



# Reference values for short-term resting-state heart rate variability in healthy adults: Results from the Brazilian Longitudinal Study of Adult Health—ELSA-Brasil study

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## Abstract

Heart rate variability (HRV) is a psychophysiological phenomenon with broad implications, providing an accessible index of vagal function, underpinning psychological constructs, including the capacity for social engagement and emotion regulation, and may predict future morbidity and mortality. However, the lack of reference values for short-term HRV indices for participants of both sexes across the age spectrum is a limiting factor. This was the objective of the present study. Resting electrocardiographic records were obtained from 13,214 participants (both sexes, 35–74 years), and HRV indices in time and frequency domains (mean  $\pm$  SD) were determined from 5-min records. Results were based on a subsample of 2,874 nonmedicated, healthy participants stratified by sex across 10-year age groupings. Men showed lower heart rate (HR,  $64 \pm 8$  bpm vs.  $68 \pm 8$  bpm,  $p < .05$ ) and normalized high frequency (HF;  $39.4 \pm 18.0$  normalized units [n.u.] vs.  $50.4 \pm 18.5$  n.u.,  $p < .05$ ) than women, and higher N-N variance ( $2,214 \pm 1,890$  ms<sup>2</sup> vs.  $1,883 \pm 1,635$  ms<sup>2</sup>,  $p < .05$ ), standard deviation of all N-N intervals (SDNN;  $43.7 \pm 17.3$  ms vs.  $40.3 \pm 15.8$  ms,  $p < .05$ ) and LF/HF ( $2.30 \pm 2.68$  vs.  $1.33 \pm 1.82$ ,  $p < .05$ ). HR and HF (n.u.) were also higher in younger than older women. LF/HF was lower in women than men. Percentile curves showed almost all HRV indices decreasing with aging. The availability of short-term, resting-state HRV reference values in a large sample of healthy and non-medicated participants from 35–74 years will provide a valuable tool for researchers, clinicians, and those in the quantified-self community.

**KEYWORDS**

autonomic nervous system, cardiovascular diseases, frequency domain analysis, heart rate variability, reference values

## 1 | INTRODUCTION

Resting-state heart rate variability (HRV) is a psychophysiological phenomenon with broad implications. It provides an index of vagal function (Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013), which supports various psychological functions such as the capacity for social engagement (Kemp et al., 2012) and emotion regulation (Williams et al., 2015), and may even predict risk for future morbidity and mortality (Hillebrand et al., 2013; May & Arildsen, 2011; Schroeder et al., 2003). We have previously reported on the adverse effects of common mental disorders on HRV (Brunoni et al., 2013), especially in participants with generalized anxiety disorder (Kemp et al., 2014). Others have subsequently shown that changes in HRV precede the appearance of depressive symptoms (Jandackova, Britton, Malik, & Steptoe, 2016), consistent with the possibility that functioning of the vagus nerve plays an etiological role in depression onset. We have also reported that reductions in vagal function—indexed by HRV—may provide a “spark” that initiates a cascade of downstream effects, subsequently leading to cognitive impairment (Kemp, Lopez et al., 2016). In this study, we demonstrated a relationship between HRV and cognitive function, consistent with the proposal that HRV may reflect activity within prefrontal-vagal pathways (Thayer, Hansen, Saus-Rose, & Johnsen, 2009). We further demonstrated that this relationship was mediated by insulin resistance (a marker of Type 2 diabetes mellitus) and carotid intima-media thickness (subclinical atherosclerosis; Kemp, Lopez et al., 2016), lending further support to the possibility that HRV provides an early indicator of ill health that may have psychological and physiological consequences. Two recently published complementary models provide a foundation on which much of the HRV literature spanning psychological science and epidemiology may be understood. These include the GENIAL model (Kemp, Arias, & Fisher, 2017), which describes a theoretical pathway involving genomic and environmental influences on the vagus nerve that both supports and is impacted by social interaction, and plays an important regulatory role over allostatic processes that may lead to ill health and premature mortality. The complementary NIACT model refers to neurovisceral integration across a continuum of time (Kemp, Koenig, & Thayer, 2017), which, like GENIAL, helps to bridge the disciplinary divide from psychological science to epidemiology and public health. Both models emphasize vagal function as a critical mediating factor of health and well-being and provide new insights into the complex interaction among factors that may lead to longevity or premature mortality.

A major goal of the present study is to provide reference values for short-term, resting-state HRV in a large healthy sample of male and female participants across different age groups ranging from 35–74 years. This contribution represents a valuable resource for researchers, clinicians, those in the quantified-self movement, and will support future efforts to understand, identify, and predict personalized pathways to health and well-being. Although reference values for short-term measures of HRV in the time and frequency domains have been reported (Nunan, Sandercock, & Brodie, 2010; Task Force, 1996), their use remains limited because of the low sample size, variable inclusion criteria, and a lack of standardization across methods of data collection and analysis, making it difficult to draw comparisons across studies. A more recent review (Nunan et al., 2010) reported values for short-term HRV based on data in studies on small samples and nonstandardized data-collection procedures, and none of the reviewed studies had sufficient power to provide normal population values as we do here. Importantly, a variety of factors influence HRV including age (Antelmi et al., 2004), sex (Koenig, Rash, Campbell, Thayer, & Kaess, 2017; Koenig & Thayer, 2016), medication (Felber et al., 2006; Kemp et al., 2014), and ill health (Buccelletti et al., 2009; Kemp et al., 2010; May & Arildsen, 2011; Schroeder et al., 2003). A population-based study (Felber et al., 2006) provided normal values for long-term HRV measurements; however, since that study was published, there has been increasing interest in use of short-term measures. The sample was also unbalanced between sexes (329 women and 170 men) and included a restricted age range of participants (50–72 years). Recently, another study (Sammito & Böckelmann, 2016) in a sample of 695 individuals (aged 20–60 years) reported reference values for time and frequency domain HRV from 24-hr records. Yet, methodological problems involving data preprocessing and analysis were detected (Bauer et al., 2017), leading to a report on new values (Sammito & Böckelmann, 2017). However, this report is based on long (24-hr) electrocardiographic (ECG) records, highlighting a continued need for short-term HRV reference values.

Therefore, the aim of the present study was to establish reference values for the most used linear indices of short-term HRV collected under a standardized and resting-state condition while participants were in the supine position. Values were derived from a robust sample of adults (35–74 years) included in the Longitudinal Study of Adult Health (ELSA-Brasil study). Data were obtained from short-term ECG recordings in healthy participants who were medication free, allowing for future unbiased comparisons with patients from a variety of clinical conditions. Given the

importance of the HRV metric as well as the increasing interest in short-term measures of HRV, it is critical that standardized indices from large and representative populations are available.

## 2 | METHOD

### 2.1 | Design, participants, and procedures

This study reports on baseline data from the ELSA-Brasil study collected between 2008 and 2010. A sample of 15,105 civil servants from five public universities and one research institute from three Brazilian regions (Northeast, Southeast, and South) were included. The ethics committee of each institution approved the research protocol, and all participants, aged 35–74 years, active or retired, signed written consent before participation. Questionnaires were administered by trained interviewers, and clinical and laboratory exams were carried out during a scheduled visit to one of the six investigation centers. Personal data, demographic characteristics, and all medications under regular use were recorded during the interview. Venous blood samples, anthropometric (body weight and stature) and hemodynamic data (blood pressure) were collected by trained technicians. Blood pressure (BP) was recorded in the morning period, in fasting conditions, and all were instructed to avoid consumption of coffee, cigarettes, and alcoholic beverages, as well as not to exercise on the day before the exam. BP was recorded at rest in the left arm in the sitting position. Measurements were performed using a validated oscillometric device (Omron HEM 705CPINT). Three measures were taken at 1-min intervals, and the arithmetic mean of the two last readings was used to determine the values of systolic and diastolic BP. Biochemical analyses of blood and urine were performed in a central laboratory using commercially available kits (Aquino et al., 2012).

