LONELINESS AND THE HEART:
EXAMINING THE ASSOCIATIONS BETWEEN TRAIT LONELINESS,
STATE LONELINESS, AND HIGH-FREQUENCY HEART RATE VARIABILITY

by

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Abstract
Loneliness is a recognized risk factor for numerous adverse health outcomes, including early death. However, state loneliness may also be evolutionarily adaptive by signaling our social connection to others is at risk and motivating social reaffiliation. Long-term and short-term changes in vagal parasympathetic functioning may represent a mechanism by which both detrimental and beneficial effects of loneliness impact human physiology. The present study investigates the differential influences of trait loneliness and state loneliness on high-frequency heart rate variability (HF-HRV), an index of vagal parasympathetic activity. In controlled laboratory settings, HF-HRV in young women (N = 148) was monitored before, during, and after a cognitive challenge task, as well as before, during, and after an induction of state loneliness. Replicating and extending prior research, higher trait loneliness predicted blunted HF-HRV reactivity to cognitive demand, controlling for covariates. Higher trait loneliness also predicted blunted HF-HRV recovery following cognitive demand, although this appeared to be a function of initial blunted HF-HRV reactivity among the chronically lonely. Consistent with the evolutionary theory of loneliness, acute state loneliness was associated with increased HF-HRV above baseline levels, regardless of self-reported trait loneliness. During recovery from state loneliness, trait loneliness predicted change in HF-HRV, such that HF-HRV decreased in high trait-lonely women, whereas HF-HRV increased in low trait-lonely women. The current findings indicate that trait loneliness is associated with a potentially maladaptive physiological response to cognitive demand. The study also provides the first evidence of increased vagal parasympathetic activity during acute state loneliness, a potential indication of a physiological state conducive to social engagement behaviours. The findings further suggest that physiological capacity for social engagement may differ as a function of trait loneliness, immediately following an acute experience of loneliness. Finally, the utility of a robust loneliness induction paradigm developed from existing methods was demonstrated, supporting its application in future research seeking to disentangle trait and state loneliness.
Lay Summary

Loneliness increases the risks for many adverse health outcomes, including early death. However, short-lived feelings of loneliness may also motivate us to socially reconnect with others. The goal of this study was to examine the physiological effects of short-term and long-term feelings of loneliness. In a sample of 148 young women, long-term loneliness was associated with a potentially maladaptive pattern of parasympathetic nervous system (PNS) responses to a cognitively demanding task. Short-term loneliness was associated with increased PNS influence on the heart; this is thought to indicate a physiological state that supports social engagement behaviours. After a brief experience of loneliness, women who reported high feelings of loneliness in daily life showed lower PNS activity compared to less lonely women. Overall, this research provides evidence that short-term and long-term loneliness have different physiological effects, and that it is important for future research not to confuse long-term and short-term loneliness.
Preface

I am the primary author of the work presented in this thesis. I identified the research questions and designed the study. I was responsible for collecting, analyzing, and interpreting the data, as well as writing the manuscript. Dr. Frances Chen acted as supervisory author by providing guidance and feedback on concept formation, study design, data analysis, and manuscript revisions. This research was conducted with the permission of The University of British Columbia Office of Research Services Behavioural Research Ethics Board, certification number H18-00196, “HRV and Emotions Study”.
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Introduction

Perceived social isolation, more commonly known as loneliness, is an independent risk factor for a plethora of adverse physical and mental health outcomes, including: cardiovascular disease, impaired immune function, accelerated aging, cognitive decline, depression, and suicide (Cole, Hawkley, Arevalo, & Cacioppo, 2011; Goossens et al., 2015; Hawkley & Cacioppo, 2010; Wilson et al., 2018). Strikingly, cumulative evidence from longitudinal research indicates that the increased risk of early death associated with loneliness exceeds the risks of mortality associated with other well-established risk factors such as obesity, physical inactivity, hypertension, and air pollution (Cacioppo, Capitanio, & Cacioppo, 2014; Holt-Lunstad, 2017; Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015; Holt-Lunstad, Smith, & Layton, 2010; Xia & Li, 2018). However, the mechanisms through which loneliness—a subjective emotional experience—operate to impact physiology and health remain poorly understood—raising the question: How does loneliness get under the skin? Moreover, what function, if any, does loneliness serve? The present research investigates altered functioning of the parasympathetic nervous system as a potential pathway by which loneliness may affect health (Gouin, Zhou, & Fitzpatrick, 2015; Wilson et al., 2018; Xia & Li, 2018). Specifically, the differential influences of trait loneliness and state loneliness on high-frequency heart rate variability (an index of parasympathetic activity) are examined in light of current evolutionary perspectives on the adaptive function of loneliness, and on the phylogenetic development of the parasympathetic nervous system.

