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Effects of chronic and state loneliness on heart rate variability in women

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Abstract

Background Loneliness, the subjective experience of social isolation, represents one of the largest risk factors for physical illness and early death in humans. However, the mechanisms by which loneliness leads to adverse health outcomes are not well understood. ***Purpose*** In this study, we examined altered parasympathetic nervous system function as a potential pathway by which chronic loneliness and state loneliness may "get under the skin" to impact cardiovascular physiology. ***Methods*** In a controlled laboratory setting, vagally-mediated resting heart rate variability (HRV), HRV reactivity to an induction of state loneliness, and HRV reactivity to a cognitive challenge task were assessed in a sample of 316 healthy women (18–28 years). ***Results*** Greater chronic loneliness in women predicted lower resting HRV, an independent risk factor for cardiovascular disease and all-cause mortality, after controlling for demographic, psychosocial, and health behavior covariates. Furthermore, women higher in chronic loneliness experienced significantly larger increases in HRV to state loneliness and reported significantly higher levels of negative affect immediately following state loneliness, compared to their less chronically lonely counterparts. Chronic loneliness also predicted blunted HRV reactivity—a maladaptive physiological response—to cognitive challenge. ***Conclusions*** The current findings provide evidence that chronic loneliness is associated with altered parasympathetic function (both resting HRV and HRV reactivity) in women, and that the immediate experience of state loneliness is linked to a proximate increase in HRV among chronically lonely women. Results are discussed in terms of implications for cardiovascular health and the evolutionary functions of loneliness.

Keywords: chronic loneliness, state loneliness, heart rate variability, HRV reactivity, vagal, parasympathetic

Effects of Chronic and State Loneliness on Heart Rate Variability in Women

Loneliness is the aversive affective and cognitive experience that arises when the quality or quantity of social relationships that one actually has is different from what one desires [1]. More than merely an unpleasant feeling, loneliness is an independent risk factor for a plethora of adverse health outcomes, including cardiovascular disease, impaired cognition, reduced immune function, and accelerated aging [2]. Cumulative longitudinal evidence has demonstrated that the increased risk of early death associated with loneliness exceeds the mortality risks associated with obesity, physical inactivity, hypertension, and air pollution [3,4]. How does loneliness—a subjective experience of the mind—get "under the skin" to have such dramatic effects on our physiology and physical health?

In the present study, we investigate whether the experience of loneliness may systematically alter parasympathetic nervous system functioning in ways that have implications for cardiovascular health. Specifically, we examine the effects of chronic loneliness and state loneliness on vagally mediated high-frequency heart rate variability (HF-HRV), a measure of parasympathetic functioning and emotion regulation that is intimately linked to cardiovascular health [5,6] (for the remainder of the article, vagally-mediated HF-HRV is referred to simply as HRV). We further distinguish between resting HRV and HRV reactivity (i.e., change in HRV in response to a stimulus), to determine how each may be associated with loneliness.

Chronic Loneliness and Low Resting HRV

Although chronic loneliness and low resting HRV are both established risk factors for heart disease and early death [3,7,8], it remains unclear whether and how loneliness and HRV are directly linked. Both the model of neurovisceral integration [9,10] and polyvagal theory [11,12] posit that strong vagal parasympathetic regulation of the heart, characterized by higher resting

HRV, enables increased attentional control and emotion regulation. This conceptualization is consistently supported by research linking higher resting HRV with greater emotion regulation, attentional control, and executive function [e.g., 5,13,14,15]. However, it is not established whether chronic loneliness, which is also consistently linked to compromised emotion regulation, attentional control, and executive function [2,16,17], is associated with diminished parasympathetic regulation of the heart. In line with predictions of the neurovisceral integration model [10], chronic exposure to perceived adverse life experiences has been associated with reduced parasympathetic cardiac function [18,19]. Similarly, the subjective experience of chronic loneliness may be adversely associated with parasympathetic regulation of the heart, reflected in lower resting HRV.

Thus, a connection between low resting HRV and loneliness is consistent with the two most influential biobehavioural models linking HRV to emotional experience, however, this connection has not been clearly supported in empirical research. Although pioneering work by Hawkley and colleagues [20] examining the physiological correlates of loneliness observed a non-significant trend for chronic loneliness to be associated with resting HRV, subsequent research on a heterogeneous sample of older adults did not detect an association between chronic loneliness and resting HRV [21]. Given the numerous biological (e.g., age, sex), environmental (e.g., temperature), and lifestyle (e.g., smoking; use of cardioactive medication) factors that contribute to variability in resting HRV [22], it is possible that associations with chronic loneliness have been obscured within the heterogeneous samples used in prior research.

The need for larger samples with sufficient statistical power to detect effects in scientific research is highlighted by recent concerns surrounding the replicability of research findings [e.g., 23]. The median statistical power of HRV studies was recently reported to be .45 [24], and is

estimated to be between .35 and .65 in psychology studies [25], levels well below the commonly-recommended power level of .80 [26]. Determining if and how loneliness is associated with HRV is critical to our understanding of how psychological factors such as negative emotions affect physiological health. Thus, the first goal of the current research is to establish whether the relationship between chronic loneliness and low resting HRV consistent with prior theory [9,10,11,12] can be detected in a larger and more homogenous sample than has been studied in prior work.

HRV Reactivity to State Loneliness

Whereas chronic loneliness is generally considered to be physiologically detrimental, the physiological implications of temporary or transient states of loneliness remain understudied and unclear. Prevailing theories about the evolutionary functions of loneliness suggest that it simultaneously triggers both hypervigilance to threat as well as motivation to socially reconnect [16]. One possibility is that an acute episode of loneliness may provoke a temporary withdrawal of parasympathetic activity, reflected in *decreased* HRV, to facilitate mobilization of energy resources to deal with perceived impending danger. Extending the principles of allostatic load [27], repeated temporary decreases of HRV elicited by states of loneliness could result in chronically low HRV (i.e., low resting HRV). Conversely, acute episodes of loneliness could activate heightened parasympathetic activity and *increased* HRV, a physiological response theorized to accompany the motivation to socially engage [12].

Individual differences in chronic loneliness may also affect how a person physiologically responds to state loneliness. Research suggests that individuals high in chronic loneliness are more reactive to situations that can elicit state loneliness; specifically, chronically lonely adolescents display hypersensitivity to social exclusion (i.e., increased negative emotions) and

report greater levels of state loneliness when alone [28,29]. It is plausible that individuals high in chronic loneliness may be more affected by, and show a greater change in HRV from baseline to, an acute experience of loneliness. HRV reactivity to an experience of state loneliness has not previously been investigated.

The lack of research on the physiological correlates of state loneliness is understandable when considering the difficulties involved in eliciting state loneliness in the laboratory. To address these difficulties, we developed a loneliness manipulation paradigm with high face validity by adapting several loneliness induction techniques applied in past research [30,31,32,33,34]. The success of this novel loneliness manipulation paradigm would enable future researchers to more directly examine the effects of state loneliness on physiological, psychological, and social parameters. Furthermore, it may be particularly valuable for clarifying the mechanisms through which individual experiences of loneliness may add up to adversely influence health. To this end, the second goal of the current research is to understand the proximate effects of state loneliness on HRV reactivity, by inducing an acute experience of loneliness under controlled laboratory conditions.