Reference values for HRV were generated from a subsample of healthy participants with a validated ECG recording. The following exclusion criteria were used: (a) current or recent (<4 months) pregnancy; (b) use of any medication ( $N = 8,892$ ), except oral contraceptives and vitamins; (c) self-report of previous cardiovascular or cerebrovascular disease or cardiac surgery (myocardial infarction,  $N = 278$ ; stroke,  $N = 201$ ), including vascular stents ( $N = 268$ ); (d) presence of obesity (body mass index  $\geq 30 \text{ kg/m}^2$ ,  $N = 3,464$ ), diabetes (fasting glycemia  $\geq 126 \text{ mg/dL}$ ,  $N = 2,970$ ), hypertension (blood pressure  $\geq 140/90 \text{ mmHg}$ ,  $N = 5,363$ ) or chronic kidney disease (creatinine  $\geq 1.4 \text{ mg/dL}$  for men,  $N = 341$ , and  $\geq 1.2 \text{ mg/dL}$  for women,  $N = 130$ ); and (e) common mental disorders (CMD, determined by the Clinical Interview Schedule–Revised; Lewis, Pelosi, Araya, & Dunn, 1992, in which a score  $\geq 12$  indicated a current CMD (within the last week),  $N = 4,036$ ). After exclusions, 2,874 apparently healthy participants were available for analysis, on which reference HRV indices were determined (Figure 1).

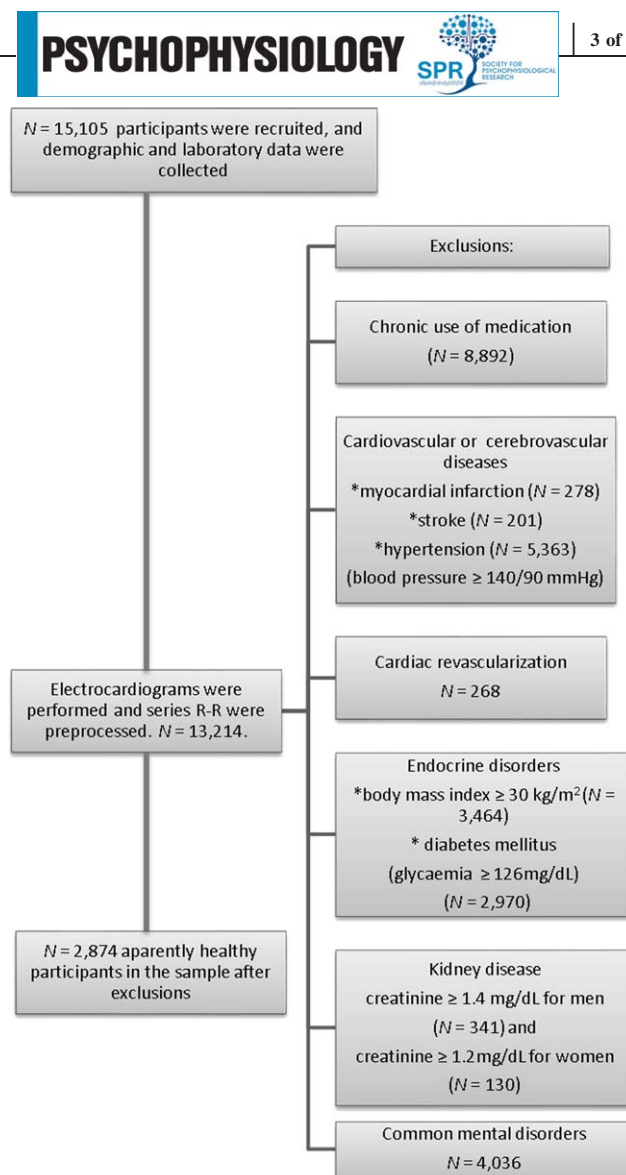


FIGURE 1 Exclusion criteria for the sample

### 2.2 | ECG records and HRV analysis

A 10-min continuous ECG recording was carried out on all participants (sampling rate of 250 Hz, Micromed, Brazil). Participants were in the supine position in a quiet and temperature-controlled room (20–24°C) while the ECG recording was obtained from a lead with the highest R wave amplitude (usually Lead 2). A computer program (WinCardio 4.4) generated the time series of R–R intervals, which were sent to a central cardiovascular physiology laboratory for further analyses (Mill et al., 2013).

The R–R series were automatically preprocessed to remove ectopic beats and artifacts. Linear interpolation was used to replace removed beats, in accordance with the following criteria: each R–R interval was compared to a reference called  $R-R_{\text{average}}$ . The  $R-R_{\text{average}}$  was calculated as the average of the last 10 normal R–R intervals. R–R intervals smaller than 80% of the  $R-R_{\text{average}}$ , or greater than 120% of the  $R-R_{\text{average}}$ , were considered as ectopic beats and were removed from the R–R series. These beats were replaced by

**TABLE 1** Demographic characteristics of the participants

	All (N)	Men (N)	Women (N)	Pearson chi-square	p
Age range (years)					
35–44	982	593 (35.3%)	389 (32.6%)	2.78	.43
45–54	1,252	719 (42.8%)	533 (44.6%)		
55–64	536	305 (18.2%)	231 (19.3%)		
65–74	104	63 (3.7%)	41 (3.4%)		
Race/ethnicity					
Black	381	213 (12.8%)	168 (14.3%)	14.72	.005
Brown	859	523 (31.5%)	336 (28.6%)		
White	1,491	871 (52.5%)	620 (52.7%)		
Asiatic	82	35 (2.1%)	47 (4.0%)		
Indigenous	22	17 (1.0%)	5 (0.43%)		
Education level					
< 8 years	122	102 (6.1%)	20 (1.7%)	55.22	< .001
8–11 years	1,140	699 (41.6%)	441 (36.9%)		
≥ 12 years	1,612	879 (52.3%)	733 (61.4%)		
Smoking status					
Current	371	237 (14.1%)	134 (11.2%)	23.67	< .001
Past	683	441 (26.2%)	242 (20.2%)		
Never	1,820	1,002 (59.6%)	818 (68.5%)		

Note. Comparisons were performed with chi-square test.

linear interpolation. If R–R series were changed by more than 20%, they were excluded from analyses (Dantas et al., 2012, 2015). According to this criterion, 3.8% of the ECG records were invalid and excluded from HRV analysis.

