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Dynamic Changes in Heart Rate and Heart Rate Variability during Slow Breathing Paced at 0.1 Hz: A Second-by-Second Analysis

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Abstract

Heart rate (HR) and heart rate variability (HRV) are key indicators of autonomic regulation. Breathing paced at approximately 0.1 Hz is the basis of HRV biofeedback and resonance paced breathing, interventions with empirical support for improving health. This study characterized second-by-second cardiovascular responses to 0.1 Hz paced breathing (PB). Ninety-one young adults (58 women; M age = 20.8 ± 2.3 years) completed consecutive 5-minute episodes of natural breathing (NB) and 0.1 Hz PB while the electrocardiogram was recorded; all showed a dominant HRV spectral peak at 0.099–0.104 Hz during paced breathing. HR, R-R interval (RRI), the standard deviation of normal-to-normal intervals (SDNN), the root mean square of successive differences (RMSSD), and low-frequency (LF) and high-frequency (HF) HRV power were analyzed in rolling 60-second windows. Linear mixed-effects models showed higher SDNN, RMSSD, and LF power and, initially, lower HR and longer RRI during PB than NB. The PB advantage in SDNN and RMSSD diminished over the episode, and HR rose and RRI fell more steeply during PB. Generalized additive mixed models showed that SDNN, RMSSD, and LF differed between conditions throughout the episode, with the largest effects in the first minute and a slow decline thereafter; HR was lower during PB early and higher after about 3 minutes. Individuals differed in trajectory shape. Women tended to have lower LF power, but no moderation of the breathing response by body mass index, gender, or alcohol use severity survived correction for multiple comparisons.

Trial registration: ClinicalTrials.gov NCT05369169, registered 16 March 2022.

Introduction

The autonomic nervous system (ANS) plays a central role in regulating cardiovascular, respiratory, and emotional processes (Bates et al., 2026), making its accurate assessment essential for both clinical and experimental contexts. Among the commonly used physiological markers, heart rate variability (HRV), the beat-to-beat fluctuation in heart periods, captures the dynamic modulation of autonomic inputs, particularly vagal activity, and thus offers a nuanced indicator of cardiovascular control. This dynamic variability reflects underlying neurocardiac integration and serves as a key marker of the system's adaptive capacity under both resting and perturbed conditions (Bates et al., 2023; Fields et al., 2025).

Growing evidence shows that autonomic and central nervous system activity can be intentionally modulated through behavioral strategies. Among these, breathing-based interventions are recognized as effective, non-invasive approaches to regulate ANS function. Breathing paced near a resonance frequency of the cardiovascular system (usually between 4.5 and 6.5 breaths per minute, averaging a little slower than six breaths per minute, or about 0.1 Hz) maximally couples heart rate and blood pressure through the baroreflex and has been shown to enhance baroreflex sensitivity, increase vagal tone, and promote cardiorespiratory synchronization (Bates et al., 2022; Lehrer et al., 2020; Lehrer & Gevirtz, 2014; Lehrer et al., 2003; Vaschillo et al., 2002; Vaschillo et al., 2006). This slow, rhythmic pattern optimizes respiratory sinus arrhythmia and amplifies HRV oscillations, reflecting greater vagally mediated HRV (Balaji et al., 2025). Resonance paced breathing has demonstrated consistent benefits for

autonomic regulation and psychological well-being across healthy and clinical populations (Chaitanya et al., 2022; Shaffer & Meehan, 2020). HRV is increasingly recognized as a sensitive marker of autonomic modulation, especially parasympathetic influence (Besson et al., 2025; Penfold et al., 2025).

Most prior studies of resonance breathing averaged HRV changes over multi-minute windows, inherently assuming a stationary signal within each period (Chaitanya et al., 2022; Steffen et al., 2017). These studies have used standard HRV indices that aggregate data across episode durations of minutes (Quigley et al., 2024), rather than characterizing second-to-second fluctuations in HRV during the episode that may potentially provide new insights into the dynamics of physiological responses during paced breathing. Such approaches may obscure more rapid autonomic fluctuations during breathing episodes and mask how quickly the response is established and how it changes over the episode. Characterizing moment-to-moment fluctuations may potentially advance real-time monitoring, improve intervention tailoring, and may aid early detection of dysregulation in clinical populations, while also guiding future research on the stationarity of HRV signals during resonance breathing.

The present study aimed to meet the need for high-resolution approaches that capture the rapid, second-by-second dynamics of autonomic activity during 0.1 Hz paced breathing. We characterized the moment-to-moment trajectories of both heart rate and heart rate variability indices across time, providing a detailed depiction of how autonomic responses unfold within a 5-minute episode of natural breathing compared to a 5-minute episode of slow breathing paced at 0.1 Hz. We further examined how quickly the response to 0.1 Hz PB is established and how it changes over the episode, and whether 60-second windows yield estimates consistent with longer analysis windows, thereby informing the methodological assumptions underlying dynamic HRV assessment. A secondary, exploratory aim was to examine inter-individual variability in these responses, including whether BMI, age, gender, and alcohol use moderate them. Participants were young adults with recent binge drinking, a group rarely considered in respiratory intervention research, even though heavier alcohol use has been linked to reduced resting HRV and autonomic dysregulation (Eddie et al., 2022; Quintana et al., 2013).

Methods

Participants

This study used data from an investigation of afferent neurocardiac signals, cue reactivity, and cognitive control in college students (ClinicalTrials.gov NCT05369169). This is a secondary analysis of data from this registered study (registered 16 March 2022): only the resonance-breathing session is analyzed here, comprising 5 minutes of natural breathing followed by 5 minutes of 0.1 Hz paced breathing. A total of 103 young adults (aged 18–35 years) were enrolled from a university campus and surrounding community via flyers, electronic advertisements, and word-of-mouth. Inclusion criteria required participants to report normal or corrected-to-normal vision and two or more binge drinking episodes in the past month (defined as ≥ 4 drinks for women or ≥ 5 drinks for men within a 2-hour period). Exclusion criteria included self-reported history of major psychiatric or neurological disorders, cardiovascular

disease, head injury with loss of consciousness, or recent illicit drug use other than cannabis. Of these, no ECG file was available for 10 participants; one participant was excluded by the researcher because of numerous artifacts and slow heart rate; and one participant was excluded because the dominant frequency of the paced-breathing recording was 0.2 Hz rather than 0.1 Hz. The final analytic sample consisted of 91 participants (58 women and 33 men; Table 1). The study was approved by the Rutgers University Institutional Review Board for the Protection of Human Subjects (Protocol Pro2021001073), and participants provided written informed consent.