Chronic Loneliness and HRV Reactivity to Cognitive Challenge

In addition to associations with resting HRV and HRV reactivity to negative affective states, there is theoretical support and preliminary empirical evidence for a link between chronic loneliness and HRV reactivity to cognitive challenge. According to polyvagal theory, decreases in HRV from resting levels (i.e., parasympathetic withdrawal) during cognitive challenge reflect an adaptive autonomic system that is able to rapidly regulate metabolic resources in response to environmental demands [12,35]. Diminished attentional control and poorer performance on cognitively demanding tasks have been found to be associated with blunted parasympathetic

withdrawal (i.e., smaller decrease in HRV) during the period of cognitive challenge [36,37]. Furthermore, chronic loneliness is associated with impaired self-regulation, including reduced attentional control [16]. Some of the most compelling evidence to date for a link between chronic loneliness and HRV was provided by Muhtadie and colleagues [38] in their study on loneliness and HRV reactivity to cognitive challenge. Chronic loneliness, but not depression, anxiety, or perceived stress, was found to predict blunted HRV reactivity, characterized by smaller decrease in HRV, to a visual attention task in a community sample of 76 men and women. Consistent with polyvagal theory, blunted HRV reactivity to cognitive challenge was interpreted by Muhtadie and colleagues as a sign of diminished parasympathetic system flexibility, and that lonelier individuals "might have a [reduced] capacity to tune into their social world and engage with others" [38]. As such, and in line with our goal to examine the relationship between chronic loneliness and low resting HRV in a larger and more homogenous sample than has been studied in prior work, the third goal of the current research is to clarify—using a larger and relatively homogenous single-sex sample, and controlling for covariates known to affect HRV, including habitual exercise, sleep, and alcohol consumption [22,39]—whether chronic loneliness and HRV reactivity to cognitive challenge are linked in women.

Sex Differences in Resting HRV and HRV Reactivity

Previous research indicates that there are important sex differences in both resting HRV and HRV reactivity. Autonomic control of the heart differs between men and women such that women typically have higher resting HRV compared to men [40], and women and men show differential patterns of HRV reactivity to negative social interactions and daily sadness [41,42]. Indeed, sex differences in HRV may have obscured potentially meaningful biobehavioral associations in past work using mixed-sex samples [43]. Women, in particular, may be more

likely to show an increase in HRV during state loneliness as a corollary to the "tend-and-befriend" social reengagement response theorized to be more characteristic of women than men [42,44]. Given the theoretical reasons for expecting sex differences in physiological responses to acute loneliness, we focused on obtaining a large, all-female sample for this initial study.

Overview and Hypotheses

The overarching goal of this study was to examine whether and how chronic loneliness, state loneliness, and vagally-mediated parasympathetic activity (both resting HRV and HRV reactivity) are linked, in ways that may help to explain how loneliness influences cardiovascular health. Chronic loneliness was measured using the UCLA Loneliness Scale [45] and high-frequency heart rate variability (HRV) was used to index parasympathetic activity. State loneliness was induced by modifying existing methods to develop a more potent loneliness manipulation. Under controlled conditions in the laboratory, we recorded women's HRV before, during, and after a cognitive challenge task, as well as before, during, and after an acute experience of state loneliness. Our main research objectives were to (a) test whether greater chronic loneliness is associated with lower resting HRV, a risk factor for cardiovascular disease, (b) investigate if and how an experience of state loneliness differentially affects HRV in high-chronically lonely and low-chronically lonely women, and (c) replicate and extend prior research by Muhtadie and colleagues [38] on chronic loneliness and HRV reactivity to cognitive challenge. On an exploratory basis, we also examined whether associations between chronic loneliness and measures of HRV were driven by specific domains of loneliness (i.e., friendship, family, and/or romantic loneliness), as assessed by the Social and Emotional Loneliness Scale for Adults (SELSA-S, short form) [46].

H1. Among women, higher self-reported chronic loneliness will predict lower resting HRV at baseline. The effect of chronic loneliness on resting HRV will be independent of individual differences in depression, anxiety, and perceived stress.

H2. Physiological response to an experience of state loneliness may differ among women as a function of chronic loneliness. Greater chronic loneliness may predict an exaggerated change in HRV during an episode of state loneliness, as individuals high in chronic loneliness are more likely to be acutely affected by feelings of loneliness. It is expected that the effect of chronic loneliness on HRV reactivity to state loneliness will not be explained by individual differences in depression, anxiety, and perceived stress. As no prior research has examined HRV during state loneliness, and findings on HRV reactivity to negative emotion states are not consistent [47], we do not predict a specific direction (increase versus decrease) for the change in HRV from baseline to state loneliness.

H3. As previously found by Muhtadie et al. [38], greater chronic loneliness is expected to predict blunted HRV reactivity to cognitive challenge. The effect of chronic loneliness on HRV reactivity to cognitive challenge will be independent of individual differences in depression, anxiety, and perceived stress.

Exploratory analyses. For each of our three hypotheses, statistical analyses were repeated using the social (i.e., friendship), family, and romantic loneliness subscales of the SELSA-S [46] in place of the UCLA Loneliness Scale [45].

Method

Participants

Given the evidence for substantial age and sex differences in heart rate variability [22,40,48,49], we restricted recruitment to women between the ages of 18 and 30 years.

Exclusion criteria were derived from recommended heart rate variability (HRV) research guidelines [39,50,51]. Specifically, individuals were ineligible for the study if they reported any of the following: a chronic heart condition, use of cardioactive medication, and habitual smoking. The study protocol was approved by the local research ethics board. All participants were recruited from the University of British Columbia (UBC) Human Subject Pool, provided written informed consent, and were compensated with course credit.

Based on recommendations for sample sizes needed in psychological research to obtain stable estimates of true population correlations [52], our aim was to collect complete data from a minimum of 250 participants. Over the course of one academic year, we recruited 316 participants. After removing data from 51 participants based on pre-established exclusion criteria, the final sample comprised 265 women (M_{age} = 20.03 years, *SD* = 1.85, age range: 18–28; see Table 1 for sample descriptives). Current use of cardioactive medication accounted for the largest number of exclusions (*n* = 31; note that all main findings remain stable if the 31 women using cardioactive medication are included in the analyses), followed by corrupted or missing data (*n* = 15), failure to follow study protocol (*n* = 2), reporting prior knowledge of the study protocol (*n* = 1), having a chronic heart condition (*n* = 1), and being outside of the specified age range (*n* = 1). Based on the effect size estimate reported in prior research on the association between loneliness and HRV reactivity (*r* = .25) [38], expected power for the current study with 265 participants, after including the uncertainty of the initial effect size estimate in the analysis, is .87 [53,54].

Procedure

Each participant attended a 90-min lab session during which they completed a set of questionnaires assessing psychosocial, demographic, and health behaviour variables before their

HRV was monitored during a series of tasks. Participants were asked to refrain from vigorous exercise, and from consuming food, caffeine, or alcohol for 2 hr prior to the lab session. All sessions were conducted between 9:00 a.m. and 5:00 p.m. [55].

At the lab session, participants were seated in front of a computer monitor in a private, sound-attenuated, and temperature-monitored room. Electrocardiograph (ECG) leads were attached to the upper body, after which participants filled out several questionnaires, including a survey assessing behaviour in the preceding 24 hr (e.g., recent caffeine consumption, hours of sleep the previous night). This survey was also used as a compliance check of study protocol requests prior to the lab session. This period provided time for participants to acclimate to the testing environment (all participants were seated for at least 5 minutes before resting baseline HRV was measured) [39].