Loneliness

Loneliness has been conceptualized as the aversive affective and cognitive experience that arises from a perceived discrepancy between an individual’s desired and actual quality
and/or quantity of social relationships (Perlman & Peplau, 1981). Loneliness is experienced as distressing and is typically characterized by increased sadness, anxiety, anger, and tension, as well as by decreased happiness (Cacioppo et al., 2006; Heinrich & Gullone, 2006; Perlman & Peplau, 1981). A fundamental cognitive feature of loneliness is that lonely individuals perceive their relationships as failing to meet their expectations. These expectations may include desires for specific types of relationships (e.g., a romantic partner, best friends), or may involve desires for more frequent interactions or greater intimacy (de Jong-Gierveld, van Tilburg, & Dykstra, 2006). Such expectations can be shaped by a variety of influences beyond individual differences in personality, such as life stage, social norms, and cultural expectations. For example, the finding that North American high school students report feeling lonelier on Friday and Saturday nights compared to other weeknights is likely influenced by Western sociocultural norms for adolescents to socialize with their peers during these times (Larson, 1999). However, beyond examining the affective, cognitive, and sociocultural aspects of loneliness, it is also possible to conduct research into loneliness at the physiological level.

**High-Frequency Heart Rate Variability (HF-HRV)**

A biomarker widely used to assess physiology underlying psychological states is high-frequency heart rate variability (HF-HRV), an index of parasympathetic nervous system (PNS) activity (Balzarotti, Biassoni, Colombo, & Ciceri, 2017; Levenson et al., 2017). Heart rate variability (HRV) refers to the variations in time between consecutive heart beats. The high-frequency (HF) band of HRV occurs at the frequency of respiration (0.12–0.40 Hz), and is used to quantify the activity of the vagus nerve, a core component of the parasympathetic branch of the autonomic nervous system (Berntson, Quigley, Norman, & Lozano, 2017; Shaffer, McCraty, & Zerr, 2014). Parasympathetic activity, which predominates during periods of rest and calm,
acts via the rapid release (< 1 s) of acetylcholine by the vagus nerve to slow heart rate. Conversely, the sympathetic (SNS) branch operates through the relatively slow-acting (> 5 s) neurotransmission of norepinephrine to accelerate heart rate and mobilize metabolic resources to meet the energy demands required of a given situation (Nunan, Sandercock, & Brodie, 2010; Shaffer et al., 2014). During inhalation, vagal outflow (i.e., PNS influence, via the vagus nerve, on the heart) is inhibited, causing heart rate to accelerate. During exhalation, vagal outflow is restored, decreasing heart rate. As the slower-acting SNS cannot follow these respiratory rhythms in heart rate variation (known as respiratory sinus arrhythmia), SNS activity is effectively filtered out of the respiratory (i.e., high-frequency) band. For this reason, high-frequency heart rate variability (HF-HRV) is considered to index vagally-mediated PNS activity, or PNS outflow to the heart (Berntson et al., 2017; Shaffer et al., 2014). Specifically, increased HF-HRV indicates greater PNS activity, whereas decreased HF-HRV indicates reduced PNS activity. Importantly, an increase in PNS activity does not inevitably imply a concomitant decrease in SNS activity. In addition to acting reciprocally, the PNS and SNS branches of the autonomic nervous system can coactivate, co-inhibit, as well as change outflows independently of one another (Berntson, Norman, Hawkley, & Cacioppo, 2008).