Measures

Alcohol use severity was measured with the Alcohol Use Disorders Identification Test (AUDIT; (Saunders et al., 1993), administered before the breathing conditions. The AUDIT is a 10-item screening instrument for hazardous and harmful alcohol consumption, with total scores from 0 to 40, that demonstrates high reliability and validity across diverse populations; scores ≥ 8 indicate hazardous drinking (Babor et al., 2001). The AUDIT was selected as the sole alcohol moderator because it is brief, widely validated, and provides a continuous index of alcohol-related risk suited to modeling variation within a binge-drinking sample. Additional alcohol-related measures (craving, alcohol sensitivity, and alcohol-related problems) were collected in the parent study.

Study Design and Procedure

A within-subjects, fixed-order design was used to examine cardiac autonomic responses during natural and 0.1 Hz PB. Each participant completed two breathing conditions consecutively in a single laboratory session, beginning with a 5-minute natural breathing (NB) period followed by a 5-minute period of 0.1 Hz PB. During NB, participants sat quietly and breathed spontaneously without external cues, with their eyes open for the first half (about 2.5 minutes) and closed for the second half. During the 0.1 Hz PB condition, participants followed a visual pacer (E-Z Air, Thought Technology Ltd.) to synchronize their breathing at a fixed rate of 0.1 Hz (6 breaths per minute), including 5 seconds of inhalation, and 5 seconds of exhalation with no gap between. This fixed order was selected to avoid potential carryover effects of paced breathing, which may produce prolonged autonomic modulation and confound subsequent baseline recordings. Participants were seated in a quiet, temperature-controlled laboratory. A standard three-lead electrocardiogram (ECG) configuration was used to continuously record cardiac activity at a sampling rate of 1,000 Hz (MP160 system; Biopac Systems, Goleta, CA).

ECG Signal Processing and HRV Analysis

ECG recordings were available for 93 of the 103 enrolled participants (10 had no ECG file). One recording was excluded because of excessive artifacts, and one because the HRV spectrum showed no sharp peak at 0.1 Hz during paced breathing (the peak was at 0.2 Hz), leaving 91 participants for analysis (58

women [64%], 33 men; age 20.8 ± 2.3 years, range 18–31; BMI 24.5 ± 4.0 kg/m²). All 91 had a dominant peak at 0.099–0.104 Hz during 0.1 Hz breathing.

R-peaks were detected and edited in MindWare BioLab software (versions 3.4–3.6) with 40–200 bpm limits, and the inter-beat interval (IBI) series were exported. Gaps (IBI > 3000 ms, indicating removed data) were closed by joining the adjacent segments without filling. IBIs were then screened for out-of-range values (< 300 or > 1500 ms) and for sudden jumps using the MED/MAD criterion (Berntson et al., 1990), computed separately for each participant and condition so that the larger respiratory swings during paced breathing were not falsely flagged. Flagged beats were reviewed individually: 15 were removed (nine in NB, six in PB; 14 were replaced by cubic-spline values from the 10 beats on each side, and one split beat was merged into a single interval), and three flagged beats were kept. The first and last (partial) intervals of each segment were removed, and analyses used the first 300 s of each recording (t = 0 at the first R-peak). Moment-to-moment HRV was computed in 60-s windows advanced in 1-s steps, and each window's value was assigned to the window midpoint (30–270 s; for example, the window spanning 0–60 s is plotted at 30 s). Within each window, time-domain indices were computed from the IBIs lying fully inside the window (mean HR = 60,000/mean IBI; SDNN; RMSSD). RRI was the mean inter-beat interval in each 60-s window (equal to 60,000/mean HR). For the frequency domain, IBIs were placed on their beat times, resampled at 4 Hz with a cubic spline, linearly detrended, and analyzed with a Hann-windowed periodogram. LF (0.04–0.15 Hz) and HF (0.15–0.40 Hz) power (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996) were calculated as the area under the spectrum (ms²) and natural-log transformed. For modeling, per-second values were summarized as the median within 5-s bins (60 bins per participant and condition). Adherence to the pacer was screened from the HRV spectrum: a dominant spectral peak near 0.1 Hz during paced breathing was required for inclusion, and, for descriptive purposes, the dominant frequency of each 60-s window was also determined and a second was counted as "at 0.1 Hz" if it fell within 0.09–0.11 Hz. Because this was an inclusion criterion, we regard it as a practical screening criterion indicating that heart rate oscillations occurred at the target frequency, not as independent verification of breathing. Indices matched MindWare's own output in the 75 participants without manual edits (r ≥ .998 for time-domain indices; LF r = .995; HF r = .965). The impedance cardiography (ICG) respiration signal was recorded but was too noisy to yield usable respiratory frequency or amplitude, so no respiratory measures were derived.

Statistical Approach

Participant exclusions are described under ECG Signal Processing and HRV Analysis; the final analytic sample comprised 91 participants. Descriptive statistics are reported as mean ± SD.

Primary analysis. To characterize the moment-to-moment trajectory of each outcome (HR, RRI, SDNN, RMSSD, LF, and HF), generalized additive mixed models (GAMMs) were fit using the mgcv package (bam), with a separate smooth function of time (thin-plate regression splines, basis dimension k = 15) for each condition, a fixed effect of condition, a random intercept and a random condition effect for each

participant, and first-order autoregressive (AR(1)) errors within each participant-by-condition series to account for serial correlation between neighboring time points (residual lag-1 autocorrelation ≈ 0.9 for the windowed indices). Models were estimated by restricted maximum likelihood (REML), and adequacy of the basis dimension was checked with k.check. Non-linearity within each condition was assessed by comparing a smooth with a linear time term using AIC (both fitted by maximum likelihood with AR(1) errors). Significant divergence between conditions over time was assessed by computing the 0.1 Hz PB − NB difference in fitted values at 1-second intervals, together with 95% confidence intervals derived from the model's coefficient covariance matrix; time windows in which this interval excluded zero were classified as showing a significant condition difference. The time to approach the maximal response was defined as the first time at which the fitted difference of its maximum absolute value. To examine whether individuals differed in trajectory shape, models with participant-specific smooth curves (LF and SDNN during 0.1 Hz PB) were compared by AIC with models allowing participants to differ only in level and linear trend.