All participants subsequently completed a 5-min baseline resting period (*resting HRV*), following which they performed a 5-min cognitive task (*cognitive challenge HRV*). Participants then completed another 5-min rest period before undergoing a 20-min state loneliness manipulation. During the loneliness manipulation, participants were asked to reflect on an acute experience of loneliness for 5 min (*state loneliness HRV*), after which they completed a final 5-min rest period. Participants all completed the cognitive task prior to undergoing the state loneliness manipulation (task order was not randomized due to the potential for the state loneliness manipulation to differentially affect participants' performance on the cognitive task). At the end of the session, height and weight were measured, and participants were debriefed. Participants completed all tasks and rest periods alone while the experimenter waited outside the testing room; the experimenter only entered the room between tasks to provide instructions for the next step of the procedure. Throughout the lab session, participants remained seated, breathed

spontaneously and were instructed to avoid excessive movement. The study procedure timeline is presented in Figure 1.

Measures

Psychological variables. To determine whether our hypothesized relationships between loneliness and HRV are independent of individual differences in depression, anxiety, and perceived stress (psychological constructs related to, but distinct from, loneliness), in addition to chronic loneliness, the same measures of depression, anxiety, and perceived stress used by Muhtadie and colleagues [38] in their study of the association between loneliness and HRV reactivity to cognitive challenge were employed. A measure of the friendship, family, and romantic dimensions of loneliness was also included for exploratory analyses, as well as other psychological measures unrelated to the present research.

Chronic loneliness. The UCLA Loneliness Scale [45] assesses feelings of loneliness or subjective social isolation, and is commonly used as a measure of chronic loneliness [29,30,56,57,58]. Individuals indicate how often they feel the way described in each of 20 statements on a 4-point scale from 1 (*never*) to 4 (*always*). Sample items are "How often do you feel that you lack companionship?" and "How often do you feel left out?" Internal reliability for this scale was excellent in the present sample (Cronbach's $\alpha = .93$).

Depression. The Center for Epidemiological Studies Depression Scale-Revised (CESD-R) [59] assesses levels of depressive symptomology, with a focus on depressed mood. The CESD-R was designed for use in studies of the relationships between depression and other variables in the general population [59]. Respondents report how frequently they experience each of 20 symptoms on a 5-point scale from 0 (*not at all*) to 4 (*nearly every day for two weeks*). Internal consistency for the CESD-R was excellent in this sample ($\alpha = .93$).

Anxiety. Anxiety was assessed with the Burns Anxiety Inventory (BAI) [60], a measure of affective, cognitive, and physical symptoms of anxiety. Individuals indicate how often they are bothered by each of 33 anxiety symptoms on a 4-point scale from 0 (*not at all*) to 3 (*a lot*). Internal reliability for the BAI was excellent in the present sample ($\alpha = .94$).

Perceived stress. The 10-item Perceived Stress Scale (PSS) [61] assesses the extent to which individuals appraise their lives as stressful. Respondents rate how frequently they have felt that situations in the past month have been unpredictable, uncontrollable, and overloaded on a 5-point scale from 0 (*never*) to 4 (*very often*). In the current study, internal consistency for the PSS was good ($\alpha = .84$).

Friendship, family, and romantic loneliness. The 15-item short form of the Social and Emotional Loneliness Scale for Adults (SELSA-S) [46] measures loneliness within the different relationship domains of friendship, family, and the romantic pair-bond. The social loneliness subscale of the SELSA-S (hereafter, referred to as the "friendship subscale") consists of five items that assess satisfaction with one's friendships: a sample item is "I feel part of a group of friends." The 5-item family loneliness subscale measures satisfaction with the quality of one's family attachments (e.g., "I feel close to my family"), and the five items of the romantic loneliness subscale assess satisfaction with the quality of one's romantic attachments (e.g., "I have an unmet need for a close romantic relationship"). For all items, respondents rate the extent to which they agree with a self-descriptive statement on a 7-point scale from 1 (*strongly disagree*) to 7 (*strongly agree*). In the current sample, internal consistency was high for the friendship ($\alpha = .88$), family ($\alpha = .92$), and romantic ($\alpha = .91$) loneliness subscales.

State affect. Participants completed an 8-item measure of state affect at three points during the lab session: following the 5-min baseline resting period, and immediately before and

after the 20-min state loneliness manipulation. Specifically, participants were asked to indicate on a scale from 0 (*not at all*) to 10 (*extremely*), the degree to which they were feeling lonely, tense, relaxed, sad, worried, happy, bored, and angry. State loneliness was measured at each momentary assessment by the item, "How lonely do you feel right now?"

Heart rate variability (HRV). The selection and implementation of all equipment and procedures were consistent with HRV research guidelines [39,50]. Electrocardiograph (ECG) activity was recorded continuously using the Bittium Faros 180 system at a sampling rate of 1000 Hz. Disposable Ag/AgCl wet gel electrodes were attached to participants in a modified Lead II configuration (i.e., right clavicle, left lower torso). The digitized ECG signal was extracted and subsequently analyzed using Kubios HRV Premium 3.1 [62]. A combination of automated and visual approaches to artifact correction was applied [50]. In addition to identifying recording artifacts (e.g., ectopic beats, excessive movement) using an automated algorithm (Kubios HRV 3.1 automatic correction) [63], each minute of the digitized ECG signal was visually inspected for artifacts and corrected when necessary. The mean beats corrected per participant was less than 0.09% (*SD* = 0.17%). Equidistantly sampled time series data were then derived from the RR intervals (i.e., the time in ms between successive R-spikes in the ECG waveform) using cubic spline interpolation [64]. The data were detrended using the smoothness priors method (smoothing parameter $\lambda = 500$) [65], and submitted to a Fast Fourier Transform (FFT) to determine spectral power values (in ms^2/Hz). HRV was calculated as the natural logarithm (ln) of the integrated power values (ms^2) in the high-frequency (0.12–0.40 Hz) spectral bandwidth [66,67]. Respiration rate was obtained using ECG derived respiration (EDR), which estimates respiratory frequency from changes in R-wave amplitude [63]. Autoregressive modelling (AR) was employed as an additional measure of high-frequency HRV with which to

compare against the main analyses that used HRV derived by FFT. All FFT- and AR- derived measures were highly correlated (all *r*s > .95, all *p*s < .001), and produced almost identical results in all study analyses; thus only results using HRV derived by FFT are reported here.

A mean HRV score for each recording period (e.g., resting baseline, cognitive challenge, state loneliness) was obtained for each participant by aggregating the HRV values of 1-min epochs during each recording period. Change scores were calculated by subtracting a participant's mean HRV score for a preceding period from her mean HRV score of the subsequent period.

Cognitive challenge. To replicate and extend earlier findings on HRV reactivity and chronic loneliness [38], we employed the same visual cognition task [68] used to elicit HRV reactivity in the prior research. Importantly, this multiple-object tracking task: (a) has been reliably demonstrated to elicit HRV reactivity [36,38,69], (b) does not rely on higher-order executive functioning (therefore, performance is less affected by differences in intelligence and education), and (c) does not contain social and emotional content, and is thus distinct from the emotionally evocative loneliness manipulation.

Specifically, this cognitive challenge task required participants to visually track dots as they moved randomly across a computer screen. The task was comprised of 16 trials that each began with a display of 12 identical dots against a plain background. A subset of dots would flash for 2 s, designating them as the target dots to be tracked during the trial. The target dots then stopped flashing, became once again indistinguishable from the other dots, and then all the dots moved randomly around the screen for 12 s. At the end of each trial, all the dots stopped moving, and the participant had to identify the target dots using the mouse. The cognitive challenge became increasingly difficult as the trials progressed, with the number of target dots

increasing after every four trials. This task, which took approximately 5 min to complete (*M* = 4.92 min, *SD* = 36 s), was used to assess HRV reactivity to cognitive challenge, and was followed by a 5 min rest period.