Methodologically, measurement of HF-HRV may involve assessing tonic HF-HRV, and/or phasic HF-HRV. Tonic HF-HRV (also termed vagal tone) refers to the measurement of HF-HRV at one time point, and is the quantification of PNS influence on the heart at that time point (Laborde, Mosley, & Thayer, 2017). Typically, the measurement of tonic HF-HRV involves taking a 5-min\textsuperscript{1} recording of HF-HRV during a resting baseline (when it may be referred to as baseline HF-HRV, or resting HF-HRV). However, tonic HF-HRV can also be

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\textsuperscript{1} 1 min is considered the absolute minimum duration acceptable for a valid assessment of HF-HRV (Malik, 1996).
measured during experimental conditions, such as during a stress- or emotion- induction task. In contrast, phasic HF-HRV refers to the change in HF-HRV between two time points, and reflects dynamic changes in PNS influence on the heart. Phasic HF-HRV is usually calculated as a change score, for example, by subtracting the value obtained for tonic HF-HRV at rest (e.g., “baseline HF-HRV”), from the value obtained for tonic HF-HRV during an experimental condition (e.g., “task HF-HRV”). The term \textit{HF-HRV reactivity} refers to the phasic difference in HF-HRV between “baseline” and “task”, whereas the term \textit{HF-HRV recovery} refers to the phasic difference in HF-HRV between “task” and “post-task”. Obtaining a phasic HF-HRV value (i.e., a change score) that is negative indicates a decrease in PNS influence on the heart; this is referred to as \textit{vagal withdrawal}. An obtained phasic HF-HRV value that is positive indicates an increase in PNS influence on the heart, and is referred to as \textit{vagal augmentation}.

\textbf{Linking Loneliness and HF-HRV}

Although the two most influential biobehavioural models of HF-HRV—polyvagal theory (Porges, 2007) and the neurovisceral integration model (Thayer & Lane, 2009)—both support a link between loneliness and HF-HRV, such a link has not yet been clearly established (Balzarotti et al., 2017). Previous research has mostly focused on associations between loneliness and tonic HF-HRV (Cacioppo et al., 2002; Gouin et al., 2015; Hawkley, Burleson, Berntson, & Cacioppo, 2003; Hawkley, Masi, Berry, & Cacioppo, 2006), which may account for the null findings in these studies. However, the assessment of phasic HF-HRV in loneliness research may be crucial for detecting individual differences. For example, in studies of loneliness and impaired cognition, individual differences in HF-HRV may only be observable in response to cognitively demanding tasks, and may thus go undetected if measurement is restricted to tonic HF-HRV.
Muhtadie, Koslov, Akinola, and Mendes (2015) have recently made a compelling argument for the use of more dynamic measures of HF-HRV. In a series of studies, Muhtadie et al. (2015) found that phasic HF-HRV provided unique and socially specific information beyond that provided by the more commonly used measure of tonic HF-HRV. In one of these studies, loneliness, as measured on the UCLA Loneliness Scale (Version 3; Russell, 1996), was uniquely associated with HF-HRV reactivity to a cognitive challenge task (i.e., phasic HF-HRV). Specifically, greater loneliness was associated with blunted HF-HRV reactivity (i.e., smaller change in phasic HF-HRV). However, loneliness was not associated with baseline HF-HRV (i.e., tonic HF-HRV). In contrast, the opposite pattern was observed for depression, anxiety, and perceived stress, which were all associated with tonic HF-HRV but were not associated with phasic HF-HRV.

Muhtadie et al. (2015) propose that this pattern of findings is evidence that phasic HF-HRV has different associations with psychological characteristics than does tonic HF-HRV. Specifically, individual differences in phasic HF-HRV are argued to reflect differences in sensitivity to the social environment. Phasic HF-HRV is posited to be uniquely associated with social or *inter*individual experiences—such as loneliness, whereas tonic HF-HRV is posited to be associated with *intra*individual experiences—such as depression (Muhtadie et al., 2015). Accordingly, the relationship between loneliness and HF-HRV may have been previously obscured by the exclusive use of tonic measures of HF-HRV in earlier studies.