Short-term HRV analyses were carried out on validated and artifact-free R–R series in the time and frequency domain using MATLAB customized software (Dantas et al., 2015). For this purpose, 5-min segments were extracted from each 10-min R–R series. Based on criteria of greater stability, a standardized 5-min segment of data was analyzed from 2 min and 30 s until 7 min and 30 s. Each segment was preprocessed aiming to remove trends, subtracting the values of a linear regression function from the R–R series. Time domain analyses consisted of the mean and variance of all normal-to-normal intervals (N–N), the standard deviation of all N–N intervals (SDNN), the percentage of successive R–R intervals differing by more than 50 ms (pNN50), where NN50 is the number of pairs of adjacent N–N intervals differing by more than 50 ms, and the square root of the mean of the sum of the squares of differences between adjacent N–N intervals (RMSSD; Task Force, 1996). Frequency domain analyses were performed by autoregressive (AR) modeling and fast Fourier transform (FFT). The AR model was estimated using the Yule Walker algorithm, using a constant model order set at 16 (Dantas et al., 2012, 2015). For FFT analysis, the R–R series were resampled in the time at a sampling rate of 4 Hz, and the frequency spectrum was divided into 1,024 points (Task Force,

1996). The very low frequency (VLF, 0–0.04 Hz), low frequency (LF, 0.04–0.15 Hz), and high frequency (HF, 0.15–0.40 Hz) were expressed in absolute values and in normalized units. Normalization was performed dividing the amount of power of each component by total power minus the power of the VLF component (Malliani, Pagani, Lombardi, & Cerutti, 1991; Task Force, 1996). The LF/HF ratio was also calculated.

## 2.3 | Statistical analysis

Data normality was evaluated by Shapiro-Wilk test and variance homogeneity by Levene's test. Demographic categorical variables were compared between sexes by chi-square test, and continuous variables by Wilcoxon-Mann-Whitney test. Next, the participants were categorized into four age groups by decades (35–74 years). The mean, standard deviation, and 2.5th, 10th, 25th, 50th, 75th, 90th, and 97.5th percentiles were calculated for each variable. A one-way analysis of variance (ANOVA) across age groups and a two-way ANOVA (including age and sex factors allowing for the interaction between age and sex to be determined) were carried out on HRV. Post hoc tests were conducted using Fisher's protected least significant difference (LSD) method. Quantile regression was used to estimate the percentiles (from 2.5th to 97.5th) of the HRV indices by age, and curves were generated for each HRV variable. In the body of the text, values are shown as mean  $\pm$  SD. Comparisons were



**TABLE 2** Characteristics of the participants

	All ( <i>N</i> = 2,871)	Men ( <i>N</i> = 1,680)	Women ( <i>N</i> = 1,191)	<i>p</i>
Age (years)	47 (11)	47 (10)	47 (11)	.2508
Height (cm)	167.3 (14.1)	172.6 (9.6)	159.3 (8.3)	< .0001
Body weight (kg)	68.7 (16.2)	74.1 (13.6)	61.15 (12.05)	< .0001
Body mass index (kg/m <sup>2</sup> )	24.6 (4.1)	25.0 (3.9)	24.1 (4.4)	< .0001
Waist circumference (cm)	85.7 (13.7)	89.5 (11.5)	80.4 (11.8)	< .0001
Systolic blood pressure (mmHg)	115 (17)	119 (16)	109 (16)	< .0001
Diastolic blood pressure (mmHg)	72 (13)	75 (12)	69 (11)	< .0001
Glycemia (mg/dL)	102 (12)	104 (11)	100 (11)	< .0001
Creatinine (mg/dL)	0.90 (0.30)	1.00 (0.20)	0.80 (0.20)	< .0001
Total cholesterol (mg/dL)	211 (52)	210 (51)	212 (52)	.3381
HDL (mg/dL)	54 (18)	50 (15)	61 (19)	< .0001
LDL (mg/dL)	130 (42)	132 (41)	128 (43)	.0089
Triglycerides (mg/dL)	101 (74)	115 (85)	86 (55)	< .0001

Note. Values are median and interquartile range (differences between percentiles 75–25). Comparisons between sexes were performed by Wilcoxon-Mann-Whitney test. *N* = number of subjects.

considered significant only when *p* was < .05. All statistical analyses were carried out with Stata 12.0/SE.

### 3 | RESULTS

Demographic characteristics of the participants (*N* = 2,874) are shown in Table 1. As expected, most of the participants were White, with medium to high level of education, and nonsmokers. In the entire group, 12.9% were current smokers, 23.7% were former smokers, and 63.3% had never smoked. The proportion of current smokers was slightly higher in men than in women (14.1 vs. 11.4%; *p* < .001). Characteristics of the sample are shown in Table 2. Except for age and total cholesterol, all other variables were significantly different between sexes.

Time and frequency domain analyses (Table 3 and 4, respectively) show differences in age on most HRV indices, such that HRV was higher in younger than in older groups. However, there were no differences for heart rate, normalized LF, and LF/HF ratio.

Comparing both sexes (in the online supporting information, Table S1 and S2), men displayed lower mean heart rate ( $64 \pm 8$  bpm vs.  $68 \pm 8$  bpm, *p* < .05) and normalized HF ( $39.4 \pm 18.0$  normalized units [n.u.] vs.  $50.4 \pm 18.5$  n.u., *p* < .05) than women, but higher N-N variance ( $2,214 \pm 1,890$  ms<sup>2</sup> vs.  $1,883 \pm 1,635$  ms<sup>2</sup>, *p* < .05), SDNN ( $43.7 \pm 17.3$  ms vs.  $40.3 \pm 15.8$  ms, *p* < .05), VLF ( $1,021.4 \pm$

$1,033.2$  ms<sup>2</sup> vs.  $832.1 \pm 821.4$  ms<sup>2</sup>, *p* < .05), absolute LF ( $580.3 \pm 608.9$  ms<sup>2</sup> vs.  $385.6 \pm 443.5$  ms<sup>2</sup>, *p* < .05), normalized LF ( $56.3 \pm 19.2$  n.u. vs.  $43.7 \pm 19.0$  n.u., *p* < .05), and LF/HF ratio ( $2.30 \pm 2.68$  vs.  $1.33 \pm 1.82$ , *p* < .05) in AR analyses. Similar differences between sexes were observed in FFT analyses (Table S4). Considering the interaction between age and sex (supporting information, Table S1, S2, S4), significant differences were observed in heart rate, N-N variance, SDNN, VLF (ms<sup>2</sup>), LF (ms<sup>2</sup>), HF (ms<sup>2</sup>), LF (n.u.), HF (n.u.), and LF/HF ratio. N-N variance, SDNN, pNN50 and RMSSD, LF (ms<sup>2</sup>), HF (ms<sup>2</sup>), HF (n.u.) were higher in younger participants of both sexes. Heart rate, HF (ms<sup>2</sup>), and HF (n.u.) were higher in younger than older women. LF/HF ratio was lower in women compared to men.