Secondary analysis. Linear mixed-effects models (LMMs) tested the effects of condition (NB as the reference level), time (continuous, in seconds, with Time = 0 at the start of the episode), and their interaction on each outcome. Models included a random intercept and random slopes for Time, Condition, and Time × Condition for each participant (correlated random effects), so that effects varying within participants were tested against between-participant variability (Satterthwaite df ≈ 81–90). Time was rescaled to 100-second units during estimation for numerical stability; coefficients are reported per second. To test moderation, a second set of models added BMI, age, gender (male as the reference level), and AUDIT total score, together with their two- and three-way interactions with condition and time (df ≈ 77–86); BMI, age, and AUDIT scores were mean-centered. Moderator models included all 91 participants (the two participants who reported their gender as "Other" were classified by sex from laboratory records, and BMI values were verified against laboratory records). Moderator results are reported for HR, SDNN, RMSSD, LF, and HF. As a check that did not depend on the within-participant dependence structure, each participant's mean 0.1 Hz PB − NB difference and its slope over time were regressed on the moderators. Parameters were estimated via REML, and fixed-effect tests used Satterthwaite degrees of freedom. Standardized effect sizes were calculated as $d = 2t/\sqrt{df}$. P-values were corrected for multiple comparisons using the Benjamini-Hochberg false discovery rate (FDR) procedure across the 18 substantive tests in Table 2 and across all 85 moderator tests in Table 3 (5 outcomes × 17 terms); effects with raw $p < .05$ but FDR-adjusted $p \geq .05$ are described as exploratory. Exploratory analyses of alcohol use severity compared participants with AUDIT ≥ 8 (Babor et al., 2001) and < 8 on their mean PB − NB change (Welch t-tests; Hedges' g) and correlated AUDIT scores with each outcome in each condition (Spearman correlations). Missing windows, including the first ~ 30 s of each recording when HRV indices are not yet estimable, were handled implicitly by the mixed models (unbalanced data). All analyses were conducted in R version 4.5.1 using lme4 for model fitting, lmerTest for p-values, and mgcv for generalized additive mixed models.

Window-length agreement. To evaluate whether 60-second windows provide adequate estimates, SDNN, RMSSD, pNN50, LF, and HF (ms^2, and ln-transformed) were also computed with the same methods in (a)

60-second and (b) 120-second windows advanced in 1-second steps, averaged across each recording, and (c) in the full first 300 s of each recording. Agreement across the three window lengths was quantified separately for NB and 0.1 Hz PB with the two-way mixed-effects, consistency, average-measures intraclass correlation coefficient (ICC(3,k)), and by expressing the group mean at 60 and 120 s as a percentage of the group mean at 300 s. ICC ≥ 0.90 is conventionally “excellent” (Koo & Li, 2016).

Results

Participants’ demographic characteristics are presented in Table 1.

Table 1
General and physiological characteristics of subjects

Variable	Category / Unit	Mean ± SD / n (%)
BMI	kg/m²	24.52 ± 4.04
Age	Years	20.76 ± 2.35
Resting HR	bpm	75.88 ± 11.26
Gender	Female	58 (63.7%)
	Male	33 (36.3%)
BMI category	Underweight (< 18.5)	4 (4.4%)
	Normal (18.5–24.9)	51 (56.0%)
	Overweight (25–29.9)	26 (28.6%)
	Obese (≥ 30)	10 (11.0%)
AUDIT total score	Score (range 0–40)	6.95 ± 3.46
AUDIT risk category	Low risk (0–7)	56 (61.5%)
	Hazardous (8–15)	31 (34.1%)
	Harmful (16–19)	4 (4.4%)
	Probable dependence (≥ 20)	0 (0%)

BMI, body mass index; HR, heart rate; SD, standard deviation; AUDIT, Alcohol Use Disorders Identification Test (scores ≥ 8 indicate hazardous drinking).

Although all participants reported at least two binge-drinking episodes in the past month, most (61.5%) scored below the AUDIT hazardous-use threshold (≥ 8).

Primary analysis: moment-to-moment trajectories.

To characterize the moment-to-moment trajectory of each HRV index, we fit generalized additive mixed models (GAMMs) allowing a flexible smooth curve over time for each condition, with a random condition effect for each participant and first-order autoregressive (AR(1)) errors to account for the strong serial correlation between neighboring time points (residual lag-1 autocorrelation 0.87–0.91; Fig. 1). During 0.1 Hz PB, SDNN, RMSSD, and LF power were highest in the first minute (SDNN ≈ 105 ms, RMSSD ≈ 65 ms, LF ≈ 8.9 ln ms²) and declined slowly thereafter (to ≈ 92 ms, ≈ 55 ms, and ≈ 8.6 ln ms² by the end of the episode), a significant departure from linearity (ΔAIC = 54, 30, and 46), whereas these indices were lower and varied little during NB (ΔAIC = 41, 5, and 13). The PB − NB difference reached 90% of its maximum within the first minute for SDNN, RMSSD, and LF. HR and RRI showed the opposite time course: both changed markedly over the episode in both conditions (ΔAIC = 131 and 134 in NB; 59 and 61 in PB), with HR lower and RRI longer during PB early on, reversing later. SDNN, RMSSD, LF, and HF (lower during PB) differed significantly between conditions across the entire analyzable window (about 30–270 s), whereas HR was lower during PB from about 30–77 s and higher from about 194–270 s, and RRI was longer during PB at 30–68 s and shorter at 190–270 s. Individuals differed substantially in the shape of their trajectories (ΔAIC = 356 for LF and 662 for SDNN during PB). Values are reported from approximately 30 s to 270 s, reflecting the 60-second moving window (advanced in 1-second steps) used to compute HRV indices, which necessarily excludes the first and last ~ 30 s of each 5-minute epoch rather than reflecting a true physiological onset or offset (Fig. 1).

Secondary analyses: linear mixed-effects models and moderators.

