childhood maltreatment predicts reaction times and heart rate variability during an emotional working memory task in borderline personality disorder. Eur J Psychotraumatol. 2022;13(1):2093037.
[39] Meyer P-W, Müller LE, Schmidinger I, Bohus M, Herpertz S, Bertsch K et al. Heart rate variability in patients with post-traumatic stress disorder or borderline personality disorder: Relationship to early life maltreatment. J Neural Transm. 2016;123(9):1107–18.
[40] Bakema MJ, van Zuiden M, Collard D, Zantvoord JB, Rooij SR de, Elsenburg LK et al. Associations between child maltreatment, autonomic regulation, and adverse cardiovascular outcome in an urban population: The HELIUS study. Front Psych 2020;11:69.
[41] Sigrist C, Mürner-Lavanchy I, Peschel SKV, Schmidt SJ, Kaess M, Koenig J. Early life maltreatment and resting-state heart rate variability: A systematic review and meta-analysis. Neurosci Biobehav Rev. 2021;120:307–34.
[42] Wesarg C, van den Akker AL, Oei NYL, Wiers RW, Staaks J, Thayer JF, et al. Childhood adversity and vagal regulation: A systematic review and meta-analysis. Neurosci Biobehav Rev. 2022;143:104920.
[43] Anda RF, Felitti VJ, Bremner JD, Walker JD, Whitfield C, Perry BD, et al. The enduring effects of abuse and related adverse experiences in childhood. Eur Arch Psychiatry Clin Neurosci. 2006;256(3):174–86.
[44] Cumming G. Replication and *p* intervals: P values predict the future only vaguely, but confidence intervals do much better. Perspect Psychol Sci. 2008;3(4):286–300.
[45] Quigley KS, Gianaros PJ, Norman GJ, Jennings JR, Berntson GG, de GEJC. Publication guidelines for human heart rate and heart rate variability studies in psychophysiology-part 1: Physiological underpinnings and foundations of measurement. Psychophysiology. 2024;61(9):e14604.
[46] Krause E, Vollmer M, Wittfeld K, Weihs A, Frenzel S, Dörr M, et al. Evaluating heart rate variability with 10 second multichannel electrocardiograms in a large population-based sample. Front Cardiovas Med. 2023;10:1144191.
[47] Völzke H, Schössow J, Schmidt CO, Jürgens C, Richter A, Werner A, et al. Cohort profile update: The study of health in Pomerania (SHIP). Int J Epidemiol. 2022;51(6):e372–83.
[48] ATC-Index. Anatomisch- therapeutisch- chemische Klassifikation mit Tagesdosen: Amtliche Fassung des ATC-Index mit DD-Angaben für Deutschland; 2007.
[49] Kligfield P, Gettes LS, Bailey JJ, Childers R, Deal BJ, Hancock EW, et al. Recommendations for the standardization and interpretation of the electrocardiogram: Part I: The electrocardiogram and its technology: A scientific statement from the American Heart Association electrocardiography and arrhythmias committee, council on clinical cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: Endorsed by the International Society for Computerized Electrocardiology. Circulation. 2007;115(10):1306–24.
[50] Stubbe B, Penzel T, Fietze I, Obst A, Garcia C, Zimmermann S, et al. Polysomnography in a large population based study-the study of health in Pomerania protocol. J Sleep Disord Manag. 2016;2(1):1–5.
[51] Iber C, Ancoli-Israel S, Chesson AL, Quand SF. The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications. 1st ed. Westchester, IL: American Academy of Sleep Medicine; 2007.
[52] Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the childhood trauma questionnaire. Child Abuse Negl. 2003;27(2):169–90.
[53] Kroenke K, Spitzer RL, Williams JB. The PHQ-9: Validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606–13.
[54] Gräfe K, Zipfel S, Herzog W, Löwe B. Screening psychischer Störungen mit dem "Gesundheitsfragebogen für Patienten (PHQ-D)". Diagnostica. 2004;50(4):171–81.
[55] Vollmer M. HRVTool – an open-source Matlab toolbox for analyzing heart rate variability. In: 2019 Computing in cardiology conference (CinC): Computing in cardiology, Singapore, IEEE; 2019:1–4.
[56] Laborde S, Mosley E, Thayer JF. Heart rate variability and cardiac vagal tone in psychophysiological research – recommendations for experiment planning, data analysis, and data reporting. Front Psychol. 2017;8:213.

[57] Bertsch K, Hagemann D, Naumann E, Schächinger H, Schulz A. Stability of heart rate variability indices reflecting parasympathetic activity. Psychophysiology. 2012;49(5):672–82.