State loneliness induction. An acute experience of loneliness was induced by modifying a combination of loneliness manipulation techniques reported in prior research [30,31,32,33,34]. To conceal the true purpose of the manipulation, participants were told that they would be helping to validate a new "text analysis application" called the Linguistic Analysis and Word Count (LAWC) program by completing an emotional writing task. This program was fictional, but based on the real LIWC2015 (Linguistic Inquiry and Word Count) program developed by Pennebaker, Boyd, Jordan, and Blackburn [70]. Specifically, each participant was told:

> The LAWC program is a text analysis application that enables rapid assessment of emotional and cognitive components in verbal and written speech. The LAWC assesses vocabulary, word patterns, and syntax to derive information about individuals' thought patterns, personality characteristics, and social relationships. Although the LAWC has been used effectively in Europe for several years, UBC is part of a Canadian initiative to validate the program with a North American student sample.

The participant was then left alone to read a set of instructions displayed on the computer screen which prompted her to write about an experience of loneliness. The participant was given 10 min to type her description of a personal loneliness experience into a text box, following which the experimenter re-entered the room and took a seat across from the participant (so that the computer screen was not visible to the experimenter). The experimenter prompted the participant to "submit" her writing sample, ostensibly for analysis by the LAWC program, by clicking a computer key. A preprogrammed page appeared onscreen displaying a "high" loneliness score of

52.98, as well as other information suggesting that the LAWC program had identified the participant as higher than average in loneliness. This additional information was designed to enhance the believability of the false feedback. The experimenter subsequently told the participant:

> The program should have provided you with a loneliness score between zero and eighty. Higher scores indicate more intrinsic loneliness, and lower scores indicate less intrinsic loneliness. The average loneliness score of a typical undergraduate student at UBC in 2017 was 36.2. Can you tell me if your score is lower, higher, or equal to the UBC average?

After the participant had replied that her loneliness score was higher than the university average, the experimenter wrote down the word "higher" on an official-looking recording sheet. The participant was then asked to spend the next 5 min reflecting on the loneliness experience that she had written about in light of the "feedback" from the text analysis program, while the experimenter waited outside the room. This 5-min period was used to assess HRV during an acute experience of loneliness. Immediately after this period, participants completed the measure of state affect previously described to ensure that the loneliness manipulation had been successful.

At the end of the lab session, participants provided their impressions of the state loneliness task by answering the item, "How effortful did you find the reflection task?" on a scale from 1 (*not at all*) to 7 (*extremely*), and the item, "How plausible did you feel the loneliness feedback was?" on a scale from 1 (*not plausible at all*) to 7 (*completely plausible*). On average, participants experienced the task as moderately effortful (M = 3.52, SD = 1.65), and reported the

false feedback to be reasonably plausible (M = 4.03, SD = 1.54). Finally, all participants were thoroughly debriefed and offered the opportunity to watch videos containing positive stimuli.

Demographics and health behaviours. Information on a number of demographic variables and health behaviours was collected for inclusion as covariates in study analyses. Participants provided information on their age, sex, ethnicity, relationship status, how long they had been attending the university, how long they had lived in the city, current health conditions, and current medication use. Participants also reported their average hours of sleep per night, as well as their typical alcohol consumption, caffeine consumption, exercise habits, smoking habits, and marijuana use.

Results

All statistical analyses were conducted in SPSS (Version 24.0) and are reported following the order of the study hypotheses.

Chronic Loneliness and Resting HRV

Consistent with our first hypothesis (H1), higher chronic loneliness was a significant predictor of lower resting HRV at baseline in a simple regression analysis, $\beta = -.20$, $t(263) = -3.23$, $p = .001$ (see Figure 2). A sequential regression analysis was then conducted using chronic loneliness to predict resting HRV, after controlling for factors that can influence HRV [39,71], namely: age, body mass index (BMI; weight in kg/height in m^2), and respiration. Inclusion of chronic loneliness significantly improved the prediction accuracy of the regression model, $\Delta R^2 = .02$, $\Delta F(1, 260) = 7.54$, $p = .006$. Specifically, higher chronic loneliness predicted lower resting HF-HRV, $\beta = -.15$, $t(260) = -2.75$, $p = .006$, even after accounting for age, BMI, and respiration (see Table 2). As such, this analysis provides the first evidence of a significant association between loneliness and low resting HRV. Notably, the relationship between chronic loneliness

and resting HRV remained significant even if the 31 women taking cardioactive medication were added to the analysis, and regardless of whether HRV was derived using frequency-domain (i.e., FFT, AR) or time-domain (i.e., RMSSD) measures.

To ensure that the relationship between chronic loneliness and resting HRV was not driven by psychosocial variables related to loneliness, we examined the associations between resting HRV and depression, anxiety, and perceived stress. Resting HRV was found to be correlated with depression ($r = –.17$, $p = .005$), anxiety ($r = –.12$, $p = .054$), and perceived stress ($r = –.18$, $p = .003$). However, when depression, anxiety, and perceived stress were individually added to the regression model, the association between chronic loneliness and resting HRV was not significantly altered, and the associations between resting HRV and depression, anxiety, and perceived stress became nonsignificant (all ps > .24).

On an exploratory basis, we also investigated the relationships between resting HRV and loneliness within the domains of friendship, family, and the romantic pair-bond. Lower resting HRV was associated with greater loneliness in the domains of friendship, $\beta = –.15$, $t(263) = –2.47$, $p = .014$, and family, $\beta = –.20$, $t(263) = –3.24$, $p = .001$, whereas the association between resting HRV and romantic loneliness was not significant, $p = .365$. Taken together, our findings suggest that the observed relationship between low resting HRV and loneliness in our sample of young women may be driven by loneliness within the domains of friendship (e.g., dissatisfaction with one's friendships) and family (e.g., perception of weak family bonds), rather than by romantic loneliness (e.g., desire for a satisfying romantic relationship).

State Loneliness Induction

Manipulation check. Self-reported state loneliness following the loneliness experience ($M = 3.47$, $SD = 2.66$) was significantly higher than self-reported state loneliness immediately

preceding the loneliness induction (M = 1.22, SD = 1.81), $t(264) = 16.44$, $p < .001$, $d = 1.01$, 95% CI [1.99, 2.53], indicating that the loneliness manipulation was successful. Furthermore, higher chronic loneliness predicted greater increases in state loneliness, $\beta = .17$, $t(263) = 2.78$, $p = .006$, suggesting that women higher in chronic loneliness had a larger affective response to the loneliness manipulation.

The significant increase in self-reported loneliness (ΔM = 2.26, SD = 2.24) observed after the loneliness experience (compared to immediately before the loneliness induction), was accompanied by significant increases in self-reported sadness (ΔM = 2.71, SD = 2.25), anger (ΔM = 0.63, SD = 1.56), worry (ΔM = 0.77, SD = 1.80), and tension (ΔM = 1.06, SD = 1.70), along with significant decreases in happiness (ΔM = –1.33, SD = 1.74) and feelings of relaxation (ΔM = –1.81, SD = 2.39). This broader pattern of emotional changes associated with states of loneliness [56,72] suggests that participants were experiencing loneliness, rather than merely exhibiting demand characteristics.