Figure 2 and 3 show the changes of the HRV indices in percentiles as a function of age. It is noteworthy that almost all HRV indices decrease as the age increases in both time and frequency domain. While the LF/HF ratio (Figure 2) determined using the AR method tends to decrease slightly (median or percentile 50) over age, LF/HF ratio increases when determined using FFT (Figure S1). This fact occurs because in AR analyses the fall in LF component (absolute and normalized) is higher than that observed in HF component. Thus, the ratio tends to be reduced over age. On the other hand, in FFT analyses, the reduction in LF component tends to be less than for the HF component. In fact, normalized LF increases while

**TABLE 3** Time domain analysis

	Age range (years)	N	p ANOVA	Mean	SD	Percentiles						
						2.5	10	25	50	75	90	97.5
Heart rate (bpm)	35–44	982	.2429	66	9	50	54	60	66	72	77	86
	45–54	1,252		65	8	51	55	60	65	71	76	83
	55–64	536		65	8	51	56	60	65	70	75	82
	65–74	104		65	9	46	55	59	64	72	78	85
N-Nvariance (ms <sup>2</sup> )	35–44	982	.0000	2378	1931	439	727	1145	1844	2966	4841	7484
	45–54	1,252		1995**	1644	360	544	882	1471	2583	4036	6685
	55–64	536		1769**	1748	223	480	759	1249	2120	3435	7142
	65–74	104		1804**	2046	221	380	619	1062	2031	4536	9101
SDNN (ms)	35–44	982	.0000	45.7	17.0	21.0	27.0	33.8	42.9	54.5	69.6	86.5
	45–54	1,252		41.6**	16.2	19.0	23.3	29.7	38.3	50.8	63.5	81.8
	55–64	536		38.7**	16.5	14.9	21.9	27.6	35.3	46.0	58.6	84.5
	65–74	104		37.9**	19.2	14.9	19.5	24.9	32.6	45.1	67.3	95.4
pNN50 (%)	35–44	982	.0000	14.0	15.4	0.0	0.4	2.2	8.0	20.6	37.2	55.1
	45–54	1,252		10.0**	12.9	0.0	0.0	0.9	4.7	14.1	28.4	47.1
	55–64	536		7.2**	11.1	0.0	0.0	0.6	2.7	8.4	21.3	40.4
	65–74	104		7.3**	13.2	0.0	0.0	0.3	1.7	8.9	21.9	60.6
RMSSD (ms)	35–44	982	.0000	34.9	17.5	12.1	17.0	23.1	31.0	42.4	58.4	81.2
	45–54	1,252		30.0**	15.6	10.0	13.8	19.1	26.6	37.1	50.2	69.4
	55–64	536		26.7**	15.3	9.5	12.6	17.5	23.1	31.9	44.0	67.5
	65–74	104		27.7**	23.6	7.4	10.3	15.0	21.1	31.6	48.8	115.7

Note. Comparisons between means performed by one-way ANOVA followed by Fisher's LSD multiple comparisons. Significant differences against 35–44 age range.

\*\* $p < .01$ .

normalized HF decreases slightly over age, and LF/HF ratio becomes higher (Table S4).

Additional analyses were performed by age range and ethnic group (Table S5 and S6), and clinical conditions such as hypertension (Table S7 and S8), and mental disorder (Table S9 and S10). Considering individuals of the same race, younger individuals (35–44 years) showed higher values of N-N variance and SDNN. Differences in between-race comparisons were noticed for time (N-N variance and SDNN) and frequency domain analyses (Ln LF ms<sup>2</sup>). Young Indigenous subjects showed the highest values of N-N variance and SDNN, while White subjects showed higher values of Ln LFms<sup>2</sup> than Brown subjects. Asiatic subjects showed lower Ln LF ms<sup>2</sup> than Brown and White subjects. For hypertensive participants, no significant difference was observed among subjects of different age ranges. In participants with mental disorders, there was a noticeable effect of age in time (N-N variance, SDNN, pNN50, RMSSD) and frequency domain (VLF, absolute LF and HF) indices, in that all were decreased in older subjects.

Finally, the analyses were performed in all individuals of the sample (Table S11 and S12). The same differences described above for apparently healthy individuals were also retained when the analyses were carried out without exclusion of the sample, highlighting the extensive impacts of gender and age. Again, most HRV variables in time and frequency domains decreased over age. The LF/HF ratio increased with age in both women and men, although in the older age range, values were lower in men.

## 4 | DISCUSSION

Although the need for HRV reference values in large population studies was identified many years ago by the Task Force of the European Society of Cardiology and the North American Society for Pacing and Electrophysiology (Task Force, 1996), few studies have addressed this need in relation to short-term HRV indices. Considering the broad interest in HRV spanning fields as diverse as psychological science and

**TABLE 4** Autoregressive frequency domain analysis

	Age range (years)	<i>N</i>	<i>p</i> ANOVA	Mean	<i>SD</i>	Percentiles						
						2.5	10	25	50	75	90	97.5
VLF (ms <sup>2</sup> )	35–44	982	.0326	1009.2	934.8	108.5	247.1	397.4	731.5	1258.5	2168.7	3654.4
	45–54	1,252		920.5*	936.5	113.4	207.6	346.8	642.3	1153.3	1986.3	3367.3
	55–64	536		900.8*	1051.1	83.6	180.7	346.6	615.7	1115.8	1900.3	3375.9
	65–74	104		800.9*	819.3	100.7	151.5	275.1	580.9	1024.3	1557.8	4201.8
LF (ms <sup>2</sup> )	35–44	982	.0000	594.8	582.3	52.2	121.3	222.8	423.8	738.5	1255.2	2194.8
	45–54	1,252		487.7**	538.7	36.7	83.5	153.1	311.3	593.8	1080.3	2069.6
	55–64	536		387.1**	525.5	21.5	54.5	111.8	216.1	429.5	926.2	1733.3
	65–74	104		319.8**	452.2	7.9	34.2	89.4	164.3	318.3	726.0	2314.3
HF (ms <sup>2</sup> )	35–44	982	.0000	575.5	667.6	41.4	94.0	182.7	343.6	700.4	1328.5	2637.4
	45–54	1,252		410.8**	491.6	23.7	61.1	117.3	257.5	495.7	930.5	1823.6
	55–64	536		325.9**	487.3	27.3	54.7	103.5	201.1	373.0	673.8	1510.3
	65–74	104		383.3**	684.2	13.3	35.8	67.8	149.6	358.5	949.1	3439.6
LF (n.u.)	35–44	982	.0106	50.8	19.9	13.7	25.3	35.1	50.7	66.0	76.6	86.8
	45–54	1,252		52.3	20.3	13.5	23.7	37.4	53.3	68.0	78.6	87.7
	55–64	536		49.6	20.0	13.1	23.1	33.9	49.4	65.0	75.9	86.4
	65–74	104		47.5	21.8	8.2	17.8	31.4	46.4	64.3	78.6	90.3
HF (n.u.)	35–44	982	.0482	45.1	19.2	10.1	20.3	30.3	44.9	60.3	70.7	80.9
	45–54	1,252		42.9**	19.2	10.0	18.1	28.2	41.9	57.0	70.4	80.6
	55–64	536		44.3	18.0	11.7	20.9	30.1	43.9	58.2	67.9	79.6
	65–74	104		45.1	20.3	8.1	17.6	28.0	46.1	60.8	72.3	81.2
LF/HF	35–44	982	.1685	1.86	2.42	0.17	0.37	0.58	1.12	2.17	3.74	8.13
	45–54	1,252		2.01	2.48	0.18	0.35	0.66	1.24	2.40	4.23	8.66
	55–64	536		1.74	2.18	0.19	0.34	0.59	1.08	2.09	3.48	7.39
	65–74	104		1.91	2.60	0.14	0.28	0.52	1.03	2.39	4.46	11.73
Ln LF (ms <sup>2</sup> )	35–44	982	.0000	5.99	0.94	3.96	4.80	5.41	6.05	6.60	7.14	7.69
	45–54	1,252		5.71**	1.05	3.61	4.43	5.04	5.74	6.39	6.99	7.64
	55–64	536		5.38**	1.10	3.07	4.00	4.72	5.38	6.06	6.83	7.46
	65–74	104		5.11**	1.26	2.01	3.53	4.49	5.10	5.76	6.58	7.74
Ln HF (ms <sup>2</sup> )	35–44	982	.0000	5.86	1.03	3.72	4.54	5.21	5.84	6.55	7.19	7.88
	45–54	1,252		5.48**	1.08	3.17	4.12	4.77	5.55	6.20	6.84	7.51
	55–64	536		5.27**	1.02	3.31	4.00	4.64	5.30	5.92	6.51	7.32
	65–74	104		5.07**	1.29	2.58	3.58	4.22	5.01	5.88	6.85	8.14