In secondary analyses, linear mixed-effects models (Table 2) showed that SDNN, RMSSD, and LF power were markedly higher during 0.1 Hz paced breathing than during natural breathing at the start of the session (SDNN: β = 49.4 ms; RMSSD: β = 21.1 ms; LF: β = 2.29 ln ms²; all p_FDR < .001), HR was lower (β = −1.99 bpm, p_FDR = .002), and RRI was longer (β = 21.3 ms, p_FDR = .003); HF power did not differ significantly (β = −0.25, p = .040, p_FDR = .072). Over the session, the PB advantage in SDNN (Time × Condition: β = −0.047 ms/s, p_FDR = .004) and RMSSD (β = −0.046 ms/s, p_FDR < .001) diminished, while HR rose (β = 0.016 bpm/s, p_FDR < .001) and RRI fell (β = −0.193 ms/s, p_FDR < .001) more steeply during PB than NB. No Time × Condition effect was significant for LF (p_FDR = .171) or HF (p_FDR = .326), and none of the indices showed a significant linear change over time during NB (all p_FDR ≥ .127).

Table 2
Fixed Effects from Linear Mixed Models Examining the Impact of 0.1 Hz PB Versus NB on Cardiovascular and HRV Outcomes

Outcome	Term	Estimate	SE	t	p	p (FDR)	d
HR (bpm)	Baseline (NB, Time = 0)	75.25	1.171	64.29	< .001	—	—
	Time (per s)	0.0030	0.0023	1.30	.196	.271	0.278
	Condition (0.1 Hz PB vs NB)	−1.987	0.563	−3.53	< .001	.002	−0.744
	Time × Condition	0.0161	0.0032	5.06	< .001	< .001	1.067
RRI (ms)	Baseline	814.15	12.71	64.08	< .001	—	—
	Time	−0.0195	0.0234	−0.83	.407	.488	−0.177
	Condition	21.35	6.455	3.31	.001	.003	0.697
	Time × Condition	−0.1926	0.0327	−5.88	< .001	< .001	−1.240
SDNN (ms)	Baseline	53.29	2.529	21.07	< .001	—	—
	Time	0.0016	0.0117	0.14	.890	.890	0.030
	Condition	49.40	3.399	14.53	< .001	< .001	3.064
	Time × Condition	−0.0472	0.0148	−3.18	.002	.004	−0.671
RMSSD (ms)	Baseline	43.64	2.536	17.21	< .001	—	—
	Time	0.0020	0.0083	0.24	.810	.858	0.052
	Condition	21.07	2.723	7.74	< .001	< .001	1.631
	Time × Condition	−0.0457	0.0112	−4.10	< .001	< .001	−0.865
LF (ln ms²)	Baseline	6.543	0.1085	60.31	< .001	—	—
	Time	−0.00020	0.00052	−0.38	.704	.792	−0.085
	Condition	2.287	0.1031	22.19	< .001	< .001	4.682

HR = Heart Rate, RRI = R-R Interval (mean inter-beat interval in 60-s windows), SDNN = Standard Deviation of NN Intervals, RMSSD = Root Mean Square of Successive Differences, LF = Low Frequency, HF = High Frequency, NB = Natural Breathing, 0.1 Hz PB = 0.1 Hz Paced Breathing, SE = Standard Error, t = t-statistic, p (FDR) = Benjamini-Hochberg FDR-corrected p-value, Cohen's d = 2t/√df, ms = millisecond, ln = natural logarithm.

Outcome	Term	Estimate	SE	t	p	p (FDR)	d
	Time × Condition	−0.00094	0.00059	−1.60	.114	.171	−0.339
HF (ln ms²)	Baseline	6.551	0.1161	56.40	< .001	—	—
	Time	−0.00067	0.00037	−1.79	.077	.127	−0.379
	Condition	−0.2475	0.1188	−2.08	.040	.072	−0.439
	Time × Condition	−0.00057	0.00049	−1.15	.253	.326	−0.242

HR = Heart Rate, RRI = R-R Interval (mean inter-beat interval in 60-s windows), SDNN = Standard Deviation of NN Intervals, RMSSD = Root Mean Square of Successive Differences, LF = Low Frequency, HF = High Frequency, NB = Natural Breathing, 0.1 Hz PB = 0.1 Hz Paced Breathing, SE = Standard Error, t = t-statistic, p (FDR) = Benjamini-Hochberg FDR-corrected p-value, Cohen's d = 2t/√df, ms = millisecond, ln = natural logarithm.

Each model included random intercepts and random slopes for Time, Condition, and Time × Condition by participant; Satterthwaite df = 81–90. Time is in seconds. RRI = means inter-beat interval in 60-s windows. Cohen's d is computed only for substantive contrasts (not reported for the baseline, which tests against zero). FDR was applied across the 18 substantive tests (3 terms × 6 outcomes, excluding baselines). Effects with $p < .05$ but p (FDR) $\geq .05$ are considered exploratory.

To test whether individual characteristics moderated the effects of 0.1 Hz paced breathing, we extended the linear mixed models with BMI, age, gender, and AUDIT score and their interactions with condition and time (85 tests; Table 3; n = 91). Three tests were nominally significant ($p < .05$), and none survived correction for multiple comparisons: women had lower LF power ($\beta = -0.78$ ln ms², $p < .001$, $p_{FDR} = .067$) and lower SDNN ($\beta = -13.1$ ms, $p = .017$, $p_{FDR} = .480$) than men, and HR was lower in older participants ($\beta = -1.32$ bpm per year, $p = .007$, $p_{FDR} = .304$). None of the interactions of age, BMI, gender, or AUDIT score with condition or time was nominally significant. A participant-level check, regressing each participant's mean 0.1 Hz PB − NB difference and its slope over time on the moderators, gave concordant results (one of 40 tests with $p < .05$, LF Condition × Gender, $p = .009$; none after correction).

Alcohol use severity. AUDIT score did not significantly moderate the effect of 0.1 Hz paced breathing on any outcome (Table 3) and was not significantly correlated with any HR or HRV measure during NB or 0.1 Hz PB (all $|\rho| \leq .12$, $p_{FDR} \geq .85$). Comparing participants with AUDIT ≥ 8 (n = 35) and < 8 (n = 56), the change from NB to PB did not differ on any outcome (all $p \geq .063$, $|g| \leq 0.40$; for example HR: $g = -0.07$, 95% CI [− 0.49, 0.36], $p = .75$), and the groups did not differ at rest or during paced breathing (all $p \geq .16$).