Chronic loneliness and HRV reactivity to state loneliness. HRV reactivity to state loneliness was calculated by subtracting the mean resting HRV obtained during baseline from the mean HRV obtained during state loneliness. A paired t test indicated that, on average, HRV did not differ between baseline and the experience of state loneliness (ΔM = 0.01 ln ms^2, SD = 0.73), $t(264) = 0.14$, $p = .886$, although a wide range of responses was observed: –2.00 to 2.70. To assess whether the effect of state loneliness on vagal parasympathetic activity differs between high-chronically lonely and low-chronically lonely women (H2), HRV reactivity to state loneliness was regressed on chronic loneliness scores. In support of a differential physiological response between high-chronically lonely and low-chronically lonely women, higher chronic loneliness was found to predict larger increases in HRV from baseline to the experience of state

loneliness, β = .15, $t(263) = 2.50$, $p = .013$ (see Figure 3). In contrast, HRV reactivity to state loneliness was not significantly correlated with the other background psychological measures that we assessed: depression, anxiety, or perceived stress (all rs < .10, all ps > .10).

Furthermore, the association between chronic loneliness and HRV reactivity to state loneliness remained significant after controlling for covariates in a sequential regression analysis. In addition to age, BMI, and respiration, we also controlled for self-reported task effortfulness, and self-reported plausibility of feedback. Inclusion of chronic loneliness significantly improved the prediction accuracy of the regression model, $\Delta R^2 = .02$, $\Delta F(1, 258) = 4.90$, $p = .028$. Specifically, higher chronic loneliness predicted greater increase in HRV during an experience of state loneliness, β = .14, $t(258) = 2.21$, $p = .028$, even after accounting for these covariates (see Table 3). These results suggest that the more chronic loneliness that a women reports feeling generally, the greater her increase in parasympathetic activity during an acute experience of state loneliness. Also in support of an association between increased parasympathetic activity and state loneliness, greater increase in HRV from just before the loneliness induction to the state loneliness experience, predicted higher self-reported loneliness immediately after the loneliness experience, β = .18, $t(263) = 2.93$, $p = .004$.

In exploratory analyses, we also examined the associations between specific dimensions of loneliness (friendship, family, and romantic) and HRV reactivity. In line with our findings on these relationship domains of chronic loneliness and resting HRV, greater loneliness within friendships was a significant predictor of larger HRV increases from baseline to the state loneliness experience, β = .18, $t(263) = 2.94$, $p = .004$. Furthermore, there was a statistical trend for family loneliness to predict HRV reactivity to state loneliness, β = .11, $t(263) = 1.84$, p =

.067, but no association was observed between romantic loneliness and HRV reactivity to state loneliness, $p = .810$.

HRV Reactivity to Cognitive Challenge

Manipulation check. To assess the effect of the cognitive challenge task on parasympathetic activity, HRV reactivity to cognitive challenge was calculated by subtracting the mean HRV obtained during baseline from the mean HRV obtained during cognitive challenge. Likewise, HRV recovery from cognitive challenge was calculated by subtracting the mean HRV observed during the cognitive challenge task from the mean HRV observed during the rest period immediately following the cognitive challenge. In line with prior research [36,38,69], mean HRV reactivity to cognitive challenge was negative, indicating an overall pattern of decreased parasympathetic activity across participants, $\Delta M = –0.51$ ln ms^2, $SD = 0.79$, although a considerable range of responses was observed: –2.68 to 2.04. A paired-samples *t* test revealed that on average, participants experienced significant parasympathetic withdrawal from baseline during the cognitive challenge, $t(264) = –10.55$, $p < .001$, $d = 0.65$, 95% CI [–0.60, –0.41]. Also as expected, mean HRV recovery was positive, indicating an overall pattern of increased parasympathetic activity across participants following cognitive challenge, $\Delta M = 0.44$ ln ms^2, $SD = 0.66$, but again, a considerable range of responses was observed: –1.32 to 2.28. A *t* test confirmed that on average participants experienced significant HRV recovery after the cognitive challenge task, $t(264) = 10.82$, $p < .001$, $d = 0.66$, 95% CI [0.36, 0.51].

Chronic loneliness and HRV reactivity to cognitive challenge. Chronic loneliness was not associated with task performance (defined as total number of correctly identified targets), all *p*s > .369. However, consistent with our third hypothesis (H3), and replicating Muhtadie et al. [38], higher chronic loneliness was correlated with blunted HRV reactivity to the cognitive

challenge task, $r(264) = .17$, $p = .007$. Specifically, higher loneliness scores were associated with smaller decreases in HRV to cognitive challenge, whereas lower loneliness scores were associated with greater decreases in HRV to cognitive challenge (see Figure 4). Also as found by Muhtadie et al. [38], HRV reactivity to cognitive challenge was not significantly correlated with depression, anxiety, or perceived stress, all rs < .10, all ps > .14. A sequential regression analysis was conducted to determine whether chronic loneliness explained a unique amount of variance in HRV reactivity to cognitive challenge, after controlling for age, BMI, concurrent changes in respiration, and task performance. The inclusion of chronic loneliness significantly improved the prediction accuracy of the regression model, $\Delta R^2 = .03$, $\Delta F(1, 254) = 8.98$, $p = .003$. Holding all other variables constant, higher chronic loneliness predicted blunted HRV reactivity to cognitive challenge, $\beta = .17$, $t(254) = 3.00$, $p = .003$ (see Table 4). To examine whether lonelier individuals were potentially less engaged in the cognitive challenge, we examined whether chronic loneliness was correlated with self-reported boredom before or after the task. Loneliness was not associated with feeling bored (all ps > .406), and when boredom was included in the regression model, the association between chronic loneliness and HRV reactivity to cognitive challenge was not significantly altered.

Mirroring our other findings on the friendship, family, and romantic dimensions of loneliness, greater friendship loneliness was a significant predictor of blunted HRV reactivity to cognitive challenge, $\beta = .21$, $t(263) = 3.44$, $p = .001$. Greater family loneliness also significantly predicted blunted HRV reactivity to cognitive challenge, $\beta = .13$, $t(263) = 2.13$, $p = .034$; however, no association was observed between romantic loneliness and HRV reactivity to cognitive challenge, $p = .264$.

Health Behaviour and Demographic Covariates

A number of health behaviour and demographic variables were examined as potential confounds. Variables found to be significantly associated with HRV measures (e.g., hours of weekly exercise, average alcohol consumption; see Table 5 for correlations) were individually added to the regression models. However, as none of these covariates significantly altered the association between measures of loneliness and HRV, the more parsimonious models without these variables are reported. In addition, reported results for all of the simple and sequential regressions for H1, H2, and H3 are essentially unchanged when the 31 women excluded for cardioactive medication use are added to the analyses.

Environmental Factors

Although room temperature was negatively correlated with resting HRV ($r = –.13$, $p = .030$), the inclusion of ambient temperature as a covariate in the regression model did not significantly alter the relationship between resting HRV and chronic loneliness. Room temperature was not associated with either HRV reactivity to state loneliness or HRV reactivity to cognitive challenge. Time of day was not related to any HRV measures and had no effect on any study analyses.

Discussion

The aim of this study was to investigate how loneliness, in both its chronic and transient forms, may influence parasympathetic functioning—as indexed by heart rate variability (HRV)—and ultimately, physical health. Among women, higher chronic loneliness predicted lower resting HRV, a risk factor for heart disease and early death, after controlling for age, body mass index (BMI), and respiration. Indeed, this finding of the first significant association between chronic loneliness and low resting HRV—a relationship consistent with prior theory

linking HRV to emotion (polyvagal theory; neurovisceral integration model)—remained robust even after psychological factors, such as depression and perceived stress, or lifestyle factors, such as quantity of exercise and sleep, were included in the analyses.

Providing further evidence for a link between loneliness and altered parasympathetic functioning, women high in chronic loneliness displayed significantly increased HRV reactivity to state loneliness, compared to their less chronically lonely counterparts, after controlling for covariates. This finding was not driven by individual differences in psychological constructs related to loneliness, namely: depression, anxiety, or perceived stress. Marking the first time that HRV has been assessed during a transient experience of loneliness, this finding provides initial evidence of a link between the immediate subjective affective and cognitive experience of loneliness and human physiology.