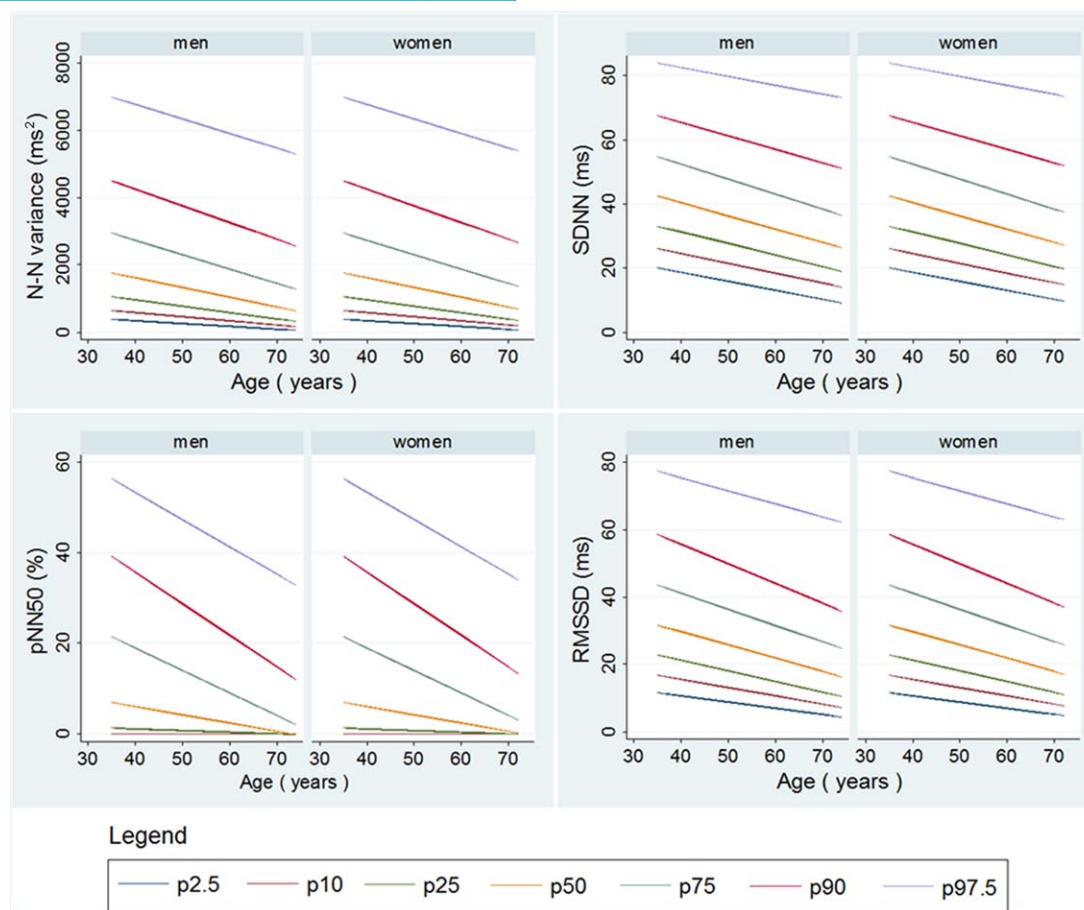
Note. Comparisons between means performed by one-way ANOVA followed by Fisher's LSD multiple comparisons. Significant differences against 35–44 age range.

\* $p < .05$ . \*\* $p < .01$ .

epidemiology, the hitherto lack of valid, short-term reference values is surprising. Therefore, our research will provide an important resource to researchers and clinicians as well as those involved in the quantified-self community. Our study also provides an important resource for personalized health care planning considering previously reported relationships between HRV and health behaviors, capacity to regulate emotion, cognitive flexibility, social functioning, mental and

physical health, and future mortality (Kemp, Arias, & Fisher, 2017; Kemp, Koenig, & Thayer, 2017). To our knowledge, the present study is the first to provide reference values for short-term resting-state HRV measurements in a large, healthy, and medication-free sample.

Reference values of HRV presented here were obtained from a large cohort study of the Brazilian population. HRV indices vary greatly among subjects as we demonstrate here.



**FIGURE 2** Percentile curves of time domain HRV indices by sex as a function of age.  $N = 2,874$

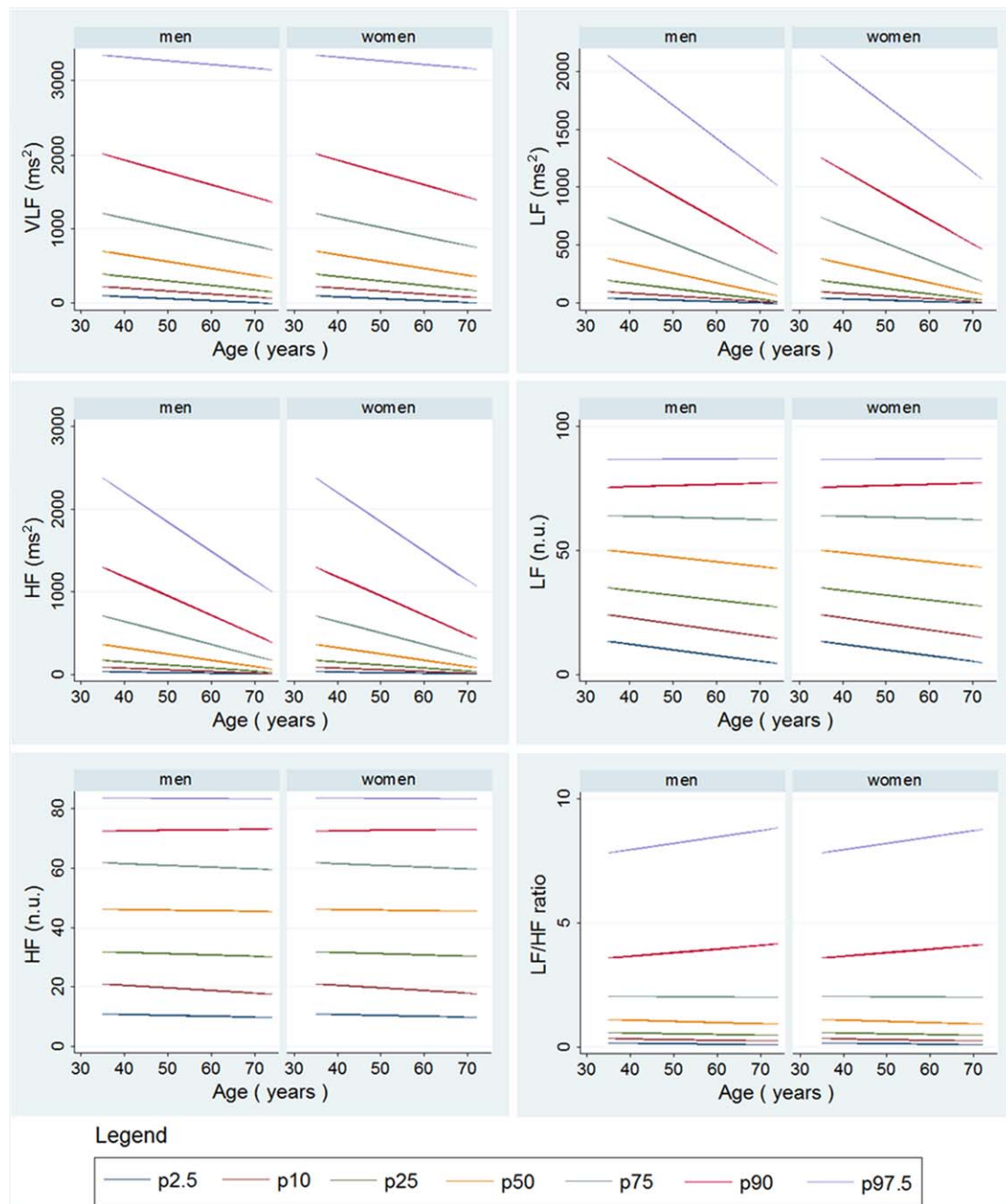
Autonomic modulation is age related: a major age-related change occurs primarily as a reduction in vagal modulation, while sympathetic modulation decreases at a slower rate (Antelmi et al., 2004; Lipsitz, Mietus, Moody, & Goldberger, 1990). Results of the present work are consistent with these earlier findings: almost all HRV variables were shown to decrease with age. Moreover, when the analyses were performed by sex, it was noticed that men show higher variance (mathematically equal to total power), LF (absolute and normalized), and lower HF (mainly the normalized) than women. Identical results were previously reported (Koenig & Thayer, 2016). The possibility that the values established in this study are consistent with healthy individuals from other regions such as North America, Europe, or Asia cannot be dismissed, and we eagerly await the publication of reference values from other large cohort studies around the world. However, it is important to reinforce the fact that many variables may impact on such comparisons.