Table 3
Moderator Effects on HRV Outcomes (Raw p < .05; Full Three-Way LMM; Exploratory Analyses)

Outcome	Term	Estimate	SE	t	p	p (FDR)	d
LF	Gender (F vs M)	−0.776	0.223	−3.48	< .001	.067	−0.754
HR	Age	−1.323	0.480	−2.76	.007	.304	−0.594
SDNN	Gender (F vs M)	−13.06	5.36	−2.44	.017	.480	−0.526

HR = Heart Rate, SDNN = Standard Deviation of NN Intervals, LF = Low Frequency, BMI = Body Mass Index, F = Female, M = Male, SE = Standard Error, t = t-statistic, p (FDR) = Benjamini-Hochberg FDR-corrected p-value, Cohen's d = 2t/√df, ms = millisecond, ln = natural logarithm.

Models included Time × Condition × Moderator (BMI, age, gender, AUDIT) interactions, with random intercepts and random slopes for Time, Condition, and Time × Condition by participant; Satterthwaite df = 85–86. Analyses included all 91 participants. Male was the reference level for gender; BMI, age, and AUDIT were mean-centered. Only terms with raw p < .05 are shown, from 85 tests (5 outcomes × 17 terms; RRI omitted). FDR was applied across all 85 tests. No term survived correction; effects with p < .05 but p (FDR) ≥ .05 are exploratory.

Window length and temporal resolution

According to recent HRV measurement guidelines, short-term segments of 30–60 seconds are adequate for reliable estimation of high-frequency (HF) components, whereas low-frequency (LF) estimation typically requires ≥ 2 minutes of data (Quigley et al., 2024). Consequently, LF (ms^2) estimates from shorter moving windows may be influenced by limited cycle representation and should be interpreted with caution. Because our goal was to examine time-varying, moment-to-moment HRV changes, we applied a 60-second moving window with a 1-second step. This approach loses approximately 30 seconds at each end of the epoch, whereas a conventional 2-minute LF window would lose approximately 60 seconds at each end (120 seconds in total). It is therefore unsuitable for capturing moment-to-moment autonomic fluctuations (Fig. 2).

To check that the 60-second windows were adequate, we compared HRV estimates from 60-, 120-, and 300-second windows computed with the same methods. Agreement across window lengths was excellent (ICC(3,k)) during both conditions. For time-domain indices, ICCs were 0.995 (SDNN), 0.999 (RMSSD), and 0.999 (pNN50) during NB, and 0.998, 0.999, and 0.999 during 0.1 Hz PB. For frequency-domain indices, LF ICCs were 0.981 (NB) and 0.999 (0.1 Hz PB), and HF ICCs were 0.996 (NB) and 0.998 (0.1 Hz PB); ICCs for ln LF and ln HF were all ≥ 0.994. Estimates from shorter windows were close to the 300-second values (60-s windows: 90–105%; 120-s windows: 96–103%), with the largest deviation for SDNN during NB (90% at 60 s and 96% at 120 s).

Table 4
Agreement Between Time- and Frequency-Domain HRV Indices Estimated From 60-s, 120-s, and 300-s Windows During Natural Breathing (NB) and 0.1 Hz Paced Breathing (0.1 Hz PB)

Index	ICC(3,k) NB	ICC(3,k) PB	60 s as % of 300 s (NB / PB)	120 s as % of 300 s (NB / PB)
SDNN (ms)	.995	.998	89.9 / 95.1	96.2 / 95.8
RMSSD (ms)	.999	.999	98.4 / 98.5	98.8 / 98.1
pNN50 (%)	.999	.999	100.0 / 100.0	99.1 / 99.0
LF (ms²)	.981	.999	97.5 / 103.3	97.3 / 102.1
ln LF	.994	.999	—	—
HF (ms²)	.996	.998	103.5 / 105.2	101.4 / 103.0
ln HF	.998	.999	—	—

ICC(3,k) = two-way mixed-effects, consistency, average-measures intraclass correlation coefficient across the three window lengths (n = 91). Values for 60-s and 120-s windows are the mean of all windows (1-s step) in each recording; 300-s values come from the full first 300 s. Percentages are the group mean at that window length divided by the group mean at 300 s (not computed for log-transformed indices). SDNN = standard deviation of NN intervals, RMSSD = root mean square of successive differences, pNN50 = percentage of successive differences > 50 ms, LF = low frequency, HF = high frequency, ln = natural logarithm.

Discussion

Temporal patterns of 0.1 Hz paced breathing

Our central question was how the effects of 0.1 Hz paced breathing unfold over the 5-minute episode. SDNN, RMSSD, and LF power were higher during 0.1 Hz paced breathing than during NB throughout the episode, and the difference was already at or near its maximum in the first minute (for example, SDNN about 105 ms versus about 56 ms during NB). The advantage then narrowed gradually: SDNN and RMSSD declined by roughly 10–13 ms between the first and last minutes of paced breathing (Time × Condition, both $p_{FDR} \leq .004$), whereas the LF difference stayed large (about 2.0–2.4 ln ms²) and its change over time was not significant after correction. Because slow paced breathing drives large heart rate oscillations near 0.1 Hz through the baroreflex (Lehrer et al., 2000; Vaschillo et al., 2006), LF and SDNN are the most direct readouts of resonance, and a response that is established almost immediately is consistent with a resonance effect that appears as soon as paced breathing begins (although breathing itself was not verified). Individuals also differed substantially in the shape of their LF and SDNN trajectories, beyond differences in level or linear trend.

Why the effect narrows over the episode cannot be determined from these data, and several explanations are possible. Participants may have drifted slightly from the paced rate; the dominant HRV frequency stayed within 0.09–0.11 Hz for 96% of the paced-breathing period on average, but the

impedance-derived respiration signal was too noisy to analyze, so adherence to the pacer could not be verified and departures cannot be excluded. Sustaining a 10-s breathing cycle for five minutes is effortful, and the effort may reduce the depth or regularity of breathing over time, which would reduce the amplitude of respiratory-driven oscillations. In addition, resonance frequency differs between individuals (typically 4.5–6.5 breaths per minute)(Lehrer & Gevirtz, 2014), whereas all participants breathed at a fixed 0.1 Hz, so many were paced slightly above or below their own resonance frequency. In a damped oscillator driven near but not at its natural frequency, the response combines a steady component with a decaying transient, so the amplitude can overshoot or fluctuate before settling, which may also contribute to a response that is largest early (in line with evidence that resonance-related dynamics are not always stable over time) (Capdevila et al., 2021).