Although incorporating HF-HRV reactivity into recent research has helped uncover an exciting new link between loneliness and phasic HF-HRV, current HRV research recommendations call for more complete assessments of HF-HRV to advance progress in the field and facilitate comparison of results across studies (Balzarotti et al., 2017; Laborde et al.,
Concretely, this requires taking three measures of tonic HF-HRV: at baseline, task, and post-task; in addition to two measures of phasic HF-HRV: between baseline and task (i.e., HF-HRV reactivity), and between task and post-task (i.e., HF-HRV recovery) (see Laborde et al., 2017). The assessment of HF-HRV recovery, in particular, has been neglected (Balzarotti et al., 2017; Cui et al., 2015). As such, it is still unknown whether loneliness predicts individual differences in HF-HRV recovery from cognitive challenge, a potentially important marker of both psychobiological dysfunction and health outcomes (Balzarotti et al., 2017; Steptoe & Marmot, 2005).

Curiously, the recent findings on loneliness and phasic HF-HRV also revealed a discrepancy within research on the relationship between loneliness and sensitivity to social context. The current interpretation of Muhtadie and colleagues’ (2015) observation that higher loneliness is associated with blunted HF-HRV reactivity, is that greater loneliness predicts reduced sensitivity to the social environment—such as an impaired ability to accurately detect social cues (Human & Mendes, 2018; Muhtadie et al., 2015). This interpretation appears to directly contradict earlier findings by Gardner, Pickett, Jefferis, and Knowles (2005), that greater self-reported loneliness is associated with heightened sensitivity to the social environment—including more accurate detection of both positively- and negatively-valenced social cues (e.g., decoding of facial expressions). However, these apparently conflicting findings may not be incompatible if an important, but relatively neglected, dimension of loneliness (Shiovitz-Ezra & Ayalon, 2010)—the duration of loneliness—is taken into account. That is, short-term or state loneliness may enhance sensitivity to social information, whereas long-term or trait loneliness may impair sensitivity to social information.
Specifically, trait loneliness is linked to impairments in both lower-order and higher-order cognitive functioning (Cacioppo et al., 2014; Wilson et al., 2007). Trait loneliness has also been associated with genes linked to faulty cognitive processing, including genes related to sensitivity to social and emotional stimuli (Qualter et al., 2015). Vagal withdrawal (i.e., decrease in HF-HRV) is considered an adaptive response to lower-order cognitive demand, as it should facilitate mobilization of metabolic resources required to meet the demand (Laborde et al., 2017; Porges, 2007). Weaker vagal withdrawal (i.e., blunted decrease in HF-HRV) is considered less adaptive, and indeed, blunted HF-HRV reactivity to cognitively demanding tasks has been found to predict poorer cognitive performance (Human & Mendes, 2018).

This research on trait loneliness corresponds with Muhtadie et al.’s finding in a diverse community sample (mean age of 44.5 years), that lonelier individuals display more blunted HF-HRV reactivity to cognitive challenge (2015). In comparison, in the study by Gardner et al. (2005) in which higher loneliness was associated with greater sensitivity to the social environment, the participants involved were much younger college students (mean age not reported). Importantly, Gardner et al. (2005) point out that they were unable to determine whether their participants were reporting short-term, situational loneliness (e.g., caused by moving to a new city) or longer-term, more stable loneliness. Consequently, the possibility cannot be ruled out that these participants were experiencing short-term or state loneliness, which—according to an evolutionary perspective of loneliness—may have heightened, rather than impaired, their sensitivity to their social environment. Therefore, it is not implausible that unmeasured temporal differences in loneliness are impeding an accurate understanding of the underlying nature and function of loneliness.
State and Trait Loneliness

The importance of distinguishing between state loneliness (also referred to as “situational”, “transient”, or “short-term” loneliness) and trait loneliness (sometimes referred to as “chronic”, “prolonged”, or “long-term” loneliness) has not gone unremarked (e.g., Jones, 1987; Qualter et al., 2015; Shaver, Furman, & Buhrmester, 1985; Shiovitz-Ezra & Ayalon, 2010; Vanhalst et al., 2015). State loneliness is generally understood to refer to transient feelings of loneliness which are likely to be situationally determined, whereas trait loneliness refers to enduring experiences of loneliness that may have dispositional, rather than situational, causes (Heinrich & Gullone, 2006; Shaver et al., 1985). However, empirical studies over the past quarter century have largely not discriminated between state and trait loneliness, with the preponderance of research utilizing measures skewed toward assessing trait loneliness (Marangoni & Ickes, 1989; van Roekel et al., 2018). As a consequence, very little is known about state loneliness (van Roekel et al., 2018). More importantly, this conflation of concepts may be obscuring critical differences in the prevalence, etiologies, and consequences of short-term and long-term loneliness. For example, although measures of higher trait loneliness are consistently associated with adverse health outcomes, it is unclear whether transient feelings of loneliness should also be considered as potentially detrimental to health.