Take, for example, ethnic factors, which exert considerable effects on HRV, as we have shown before (Kemp, Koenig et al., 2016). We have demonstrated previously that Black and Brown individuals display higher HRV than White, findings similar to those reported from North American samples (Hill et al., 2015). Extending beyond between-

groups differences, we further hypothesized (Kemp, Koenig et al., 2016) that Black individuals may suffer repeated experience of discrimination, and that this discrimination might mediate the relationship between race and HRV—this hypothesis was supported. We concluded that higher HRV in Black and Brown individuals may reflect a sustained compensatory psychophysiological response to the adverse effects of discrimination.

While chronic reductions in a broad range of HRV indices in comparison to healthy controls—reference values for which we present here—are usually considered to reflect ill health, there has been controversy over the interpretation of the LF component. Historically, the LF component has been considered as a marker of sympathetic nervous system function (Malliani et al., 1991; Montano et al., 1994; Rimoldi et al., 1990), especially when it was evaluated in normalized units, while others interpreted it as an unspecific index of sympathetic and parasympathetic activity (Akselrod et al., 1981; Berntson et al., 1997; Task Force, 1996). More recently, others have argued that this component, as with other HRV indices, predominantly reflects parasympathetic nervous system activity (Reyes del Paso et al., 2013). We have provided reference values for both of these variables, as new insights may emerge in future work.





**FIGURE 3** Percentile curves of frequency domain HRV indices by sex as a function of age.  $N = 2,874$ . Indices were determined by the AR method

Our study has a number of strengths. First, our study provides reference values for men and women in a large sample of participants, once all potential confounding was addressed. Participants were removed from analysis if they presented with hypertension (Schroeder et al., 2003), diabetes (May & Arildsen, 2011), coronary heart disease and myocardial infarction (Buccelletti et al., 2009), obesity (Karason, Molgaard, Wikstrand, & Sjostrom, 1999), kidney disease (Furuland, Linde, Englund, & Wikstrom, 2008), and stroke (Graff et al., 2013). Participants on medications including angiotensin-converting enzyme inhibitors, antiarrhythmics, beta blockers, diuretics and sympathetic agonists (Felber et al., 2006), and antidepressants (Kemp et al., 2010, 2014)

were also excluded from the present analysis. Therefore, analyses were conducted on a robust sample of healthy adults allowing for reference values by sex and age across four decades to be determined.

Second, we provide reference values based on two different methods, the FFT and the AR model, as described in international guidelines (Task Force, 1996). While FFT has been widely used due to algorithmic simplicity, the AR method has smoother spectral components and provides a more precise estimation of power spectral density even when short records are used (Task Force, 1996). Although there is a need to verify the complexity of the chosen model (i.e., the order of the model), there is evidence (Dantas et al., 2012)

demonstrating that model orders from 9 to 25 produce similar results. According to the Task Force (1996), results of FFT analyses are comparable to those of AR (Task Force, 1996), although more recent work is characterized by disagreement in adult populations (Silva et al., 2009) and even in 5-month-old infants (Poliakova et al., 2014). There are some possible explanations for these discrepancies including preprocessing steps, such as interpolation and de-trending, as well as the processing itself. Firstly, according to a previous study (Poliakova et al., 2014), AR modeling is based on use of the most significant peaks, whereas FFT tends to include all the components within a frequency band, and therefore the tails of neighboring components might be assigned into different bands by the FFT and AR approach. Moreover, discriminative capacity of FFT has been shown to be dependent on the record duration (Poliakova et al., 2014; Task Force, 1996). Thus, the AR method may provide a better resolution when short data frames are used (Kay & Marple, 1981; Poliakova et al., 2014; Task Force, 1996), and this may explain the small differences that we observed here between AR and FFT results.

Our study also has a number of limitations. First, the ECG evaluations on our participants were performed by using a digital ECG with 250 Hz of sampling rate. When the study was started in 2008, the technical specification of the used device was based on recommendations of international guidelines (Task Force, 1996). Although devices with higher sampling rates (>1000 Hz) are available today, data from recent studies (Ellis, Zhu, Koenig, Thayer, & Wang, 2015; Mahdiani, Jeyhani, Peltokangas, & Vehkaoja, 2015) have shown that ECGs with lower sampling rate (<100 Hz) might even be used without affecting HRV indices significantly. Second, we did not collect data on respiration rate or tidal volume, which may influence estimates of HRV. Thus, if we had performed the breathing control, spectral results, mainly the HF power (Driscoll & Dicicco, 2000), could have been quite different to those shown here. Nevertheless, we suggest that spontaneous respiration conditions are more comparable to clinical settings. Therefore, the reference values generated in our study are likely to be similar to those obtained in the clinical settings allowing better comparison with patient groups. We further note that the RMSSD, unlike frequency-based measures, may actually be more robust to changes in breathing patterns (Penttilä et al., 2001). Third, it is known that N-N variance and its derived indices (e.g. SDNN, which is the square root of the N-N variance) depend on the length of the recording, increasing sensitively in longer records (Task Force, 1996). For this reason, these HRV indices should only be compared with recordings of identical length (5-min duration). Finally, the present work focuses on linear HRV indices only. Therefore, it will be necessary to describe nonharmonic and nonlinear components in future work once the underlying mechanisms are better understood.

In conclusion, we provide—for the first time—reference values along with percentile curves for major short-term resting-state HRV indices across age and sex in a large, healthy, and medication-free population allowing for unbiased comparisons in future work. These data provide a foundation for reliable interpretation of HRV collected in research and clinical settings, and may also be of interest to those in the quantified-self community. There is now a need for longitudinal population-based studies to identify HRV thresholds for different age groups beyond which adverse downstream psychological and physiological sequelae may arise.