Evidence for a connection between chronic loneliness and altered parasympathetic activity was not restricted to the emotional domain: In a successful replication of work by Muhtadie and colleagues [38], high chronically lonely women displayed blunted HRV reactivity (smaller decrease in HRV) to a non-emotional cognitive challenge—a situation in which greater HRV reactivity (larger decrease in HRV) is considered optimal (to facilitate mobilization of metabolic energy for the visual tracking task) [36]. The present study further extends upon prior research by showing that this relationship holds in a larger sample (i.e., at the size at which correlation estimates are thought to stabilize) [52], by testing for associations with additional factors related to loneliness (e.g., health behaviours) [2], by controlling for task performance, and by determining that significant HRV recovery occurred after the cognitive challenge.

In exploratory analyses, all our observed associations between chronic loneliness and HRV measures were also predicted by higher loneliness in the domains of family and friendship,

but not by romantic loneliness. Average romantic loneliness was significantly higher than both averages for family loneliness and friendship loneliness in our sample. This observation may be explained by the youth of our university student sample: only 25.7% reported being in a committed relationship. Whereas being without a romantic partner is a situation that many in Western societies can expect to experience at some point in their early twenties, lacking family or friends is arguably less normative. As such, although romantic loneliness was not predictive of HRV measures in the current sample, the relationship between romantic loneliness and parasympathetic functioning may be different among the middle-aged or elderly.

Our observation that greater chronic loneliness is associated with lower resting HRV, combined with our replication of a previous finding that greater chronic loneliness is associated with blunted HRV reactivity to cognitive challenge [38], provides compelling evidence for a link between chronic loneliness and altered functioning of the parasympathetic nervous system in women. Furthermore, these results support the need for research into how altered parasympathetic functioning may lead to the adverse health outcomes observed among the chronically lonely. The fact that higher chronic loneliness predicted lower resting HRV and blunted HRV reactivity to cognitive challenge among the present sample of young women suggests that the deleterious effects of loneliness may be incurred much earlier in life than has previously been suspected [16].

The present findings are also consistent with research suggesting that loneliness and parasympathetic function may operate on common neural substrates [73]. The observation that parasympathetic activity was significantly increased during state loneliness in high chronically lonely women may represent a shift to a physiological state supportive of social reengagement behaviours triggered by acute and unpleasant feelings of loneliness [12,74,75]. On the other

hand, increased parasympathetic activity may represent a compensatory neurophysiological response to heightened amygdala activity thought to occur following presentation of negative stimuli [76,77]. As it has been theorized that this compensatory neurophysiological mechanism may be specific to women [43], future research examining HRV reactivity to state loneliness should include mixed sex samples large enough to allow analyses to be stratified by sex. This would enable researchers to examine potentially important sex differences in autonomic responses to affective stimuli.

However, caution should be exercised when interpreting the current findings for the non-directional hypothesis that HRV responses to an experience of state loneliness differ among women as a function of chronic loneliness. Specifically, we do not know what was in participants' minds during the five minutes that they spent reflecting on a personal experience of loneliness in light of the feedback suggesting they were lonelier than their peers. For example, one could interpret greater HRV decreases among low chronically lonely women as indicating greater cognitive effort from trying to resolve the false feedback of being lonely with one's own self-reported loneliness. Other alternative interpretations of greater HRV increases among high chronically lonely women include the possibility that the lonelier women felt affirmed by the feedback that they are very lonely, or that lonelier people are more tolerant of acute experiences of loneliness. Notably, chronic loneliness remained a significant predictor of HRV responses to state loneliness after controlling for participants' self-reports on how effortful they found the reflection period and how plausible they perceived the feedback. Although controlling for these covariates begins to address some of these alternative explanations, future research is needed to clarify the relationship between HRV and acute experiences of loneliness. More concretely, future research could employ an experimental design that includes a comparison condition to

show possible differences in responses to loneliness feedback compared to an "average" loneliness condition, as well as a more direct measure of individuals' cognitive effort while they reflect on their feelings of loneliness. In addition, external validity could be enhanced through future research examining the effects of state loneliness on HRV in individuals' daily lives by using ecological momentary assessments throughout the day.

Effect sizes for associations between loneliness and HRV in the current study were small, and in line with expectation given the quantity of biological, psychological, and environmental factors that contribute to HRV at a given time [78]. Such influences include individual differences in respiration, estrogen and testosterone levels, body temperature, postural changes, and fatigue, to name a few [79]. Considering such a range of antecedents, we would expect any single factor to account for only a fraction of the variance in a person's HRV in any particular context. The current findings suggest that loneliness accounts for a relatively small, but meaningful, proportion of variance in women's HRV and HRV reactivity. Indeed, this discussion highlights several strengths of our study, namely that we were able to control for age, BMI, and respiration; and furthermore, that the effects of loneliness on HRV were independent of individual differences in depression, anxiety, stress, and health behaviours. In addition, we observed associations between chronic loneliness and HRV in women using multiple operationalizations of loneliness (i.e., the UCLA and SELSA-S subscales) in a larger sample, increasing confidence that the observed effect more accurately reflects the true population effect size [52,80].

We decided to restrict study participation to healthy women in order to reduce physiological variability in our sample and increase statistical power to detect a true effect. However, it remains unclear whether psychophysiological relationships between loneliness and

HRV that we observed in women will be similar in men. In particular, state loneliness may not elicit the same pattern of HRV reactivity in men, and it is plausible that HRV may decrease among chronically lonely men in response to acute loneliness, reflecting the parasympathetic withdrawal characteristic of a flight-or-flight response [12,43]. Furthermore, the current sample was drawn from a non-clinical population of university students, and averages for loneliness and depression among our participants were comparable to averages found in similar demographic samples [45,81,82]. Thus, future research will be necessary to determine the generalizability of our findings to men and to clinical populations.

In the present study, in addition to measuring loneliness immediately before and after the loneliness manipulation, the assessment of a broader pattern of emotional changes associated with loneliness enabled us to demonstrate the efficacy of a novel loneliness induction method under controlled laboratory conditions. Initial evidence that state loneliness is associated with increased HRV among chronically lonely women further suggests that future research should take into account what has so far been a relatively neglected dimension of loneliness: duration. Used responsibly, similar state loneliness induction methods can be applied in different research fields to help disentangle the physiological, psychological, and social effects of chronic and state loneliness. Such research is needed to answer important questions about the process by which experiences of loneliness may exert a cumulative impact on parasympathetic functioning over the life span.

Loneliness is a well-established risk factor for numerous adverse health outcomes, including cardiovascular disease and early death. Understanding how loneliness, a subjective experience of the mind, manifests itself within the human body to influence physical health is an important first step towards the development of effective health interventions. The current

research provides novel evidence that chronic loneliness predicts lower resting HRV in women, which is associated with elevated risk of incident coronary heart disease and death. Through the assessment of HRV reactivity, this study also indicates altered parasympathetic functioning among chronically lonely women in response to an experience of state loneliness, as well as to a cognitively challenging task. By combining background measures of chronic loneliness with an experimental manipulation of state loneliness, this work documents one mechanism through which loneliness, in both its chronic and transient forms, may impact human psychological, social, and physical well-being.

Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards The authors have no conflicts of interest to disclose.

Ethical Approval All procedures were conducted in accordance with modern ethical standards and have been approved by the appropriate Institutional Review Board.