As an important theoretical dimension, chronicity of loneliness was widely recognized in the 1980s (Cutrona, 1982; Hojat, 1983; Jones, 1987; Marangoni & Ickes, 1989; Perlman & Peplau, 1984; Shaver et al., 1985). However, it is only in the last few years that a resurgence of interest in the conceptual distinction between state and trait loneliness has become apparent, reflected by an increasing number of studies that have begun to (re-)address temporal differences in loneliness (Shiovitz-Ezra & Ayalon, 2010; Tam & Chan, 2019; Vanhalst et al., 2015; van
Roekel et al., 2018). For example, Vanhalst and colleagues (2015) have proposed that the duration of loneliness plays a central role in determining how a new social situation will be perceived and responded to by a lonely individual. Specifically, among lonely adolescents, Vanhalst et al. (2015) found that hypervigilance to social exclusion and hypovigilance to social inclusion was only present in adolescents who had experienced persistent loneliness throughout adolescence. Although the number of studies examining state and trait loneliness has grown, no research has yet specifically examined the physiology underlying these distinctions in loneliness. Nevertheless, an evolutionary perspective may offer relevant insights on circumstances in which loneliness may be beneficial, rather than harmful.

According to the evolutionary model of loneliness (Cacioppo et al., 2006), feelings of loneliness may be a biological adaptation that promotes survival in social species by activating motivation for social affiliation (known as the reaffiliation motive or RAM; Qualter et al., 2015) in response to perceived social threats (Cacioppo et al., 2015). Specifically, by forming social connections with conspecifics and thus deriving the benefits of mutual protection and assistance, individuals are able to increase their chances of survival and opportunities to pass on their genes. In contrast, social isolation, whether objective or perceived (for example, being among others whom one feels unable to trust, rely on, or confide in), increases an individual’s risk of injury or death (Cacioppo, Cacioppo, & Boomsma, 2014). Analogous to the biological mechanism of physical pain that alerts an individual to injury and motivates actions to protect one’s physical body, loneliness is posited to be an aversive signal that is activated when discrepancies are detected between an individual’s actual and preferred social environment (Cacioppo et al., 2015). In this way, feelings of loneliness are argued to warn of damage or threats to one’s “social
body”, motivating individuals to initiate or repair their social relationships, and thereby protect themselves from the dangers associated with social isolation.

However, in addition to motivating an explicit desire for social connection, loneliness is thought to concurrently trigger a battery of neural and behavioural responses that help to protect the individual in times of perceived social isolation (i.e., when the individual is without mutual assistance and protection). Together, these responses constitute a self-preservation mode characterized by implicit hypervigilance for threat (Cacioppo et al., 2014). Although a self-protective stance activated by loneliness may be adaptive in the short term, it could be detrimental for an individual to remain in a state of perceived social isolation for an extended period (Cacioppo et al., 2014). Included among the neural and behavioural effects triggered in this self-preservation mode are outcomes that have been reported in studies of lonely older adults, such as heightened anxiety and hostility (Domènech-Abella, Mundó, Haro, & Rubio-Valera, 2019; Hawkley, Thisted, Masi, & Cacioppo, 2010), increased sleep fragmentation (to reduce risk of predation during sleep) (Jacobs, Cohen, Hammerman-Rozenberg, & Stessman, 2006; Kurina et al., 2011), and elevated hypothalamic pituitary adrenal (HPA) axis activity (Adam, Hawkley, Kudielka, & Cacioppo, 2006; Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004).