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## REFERENCES

- Akselrod, S., Gordon, D., Ubel, F. A., Shannon, D. C., Berger, A. C., & Cohen, R. J. (1981). Power spectrum analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science*, 213, 220–222. <https://doi.org/10.1126/science.6166045>
- Antelmi, I., de Paula, R. S., Shinzato, A. R., Peres, C. A., Mansur, A. J., & Grupi, C. J. (2004). Influence of age, gender, body mass index, and functional capacity on heart rate variability in a cohort of subjects without heart disease. *The American Journal of Cardiology*, 93, 381–385. <https://doi.org/10.1016/j.amjcard.2003.09.065>
- Aquino, E. M., Barreto, S. M., Bensenor, I. M., Carvalho, M. S., Chor, D., Duncan, B. B., ... Szklo, M. (2012). Brazilian Longitudinal Study of Adult Health (ELSA-Brasil): objectives and design. *American Journal of Epidemiology*, 175, 315–324. <https://doi.org/10.1093/aje/kwr294>
- Bauer, A., Camm, A. J., Cerutti, S., Guzik, P., Huikuri, H., Lombardi, F., ... Yamamoto, Y. (2017). Reference values of heart rate variability. *Heart Rhythm*, 14, 302–303. <https://doi.org/10.1016/j.hrthm.2016.12.015>
- Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., ... Van der Molen, M. W. (1997).

- Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, 34, 623–648. <https://doi.org/10.1111/j.1469-8986.1997.tb02140.x>
- Brunoni, A. R., Kemp, A. H., Dantas, E. M., Goulart, A. C., Nunes, M. A., Boggio, P. S., ... Bensenor, I. M. (2013). Heart rate variability is a trait marker of major depressive disorder: Evidence from the sertraline vs. electric current therapy to treat depression clinical study. *International Journal of Neuropsychopharmacology*, 16, 1937–1949. <https://doi.org/10.1017/S1461145713000497>
- Buccelletti, E., Gilardi, E., Scaini, E., Galiuto, L., Persiani, R., Biondi, A., ... Silveri, N. G. (2009). Heart rate variability and myocardial infarction: Systematic literature review and metanalysis. *European Review for Medical and Pharmacological Sciences*, 13, 299–307.
- Dantas, E. M., Andreao, R. V., da Silva, V. J., Ribeiro, A. L., Kemp, A. H., Brunoni, A. R., ... Mill, J. G. (2015). Comparison between symbolic and spectral analyses of short-term heart rate variability in a subsample of the ELSA-Brasil study. *Physiological Measurement*, 36, 2119–2134. <https://doi.org/10.1088/0967-3334/36/10/2119>
- Dantas, E. M., Sant'Anna, M. L., Andreao, R. V., Goncalves, C. P., Morra, E. A., Baldo, ... Mill, J. G. (2012). Spectral analysis of heart rate variability with the autoregressive method: What model order to choose? *Computers in Biology and Medicine*, 42, 164–170. <https://doi.org/10.1016/j.combiomed.2011.11.004>
- Driscoll, D. & Diccio, G. (2000). The effects of metronome breathing on the variability of autonomic activity measurements. *Journal of Manipulative Physiological Therapeutics*, 23, 610–614. <https://doi.org/10.1067/mmt.2000.110944>
- Ellis, R. J., Zhu, B., Koenig, J., Thayer, J. F., & Wang, Y. (2015). A careful look at ECG sampling frequency and R-peak interpolation on short-term measures of heart rate variability. *Physiological Measurement*, 36, 1827–1852. <https://doi.org/10.1088/0967-3334/36/9/1827>
- Felber, D. D., Schindler, C., Schwartz, J., Barthelemy, J. C., Tschopp, J. M., Roche, F., ... Ackermann-Liebrich, U. (2006). Heart rate variability in an ageing population and its association with lifestyle and cardiovascular risk factors: Results of the SAPALDIA study. *Europace*, 8, 521–529. <https://doi.org/10.1093/europace/eul063>
- Furuland, H., Linde, T., Englund, A., & Wikstrom, B. (2008). Heart rate variability is decreased in chronic kidney disease but may improve with hemoglobin normalization. *Journal of Nephrology*, 21, 45–52.
- Graff, B., Gasecki, D., Rojek, A., Boutouyrie, P., Nyka, W., Laurent, S., & Narkiewicz, K. (2013). Heart rate variability and functional outcome in ischemic stroke: A multiparameter approach. *Journal of Hypertension*, 31, 1629–1636. <https://doi.org/10.1097/HJH.0b013e328361e48b>
- Hill, L. K., Hu, D. D., Koenig, J., Sollers, J. J., III, Kapuku, G., Wang, X., ... Thayer, J. F. (2015). Ethnic differences in resting heart rate variability: A systematic review and meta-analysis. *Psychosomatic Medicine*, 77, 16–25. <https://doi.org/10.1097/PSY.0000000000000133>
- Hillebrand, S., Gast, K. B., de Mutsert, R., Swenne, C. A., Jukema, J. W., Middeldorp, S., Rosendaal, F. R., & Dekkers, O. M. (2013). Heart rate variability and first cardiovascular event in populations without known cardiovascular disease: Meta-analysis and dose-response meta-regression. *Europace*, 15, 742–749. <https://doi.org/10.1093/europace/eus341>
- Jandackova, V. K., Britton, A., Malik, M., & Steptoe, A. (2016). Heart rate variability and depressive symptoms: A cross-lagged analysis over a 10-year period in the Whitehall II study. *Psychological Medicine*, 46, 2121–2131. <https://doi.org/10.1017/S003329171600060X>
- Karason, K., Molgaard, H., Wikstrand, J., & Sjostrom, L. (1999). Heart rate variability in obesity and the effect of weight loss. *The American Journal of Cardiology*, 83, 1242–1247. [https://doi.org/10.1016/S0002-9149\(99\)00066-1](https://doi.org/10.1016/S0002-9149(99)00066-1)
- Kay, S. M. & Marple, S. L. (1981). Spectrum analysis—A modern perspective. *Proceedings of the IEEE*, 69, 1380–1419. <https://doi.org/10.1109/PROC.1981.12184>
- Kemp, A. H., Arias, J., & Fisher, Z. (2017). Social ties, health and wellbeing: A literature review and model. *Neuroscience and Social Science*, 397–427. [https://doi.org/10.1007/978-3-319-68421-5\\_17](https://doi.org/10.1007/978-3-319-68421-5_17)
- Kemp, A. H., Brunoni, A. R., Santos, I. S., Nunes, M. A., Dantas, E. M., de Carvalho, F. R., ... Lotufo, P. A. (2014). Effects of depression, anxiety, comorbidity, and antidepressants on resting-state heart rate and its variability: An ELSA-Brasil cohort baseline study. *The American Journal of Psychiatry*, 171, 1328–1334. <https://doi.org/10.1176/appi.ajp.2014.13121605>
- Kemp, A. H., Koenig, J., & Thayer, J. F. (2017). From psychological moments to mortality: A multidisciplinary synthesis on heart rate variability spanning the continuum of time. *Neuroscience and Biobehavioral Reviews*, 83, 547–567. <https://doi.org/10.1016/j.neubiorev.2017.09.006>
- Kemp, A. H., Koenig, J., Thayer, J. F., Bittencourt, M. S., Pereira, A. C., Santos, I. S., ... Lotufo, P. A. (2016). Race and resting-state heart rate variability in Brazilian civil servants and the mediating effects of discrimination: An ELSA-Brasil Cohort Study. *Psychosomatic Medicine*, 78, 950–958. <https://doi.org/10.1097/PSY.0000000000000359>
- Kemp, A. H., Lopez, S. R., Passos, V. M. A., Bittencourt, M. S., Dantas, E. M., Mill, J. G., ... Lotufo, P. A. (2016). Insulin resistance and carotid intima-media thickness mediate the association between resting-state heart rate variability and executive function: A path modelling study. *Biological Psychology*, 117, 216–224. <https://doi.org/10.1016/j.biopsycho.2016.04.006>
- Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. *Biological Psychiatry*, 67, 1067–1074. <https://doi.org/10.1016/j.biopsycho.2009.12.012>
- Kemp, A. H., Quintana, D. S., Kuhnert, R. L., Griffiths, K., Hickie, I. B., & Guastella, A. J. (2012). Oxytocin increases heart rate variability in humans at rest: Implications for social approach-related motivation and capacity for social engagement. *PLOS One*, 7, e44014. <https://doi.org/10.1371/journal.pone.0044014>
- Koenig, J., Rash, J. A., Campbell, T. S., Thayer, J. F., & Kaess, M. (2017). A meta-analysis on sex differences in resting-state vagal activity in children and adolescents. *Frontiers in Physiology*, 8, 582. <https://doi.org/10.3389/fphys.2017.00582>