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Tables

Table 1

Sample Descriptive Statistics

Variable	% or *M* (*SD*)	Range	[Possible range]
Demographics			
Age	20.03 (1.85)		
Body mass index[a]	22.47 (3.45)		
% in a committed relationship	25.7%		
% living in Vancouver for one year or less	20.4%		
% first year university students	34.3%		
% report medical condition	9.8%		
% use hormonal contraceptives	30.2%		
Health behaviours			
Average hours of sleep per night	6.97 (1.00)		
% exercise one hour per week or less	28.7%		
% drink caffeine once per week or less	36.6%		
% drink alcohol once per month or less	69.8%		
% use marijuana less than once per month or never	91.3%		
Heart rate variability[b] (HRV)			
Resting Baseline	7.22 (1.03)	4.26 – 9.48	–
Cognitive Challenge	6.71 (0.90)	3.98 – 9.14	–
State Loneliness	7.22 (0.87)	4.56 – 9.54	–
Psychological measures			
Loneliness (UCLA)	42.11 (9.54)	23 – 67	[20 – 80]
Depression (CESD-R)	17.65 (13.18)	0 – 74	[0 – 80]
Anxiety (BAI)	20.78 (15.85)	0 – 85	[0 – 99]
Perceived stress (PSS)	20.22 (6.06)	4 – 36	[0 – 40]
Friendship Loneliness (SELSA-S)	12.31 (5.68)	5 – 34	[5 – 35]
Family Loneliness (SELSA-S)	10.88 (6.62)	5 – 34	[5 – 35]
Romantic Loneliness (SELSA-S)	21.38 (10.35)	5 – 35	[5 – 35]

Note. $N = 265$. All participants were female. [a]Body mass index calculated as kg/m^2. [b]HRV is reported in natural log units (ln ms^2). UCLA = UCLA Loneliness Scale; PSS = Perceived Stress Scale; BAI = Burns Anxiety Inventory; CESD-R = Center for Epidemiological Studies Depression Scale-Revised; SELSA-S = Social and Emotional Loneliness Scale for Adults-Short form.

Table 2

Sequential Regression Analysis Predicting Resting HRV

Variable	*b*	*SE b*	β	*t*	*p*	ΔR^2
Step 1						
Age	–0.03	0.03	–.06	–1.06	.291	
BMI	0.06	0.02	.19	3.49	.001	
Respiration	–7.03	0.95	–.41	–7.36	<.001	
Step 2						
Age	–0.04	0.03	–.08	–1.38	.168	
BMI	0.05	0.02	.18	3.29	.001	
Respiration	–6.81	0.95	–.39	–7.20	<.001	
Loneliness (UCLA)	–0.02	0.01	–.15	–2.75	.006	.02**

Note. $^{**}p < .01$; $N = 265$. BMI = body mass index calculated as kg/m^2; UCLA = UCLA Loneliness Scale.

Table 3

Sequential Regression Analysis Predicting HRV Reactivity to State Loneliness

Variable	*b*	*SE b*	β	*t*	*p*	ΔR^2
Step 1						
Age	–0.03	0.02	–.07	–1.12	.264	
BMI	–0.01	0.01	–.04	–0.62	.534	
Respiration	–5.02	0.80	–.36	–6.25	<.001	
Task effortfulness	–0.02	0.03	–.05	–0.86	.393	
Feedback plausibility	–0.01	0.03	–.01	–0.20	.843	
Step 2						
Age	–0.02	0.02	–.05	–0.92	.359	
BMI	–0.00	0.01	–.02	–0.35	.729	
Respiration	–4.91	0.80	–.35	–6.13	<.001	
Task effortfulness	–0.02	0.03	–.05	–0.79	.431	
Feedback plausibility	–0.03	0.03	–.06	–0.96	.339	
Loneliness (UCLA)	0.01	0.01	.14	2.21	.028	.02*

Note. *$p < .05$; $N = 265$. BMI = body mass index calculated as kg/m^2; UCLA = UCLA Loneliness Scale.

Table 4

Sequential Regression Analysis Predicting HRV Reactivity to Cognitive Challenge

Variable	*b*	*SE b*	β	*t*	*p*	ΔR^2
Step 1						
Age	0.01	0.02	.03	0.52	.606	
BMI	0.00	0.01	.00	–0.06	.950	
Respiration	–5.71	0.79	–.41	–7.19	<.001	
Task performance	–0.01	0.01	–.04	–0.72	.470	
Step 2						
Age	0.02	0.02	.05	0.92	.357	
BMI	0.00	0.01	.01	0.19	.848	
Respiration	–5.63	0.78	–.41	–7.20	<.001	
Task performance	–0.01	0.01	–.03	–0.58	.560	
Loneliness (UCLA)	0.01	0.01	.17	3.00	.003	.03**

Note. $^{**}p < .01$; $N = 265$. BMI = body mass index calculated as kg/m^2; UCLA = UCLA Loneliness Scale. Task performance is operationalized as the total number of correctly identified targets in the cognitive challenge.

Table 5

Correlations between Heart Rate Variability[a] (HRV), Demographics, and Health Behaviours

		1	2	3	4	5	6	7	8	9	10	11	12	13
1	Resting HRV (baseline)		-.56***	-.54***	-.04	.22***	-.12*	.01	.12*	-.03	.24***	.08	.15*	.11
2	HRV reactivity to state loneliness			.68***	-.05	-.05	.10	.03	-.05	.00	-.07	.03	-.09	-.11
3	HRV reactivity to cognitive challenge				.04	-.02	.10	.04	-.06	-.01	-.10	.05	-.15*	-.10
4	Age					.02	.18**	-.02	.00	.08	.05	.08	.14*	-.11
5	Body mass index[b]						-.16**	.00	-.04	-.15*	.08	-.09	.02	.06
6	Relationship status[c]							.03	.25***	.14*	.00	.03	.19**	.00
7	Medical condition[d]								.11	-.04	.02	.02	-.02	.13*
8	Hormonal contraceptive[e]									.13*	.20**	.19**	.27***	.25***
9	Sleep										.07	-.00	.16**	-.03
10	Exercise											.18**	.24***	.06
11	Caffeine												.29***	.07
12	Alcohol													.26***
13	Marijuana													

Note. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$; [a]HRV is reported in natural log units (ln ms^2). [b]Body mass index calculated as kg/m^2. [c]1 = single, 2 = dating, 3 = in committed relationship, not cohabiting with partner, 4 = in committed relationship, cohabiting with partner. [d]0 = no medical condition, 1 = unsure medical condition, 2 = medical condition. [e]0 = no hormonal contraceptive, 1 = hormonal contraceptive.