RMSSD, which emphasizes faster beat-to-beat variability, is a less sensitive index of slow resonance oscillations, although it was also higher during 0.1 Hz paced breathing throughout the episode and declined over time. HF power was lower during 0.1 Hz paced breathing than during NB across the episode in the trajectory analysis (the linear-model Condition effect did not survive correction, p_FDR = .072) and did not change significantly over time. This is expected: HF power reflects respiratory sinus arrhythmia only when breathing falls within about 9–24 breaths per minute (Laborde et al., 2017; Shaffer & Ginsberg, 2017); at slower rates such as 6 breaths per minute, vagally mediated respiratory oscillations move into the LF band (Shaffer & Ginsberg, 2017), so the HF power that remains likely reflects noise and harmonics (for example, near 0.2 Hz) rather than a distinct autonomic process; HF is therefore of limited interest in this paradigm.

Practically, the effects of 0.1 Hz paced breathing on SDNN, RMSSD, and LF were present from the first minute, which suggests that brief sessions may capture much of the effect and that HRV sampled early in the episode may best reflect the paced-breathing response. Because the effect narrowed over five minutes and individuals differed in trajectory shape, the optimal duration and pacing rate may vary between people, which could be tested with individually determined resonance frequencies and monitoring of adherence. These are hypotheses for future work. These findings emphasize the potential value of examining continuous HRV signals rather than relying solely on pre–post change scores, which may obscure meaningful physiological transitions that occur over time.

Heart rate and R-R interval

HR and RRI showed a different time course from the HRV indices. HR was lower (and RRI longer) during 0.1 Hz paced breathing at the start of the episode, but it rose more steeply than during NB (and RRI fell more steeply), so that the two conditions crossed at about 3 minutes and HR was higher (and RRI shorter) during paced breathing for the remainder of the episode (by about 2 bpm at the end). Both HR and RRI changed non-linearly in both conditions.

The combination of high and slowly declining variability with a rising heart rate suggests that sustaining 0.1 Hz breathing was mildly activating, even as SDNN, RMSSD, and LF remained elevated. Heart period reflects the combined influence of vagal and sympathetic activity, so these data cannot show which

changed. The dissociation does suggest that one aspect of vagal-related control (variability) moved up while another (mean heart rate) moved toward arousal. Such dissociations are not unusual, and they argue against describing 0.1 Hz paced breathing as stimulating the vagus in general; its effects appear concentrated in regulatory functions rather than in tonic control of heart rate. This interpretation is tentative.

The rise in HR during 0.1 Hz paced breathing may reflect the effort the task demands: breathing in and out for 5 s each is unusual, and sustaining it for 5 minutes may take more effort the longer it continues, especially for people who are less fit, while the large HR swings within each breath cycle kept HRV high. The lower HR during the first minutes of paced breathing has several possible causes that cannot be separated here: slow, deep breathing alters intrathoracic pressure, venous return, and baroreflex activity (Russo et al., 2017), but usable respiration data (the impedance-derived signal was too noisy to analyze) and blood pressure were not available. During NB, HR also changed over the episode (a rise followed by a plateau), which may reflect increasing arousal or reduced engagement during extended seated rest, or anticipation of the tasks that followed (Marschin & Herbert, 2026); the change in eye state during NB may also have contributed.

Exploratory moderators

Women had lower LF power and lower SDNN than men overall, but neither effect survived correction for multiple comparisons ($p_{FDR} = .067$ and .480), so they are treated as exploratory; the direction is consistent with meta-analytic evidence of lower LF power in women (Koenig & Thayer, 2016). These are main effects of gender and do not indicate that gender moderated the response to 0.1 Hz paced breathing. HR was lower in older participants, again not surviving correction. After correction, no interaction of BMI, age, gender, or alcohol use severity with breathing condition or time was significant. The absence of moderation is not surprising in a homogeneous sample of healthy young adults of similar age, with a restricted range of alcohol use severity (61.5% below the AUDIT hazardous-use threshold), and the study was not powered to detect small interactions. Participants with AUDIT scores ≥ 8 did not differ from those below 8 at rest, during paced breathing, or in their change between conditions, and AUDIT scores were not correlated with any index. Because the sample included about 64% women, gender comparisons were exploratory and limited in power.

Main effects of 0.1 Hz paced breathing

These effects are well established and are not central to the present hypothesis, so we summarize them briefly. Consistent with prior studies, 0.1 Hz paced breathing in the present sample elicited robust increases in SDNN and RMSSD compared with normal breathing rates, supporting that slow breathing enhances vagally mediated HRV (Laborde et al., 2017; Lehrer & Gevirtz, 2014). In line with theoretical models of cardiorespiratory resonance, LF power was markedly elevated during 0.1 Hz paced breathing, reflecting the expected enhancement of low-frequency oscillations associated with baroreflex-driven breathing rhythms (Bates et al., 2022). HR was lower and RRI longer at the start of paced breathing, and HF power tended to be lower.

Strengths

The present study had several strengths. It was conducted in a moderately sized, homogeneous sample of young adults, providing adequate statistical power to detect the large main effects of paced breathing, although not small interactions. By employing linear mixed models alongside generalized additive mixed models and second-by-second analyses, we were able to capture not only average differences between NB and 0.1 Hz paced breathing conditions but also the dynamic, non-linear temporal trajectories of HRV responses. The inclusion of both time-domain and frequency-domain indices strengthens the robustness of the findings, and alcohol use severity, which is rarely considered in respiratory intervention research, was examined as a source of inter-individual variability. Beat-level data were cleaned with an explicit, reproducible procedure, the derived indices closely matched MindWare's own output, and the dominant HRV spectral peak during paced breathing fell at the target frequency in all included participants (0.099–0.104 Hz). Random slopes by participant, AR(1) errors in the trajectory models, and correction for multiple comparisons guarded against overinterpreting effects.