[58] Baek HJ, Cho C-H, Cho J, Woo J-M. Reliability of ultra-short-term analysis as a surrogate of standard 5-min analysis of heart rate variability. Telemed J e-Health. 2015;21(5):404–14.
[59] Munoz ML, van Roon A, Riese H, Thio C, Oostenbroek E, Westrik I, et al. Validity of (ultra-)short recordings for heart rate variability measurements. PLoS One. 2015;10(9):e0138921.
[60] Schumann A, Bär K-J. Autonomic aging – a dataset to quantify changes of cardiovascular autonomic function during healthy aging. Sci Data. 2022; 9(1):95.
[61] Koenig J, Kemp AH, Beauchaine TP, Thayer JF, Kaess M. Depression and resting state heart rate variability in children and adolescents – a systematic review and meta-analysis. Clin Psychol Rev. 2016;46:136–50.
[62] Agelink MW, Majewski T, Wurthmann C, Lukas K, Ullrich H, Linka T, et al. Effects of newer atypical antipsychotics on autonomic neurocardiac function: A comparison between amisulpride, olanzapine, sertindole, and clozapine. J Clin Psychopharmacol. 2001;21(1):8–13.
[63] Maciorowska M, Krzesiński P, Wierzbowski R, Uziębło-Życzkowska B, Gielerak G. Associations between heart rate variability parameters and hemodynamic profiles in patients with primary arterial hypertension, including antihypertensive treatment effects. J Clin Med. 2022;11(13):1–11.
[64] Benjamini Y, Yekutieli D. The control of the false discovery rate in multiple testing under dependency. Ann Stat. 2001;29(4):1165–88.
[65] R Core Team. R: A language and environment for statistical computing. Vienna: R Foundation for Statistical Computing; 2021.
[66] Harrell FE JR. RMS: Regression Modeling Strategies; 2025.
[67] van Ockenburg SL, Tak LM, Bakker SJL, Gans ROB, de JP, Rosmalen JGM. Effects of adverse life events on heart rate variability, cortisol, and C-reactive protein. Acta Psychiatr Scand. 2015;131(1):40–50.
[68] Hughes K, Bellis MA, Hardcastle KA, Sethi D, Butchart A, Mikton C, et al. The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. Lancet Public Health. 2017;2(8):e356–66.
[69] Jaffee SR, Ambler A, Merrick M, Goldman-Mellor S, Odgers CL, Fisher HL, et al. Childhood maltreatment predicts poor economic and educational outcomes in the transition to adulthood. Am J Public Health. 2018;108(9): 1142–7.
[70] Thayer JF, Yamamoto SS, Brosschot JF. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. Int J Cardiol. 2010;141(2):122–31.
[71] El-Malahi O, Mohajeri D, Mincu R, Bäuerle A, Rothenaicher K, Knuschke R, et al. Beneficial impacts of physical activity on heart rate variability: A systematic review and meta-analysis. PLoS One. 2024;19(4):e0299793.
[72] Ralevski E, Petrakis I, Altemus M. Heart rate variability in alcohol use: A review. Pharmacol Biochem Behav. 2019;176:83–92.
[73] Dinas PC, Koutedakis Y, Flouris AD. Effects of active and passive tobacco cigarette smoking on heart rate variability. Int J Cardiol. 2013;163(2):109–15.
[74] Seeman T, Epel E, Gruenewald T, Karlamangla A, McEwen BS. Socioeconomic differentials in peripheral biology: Cumulative allostatic load. Ann N Y Acad Sci. 2010;1186:223–39.
[75] Leeb RT, Paulozzi LJ, Melanson C, Simon TR, Arias I. Child maltreatment surveillance: Uniform definitions for public health and recommended data elements. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2008.
[76] Lupien SJ, McEwen BS, Gunnar MR, Heim CM. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci. 2009;10(6):434–45.
[77] McLaughlin KA. Future directions in childhood adversity and youth psychopathology. J Clin Child Adolesc Psychol. 2016; 45(3):361–82.
[78] Chen MA, Suchting R, Thayer JF, Fagundes CP. Resilience to stress across the lifespan: Childhood maltreatment, heart rate variability, and bereavement. Psychol Aging. 2023;38(3):247–62.
[79] Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. Physiol Behav. 2012;106(1):29–39.
[80] Stevens SK, Williams DP, Thayer JF, Zalta AK. Differential associations of childhood abuse and neglect with adult autonomic regulation and mood-related pathology. Psychosom Med. 2023;85(8):682–90.
[81] Baldwin JR, Reuben A, Newbury JB, Danese A. Agreement between prospective and retrospective measures of childhood maltreatment: A systematic review and meta-analysis. JAMA Psychiatry. 2019;76(6): 584–93.