Figures

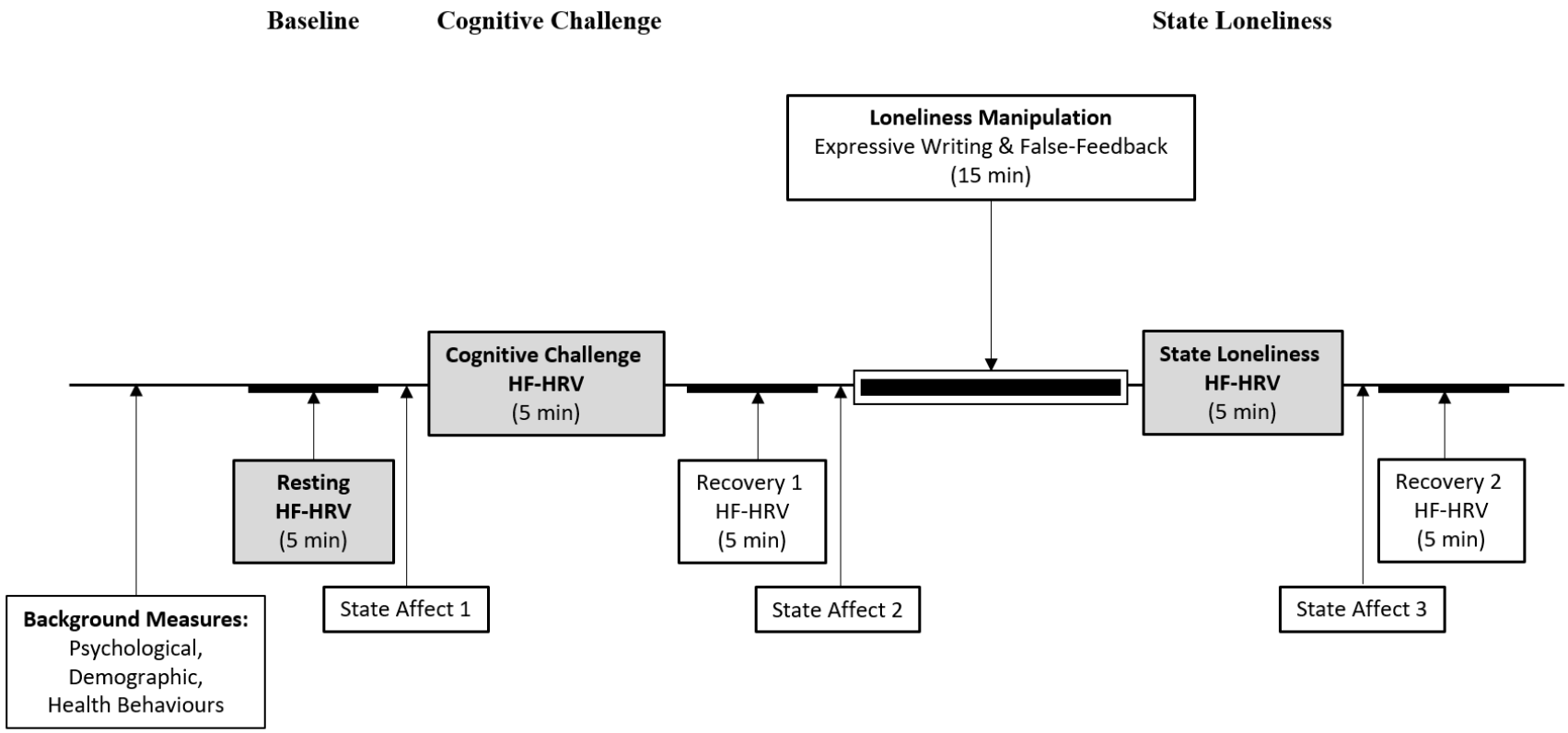

Figure 1. Timeline of study procedure.

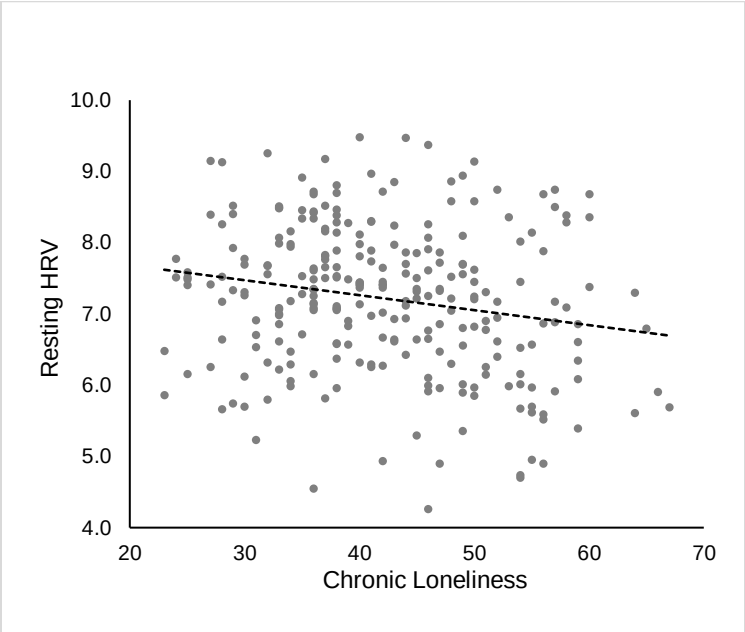

Figure 2. Linear regression predicting resting heart rate variability (HRV) from chronic loneliness. HRV is displayed in natural log units (ln ms^2).

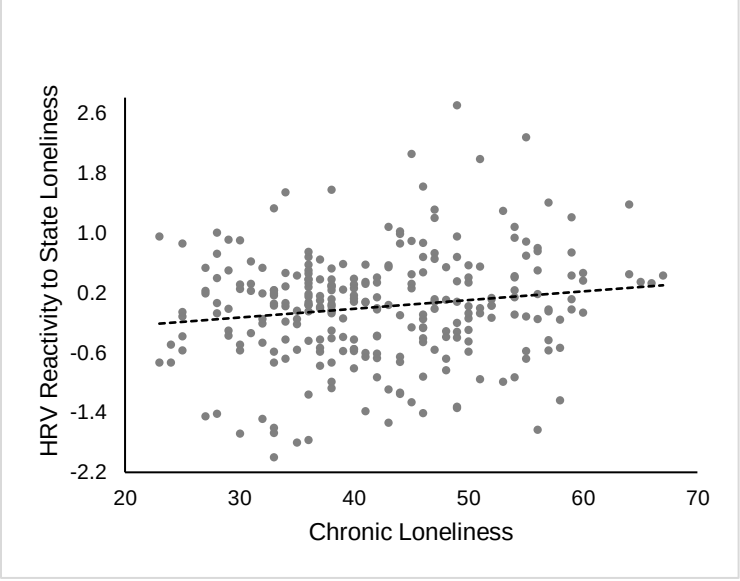

Figure 3. Linear regression predicting heart rate variability (HRV) reactivity to an experience of state loneliness from chronic loneliness. HRV is displayed in natural log units (ln ms^2).

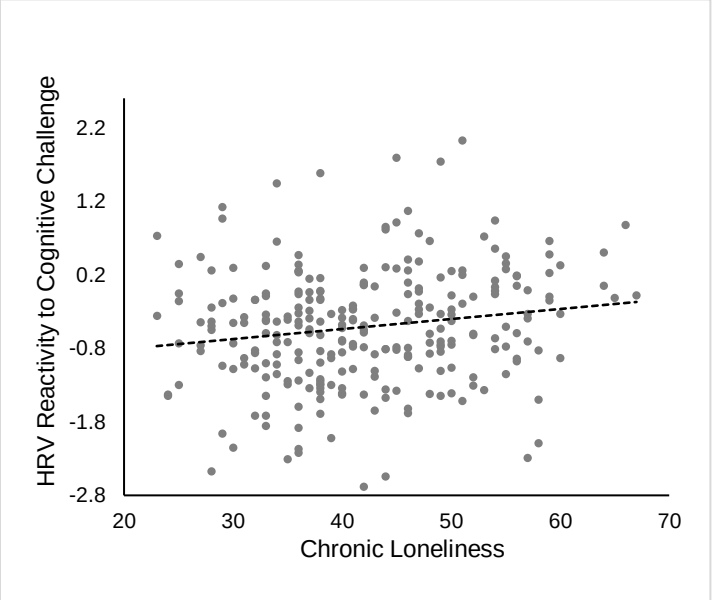

Figure 4. Linear regression predicting heart rate variability (HRV) reactivity to cognitive challenge from chronic loneliness. HRV is displayed in natural log units (ln ms^2).