Limitations

At the same time, certain limitations warrant consideration. The sample was restricted to apparently healthy young adults with recent binge drinking. It thus represents a convenience sample that may not generalize to lighter, non-drinking populations, older aged people, or clinical populations. The intervention was relatively brief (five minutes), and longer breathing sessions may reveal additional adaptations not captured here. All participants breathed at a fixed 0.1 Hz rather than at an individually determined resonance frequency. The impedance-derived respiration signal was too noisy to yield respiratory measures, so adherence to the paced rate was screened only from the dominant frequency of the HRV spectrum, which indicates that heart rate oscillations occurred at the target frequency but does not verify breathing, and blood pressure oscillations were not measured, constraining mechanistic inferences about baroreflex involvement. Following the visual pacer during 0.1 Hz paced breathing added visual attention and effortful breathing demand, and NB included a change from eyes open to eyes closed whereas eyes were open throughout paced breathing, so condition differences may not reflect breathing rate alone; together with the fixed order (NB always first), effects of condition cannot be fully separated from effects of time in the session. The study was not powered to detect small moderation effects, and the range of alcohol use severity was restricted, with most participants below the AUDIT hazardous-use threshold. Because consecutive rolling windows overlap and are strongly autocorrelated, we modeled serial correlation explicitly, but residual dependence cannot be fully excluded. Differences in HR, RRI, and HF between conditions were confined to certain time windows or did not survive correction, and should be interpreted cautiously pending replication. Also, the laboratory setting may not fully reflect how 0.1 Hz paced breathing operates in daily life. These factors should be addressed in future work to validate and extend the present findings. Thus, results should be interpreted cautiously with respect to generalizability to lighter or non-drinkers, older age groups, and clinical populations.

Conclusion

This study shows that the effects of 0.1 Hz paced breathing unfold over time rather than remaining constant. SDNN, RMSSD, and LF power were higher during 0.1 Hz paced breathing throughout the episode, with the largest effects in the first minute and a slow narrowing thereafter, whereas heart rate started lower and rose above the natural-breathing level after about 3 minutes, indicating a selective rather than global effect on vagal control. Individuals differed in the shape of their trajectories, but moderation by gender, BMI, and alcohol use severity was not supported after correction for multiple comparisons. Future research should expand to older and clinical populations, measure respiration directly and individualize pacing to each person's resonance frequency, examine longer training protocols, and leverage digital health technologies to optimize delivery and monitoring. Together, this work advances our understanding of the temporal nature of 0.1 Hz paced breathing, supporting its potential as a practical tool for enhancing autonomic health and stress regulation.

Declarations

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Ethics approval

The study was approved by the Rutgers University Institutional Review Board for the Protection of Human Subjects (Protocol Pro2021001073) and was conducted in accordance with the ethical standards of the 1964 Declaration of Helsinki and its later amendments.

Consent to participate

All participants provided written informed consent before taking part in the study.

Consent to publish

Not applicable.

Data availability

The data are not publicly available because they contain sensitive information on alcohol and other substance use from young adults. De-identified data and the analysis code are available from the corresponding author on reasonable request, subject to institutional and participant-consent requirements.

Author contributions

M.K. conceived the study, performed the formal analysis, prepared the figures, and wrote the original draft. A.S. contributed to the statistical analysis. M.M. cleaned the data. P.L. contributed to the methodology and statistical approach and to drafting the manuscript. B.A. and M.E.B. led the parent study, acquired the funding, administered the project, and supervised the work. All authors reviewed and edited the manuscript and approved the final version.

Conflict-of-Interest Statement

The authors declare that they have no conflict of interest.