- Koenig, J. & Thayer, J. F. (2016). Sex differences in healthy human heart rate variability: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, 64, 288–310. <https://doi.org/10.1016/j.neubiorev.2016.03.007>
- Lewis, G., Pelosi, A. J., Araya, R., & Dunn, G. (1992). Measuring psychiatric disorder in the community: A standardized assessment for use by lay interviewers. *Psychological Medicine*, 22, 465–486.
- Lipsitz, L. A., Mietus, J., Moody, G. B., & Goldberger, A. L. (1990). Spectral characteristics of heart rate variability before and during postural tilt: Relations to aging and risk of syncope. *Circulation*, 81, 1803–1810. <https://doi.org/10.1161/01.CIR.81.6.1803>
- Mahdiani, S., Jeyhani, V., Peltokangas, M., & Vehkaoja, A. (2015). Is 50 Hz high enough ECG sampling frequency for accurate HRV analysis? *37th Annual International Conference of the IEEE Engineering and Medicine and Biology Society (EMBC)*, 5948–5951. <https://doi.org/10.1109/EMBC.2015.7319746>
- Malliani, A., Pagani, M., Lombardi, F., & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, 84, 482–492. <https://doi.org/10.1161/01.CIR.84.2.482>
- May, O. & Arildsen, H. (2011). Long-term predictive power of heart rate variability on all-cause mortality in the diabetic population. *Acta Diabetologica*, 48, 55–59. <https://doi.org/10.1007/s00592-010-0222-4>
- Mill, J. G., Pinto, K., Griep, R. H., Goulart, A., Foppa, M., Lotufo, P. A., ... Bensenor, I. M. (2013). Medical assessments and measurements in ELSA-Brasil. *Revista de Saude Publica*, 47(Suppl 2), 54–62. <https://doi.org/10.1590/S0034-8910.2013047003851>
- Montano, N., Ruscone, T. G., Porta, A., Lombardi, F., Pagani, M., & Malliani, A. (1994). Power spectrum analysis of heart rate variability to assess the changes in sympathovagal balance during graded orthostatic tilt. *Circulation*, 90, 1826–1831. <https://doi.org/10.1161/01.CIR.90.4.1826>
- Nunan, D., Sandercock, G. R., & Brodie, D. A. (2010). A quantitative systematic review of normal values for short-term heart rate variability in healthy adults. *Pacing and Clinical Electrophysiology*, 33, 1407–1417. <https://doi.org/10.1111/j.1540-8159.2010.02841.x>
- Penttilä, J., Helminen, A., Jartti, T., Kuusela, T., Huikuri, H. V., Tulppo, M. P., ... Scheinin, H. (2001). Time domain, geometrical and frequency domain analysis of cardiac vagal outflow: Effects of various respiratory patterns. *Clinical Physiology and Functional Imaging*, 21, 365–376. <https://doi.org/10.1046/j.1365-2281.2001.00337.x>
- Poliakova, N., Dionne, G., Dubreuil, E., Ditto, B., Pihl, R. O., Perusse, D., ... Boivin, M. (2014). A methodological comparison of the Porges algorithm, fast Fourier transform, and autoregressive spectral analysis for the estimation of heart rate variability in 5-month-old infants. *Psychophysiology*, 51, 579–583. <https://doi.org/10.1111/psyp.12194>
- Reyes del Paso, G. A., Langewitz, W., Mulder, L. J., van Roon, A., & Duschek, S. (2013). The utility of low frequency heart rate variability as an index of sympathetic cardiac tone: A review with emphasis on a reanalysis of previous studies. *Psychophysiology*, 50, 477–487. <https://doi.org/10.1111/psyp.12027>
- Rimoldi, O., Pierini, S., Ferrari, A., Cerutti, S., Pagani, M., & Malliani, A. (1990). Analysis of short-term oscillations of R-R and arterial pressure in conscious dogs. *The American Journal of Physiology*, 258, H967–H976.
- Sammito, S. & Böckelmann, I. (2016). Reference values for time- and frequency-domain heart rate variability measures. *Heart Rhythm*, 13, 1309–1316. <https://doi.org/10.1016/j.hrthm.2016.02.006>
- Sammito, S. & Böckelmann, I. (2017). New reference values of heart rate variability during ordinary daily activity. *Heart Rhythm*, 14, 304–307. <https://doi.org/10.1016/j.hrthm.2016.12.016>
- Schroeder, E. B., Liao, D., Chambless, L. E., Prineas, R. J., Evans, G. W., & Heiss, G. (2003). Hypertension, blood pressure, and heart rate variability: The Atherosclerosis Risk in Communities (ARIC) study. *Hypertension*, 42, 1106–1111. <https://doi.org/10.1161/01.HYP.0000100444.71069.73>
- Silva, G. J., Ushizima, M. R., Lessa, P. S., Cardoso, L., Drager, L. F., Atala, M. M., ... Krieger, E. M. (2009). Critical analysis of autoregressive and fast Fourier transform markers of cardiovascular variability in rats and humans. *Brazilian Journal of Medical and Biological Research*, 42, 386–396. <https://doi.org/10.1590/S0100-879X2009000400012>
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (Task Force). (1996). Heart rate variability: Standards of measurement, physiological interpretation and clinical use. *Circulation*, 93, 1043–1065. <https://doi.org/10.1161/01.CIR.93.5.1043>
- Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine*, 37, 141–153. <https://doi.org/10.1007/s12160-009-9101-z>
- Williams, D. P., Cash, C., Rankin, C., Bernardi, A., Koenig, J., & Thayer, J. F. (2015). Resting heart rate variability predicts self-reported difficulties in emotion regulation: A focus on different facets of emotion regulation. *Frontiers in Psychology*, 6, 261. <https://doi.org/10.3389/fpsyg.2015.00261>

## SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

### Table S1–S12

### Figure S1

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