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Figures

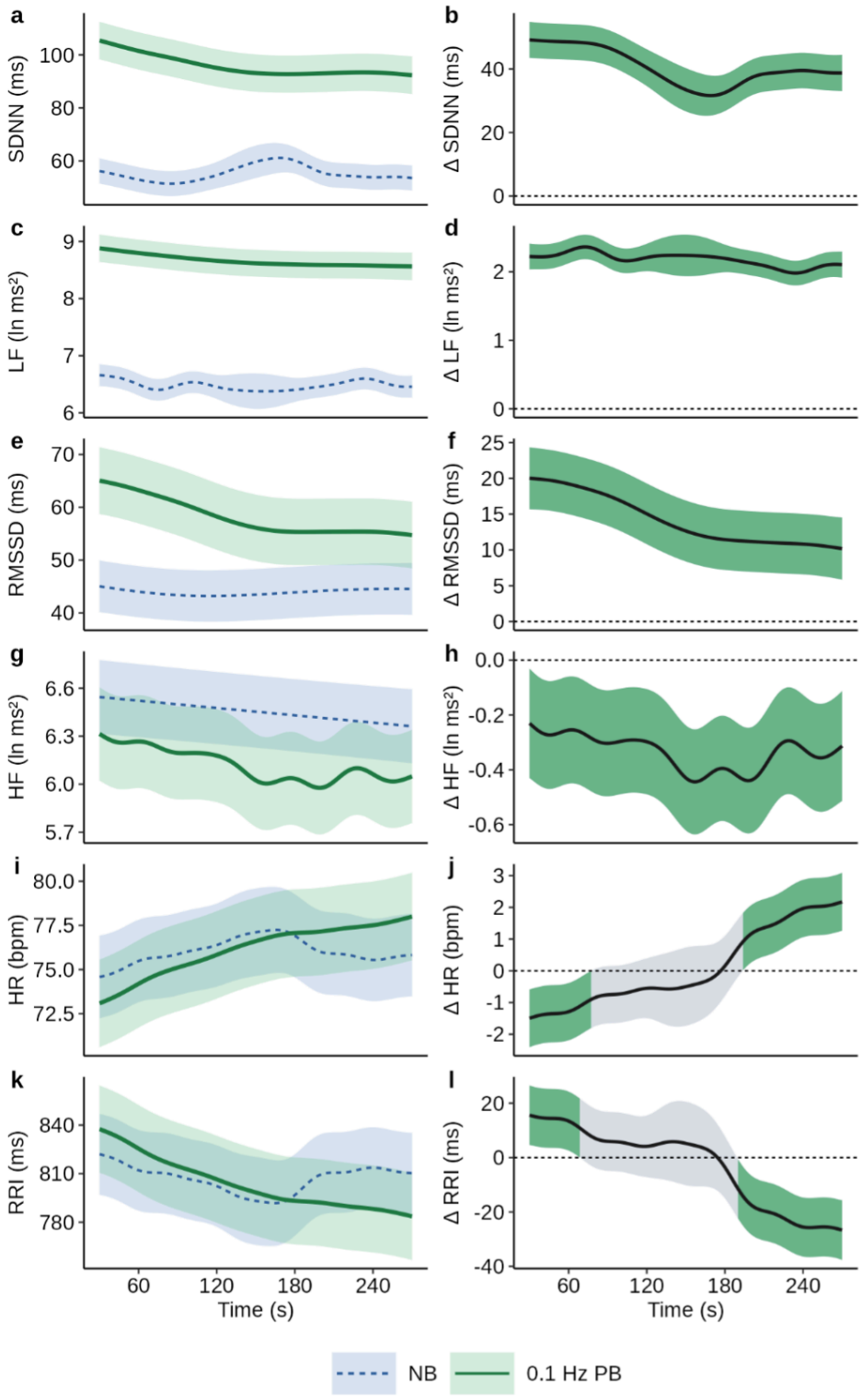


Figure 1

Trajectories of heart rate variability indices and heart rate during natural breathing (NB) and 0.1 Hz paced breathing (PB). Panels a, c, e, g, i, and k show model-predicted group curves (NB, dashed line; PB, solid line) with 95% confidence bands for SDNN (standard deviation of NN intervals), LF (ln low-frequency power), RMSSD (root mean square of successive differences), HF (ln high-frequency power), HR (heart rate), and RRI (mean R-R interval in 60-s windows). Panels b, d, f, h, j, and l show the difference between

conditions (PB − NB) with its 95% confidence band; darker shading marks time windows in which the interval excluded zero. Curves are from generalized additive mixed models with a random condition effect for each participant and first-order autoregressive errors

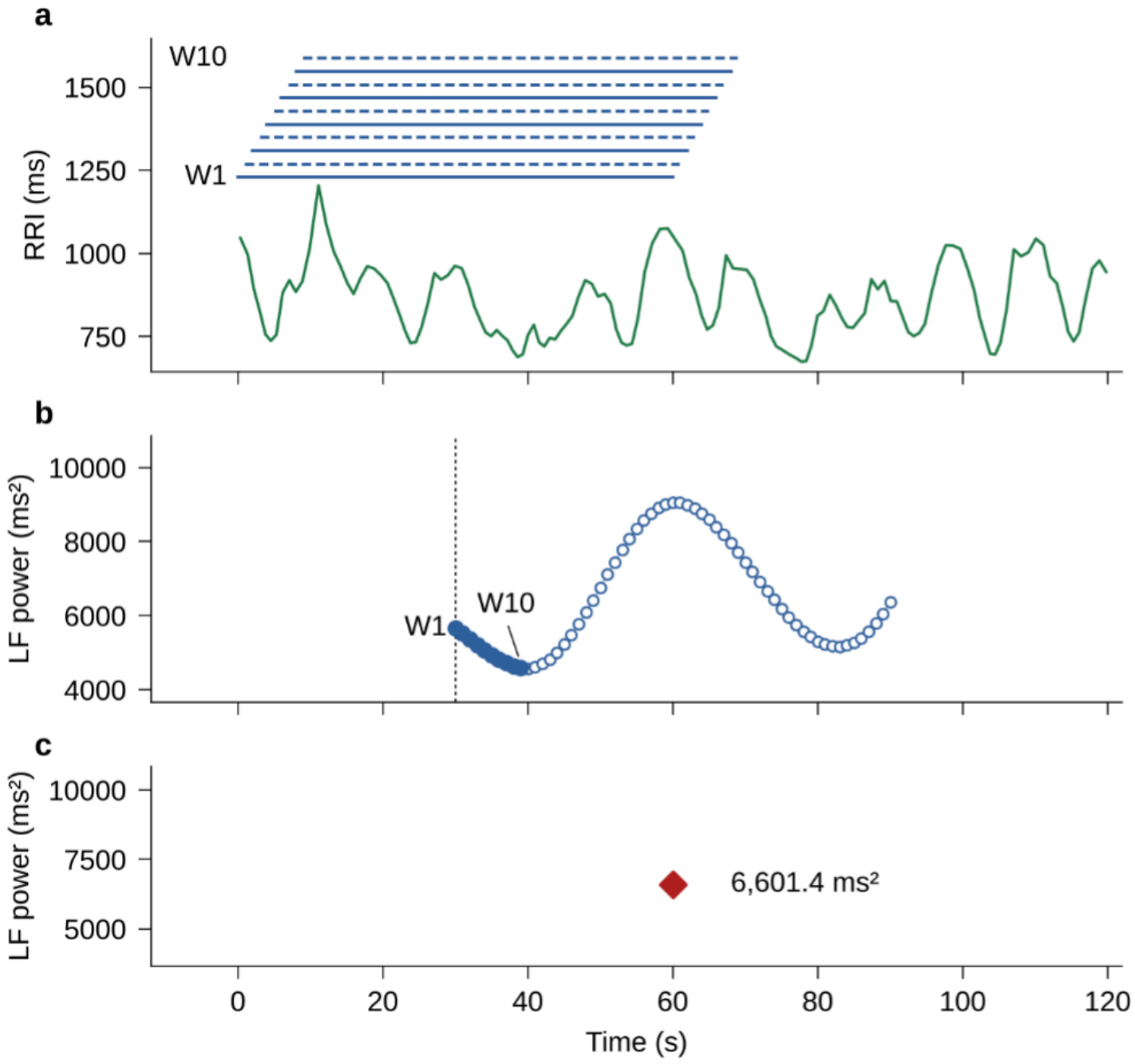


Figure 2

Comparison of 60-s moving-window and 120-s window HRV analysis, illustrated with a 120-s excerpt of an RRI signal from one participant. a RRI time series with horizontal bars showing the first ten successive 60-s windows (W1–W10), each advanced by 1 s. b LF power (ms²) computed in each 60-s window yields 61 values across the excerpt, each plotted at the window midpoint (first value at 30 s);

filled circles correspond to W1–W10, open circles are the remaining windows, and the dashed vertical line marks the first window. c A single 120-s window yields one LF value (diamond, 6,601.4 ms²), plotted at the window midpoint (60 s), and provides no information about change over time