These findings suggest that whereas trait, or chronic, loneliness may increase an individual’s risk for adverse health outcomes, state loneliness may actually confer important benefits (i.e., increased desire for social connection, heightened monitoring for potential threats), in the right circumstances. However, the evolutionary model also raises an important question: Given that loneliness is posited to provoke a vigilant, defensive mode via the activation of a range of neurophysiological effects, how does loneliness concurrently increase motivation for
social connection? As the immediate effects of state loneliness on physiology have not been examined, an important remaining gap in the literature is to investigate what physiological effects occur during an acute experience of loneliness.

**Social Affiliation and the Parasympathetic Nervous System**

That state loneliness may play an evolutionarily adaptive role by motivating individuals to socially reengage follows from the conceptual distinction between state and trait loneliness. Moreover, such an association between motivation for social affiliation as a function of state loneliness and vagally-mediated parasympathetic activity is supported by theoretical models, as well as empirical research. Indeed, the vagus nerve, through its structural and functional connections between the heart and the brain, has long been implicated in the regulation of emotional responses to the social environment (e.g., Bernard, 1867; Darwin, 1872/2009).

Much current research on HF-HRV is informed by polyvagal theory (Porges, 2001, 2003, 2007, 2011), an influential biobehavioural model that relates autonomic functioning to behaviour. Polyvagal theory posits that the myelinated component of the vagus nerve connecting the brainstem with the sinoatrial node of the heart (i.e., the pacemaker), emerged during the phylogenetic development of the mammalian parasympathetic nervous system (PNS). This component of the vagus nerve is thought to facilitate social affiliative and engagement behaviours as part of an integrated *social engagement system* that links brainstem nuclei, PNS influence on the heart, and the striated muscles of the face (Porges, 2003, 2007). These anatomical connections of the social engagement system enable the coordination of physiological states with behaviours that support social engagement responses (Porges, 2003). Specifically, the vagus nerve interlinks with neural pathways that innervate facial muscles necessary for social communication, such as the muscles responsible for facial expressions,
gazing, nodding, turning the head, distinguishing the human voice, and aspects of human speech (e.g., intonation) (Porges, 2007).

In addition, by modulating PNS influence on the heart, the vagus nerve facilitates different types of behavioural responses by rapidly adjusting physiological states. This modulation of PNS influence on the heart allows for flexible adaptation to the environment. For example, an increase in PNS influence, or *vagal augmentation*, can support social affiliative and social engagement behaviours generally adaptive to states of calm and rest (Porges, 2007). This suggests that situations of perceived social isolation, or state loneliness, should be associated with increased vagally-mediated PNS influence—as quantified by increased HF-HRV—reflecting a physiological state that supports motivation for social affiliation (i.e., the reaffiliation motive) and social engagement. Importantly, such an increase in PNS influence could occur independently of changes in SNS outflow, which may explain how loneliness can concurrently activate both the reaffiliation motive and hypervigilance for threat. However, these effects have not yet been empirically documented.

Furthermore, based on work into the neurophysiological interconnections between the myelinated vagus, brain stem nuclei involved in regulating social behaviour, and oxytocin receptors in cardiac tissue and the amygdala (Davis & Whalen, 2001; Gamer, Zurowski, & Büchel, 2010; Jankowski et al., 2004; Porges, 2011; Tribollet, Dubois-Dauphin, Dreifuss, Barberis, & Jard, 1992), other researchers have explicitly proposed HF-HRV to be a biomarker of an individual’s motivation for social affiliation and social engagement (Quintana, Kemp, Alvares, & Guastella, 2013). This argument has been supported by the empirical finding that administration of intranasal oxytocin, a mammalian neuropeptide centrally implicated in human social bonding (Carter, Williams, Witt, & Insel, 1992), increased HF-HRV in a placebo-
controlled, within-subjects experiment (Kemp et al., 2012). These results were interpreted as reflecting an increase in participants’ motivation for social approach and capacity for social engagement (Kemp et al., 2012; Kemp & Quintana, 2013).

Moreover, in a double-blind, between-subjects experiment, intranasal oxytocin administration was observed to concurrently increase both cardiac PNS influence and SNS influence, indexed by HF-HRV and pre-ejection period (PEP), respectively (Norman et al., 2011). Interestingly, this effect was moderated by trait loneliness, such that chronically lonelier individuals displayed a selectively blunted (i.e., smaller) increase of PNS activation. Combined, these findings suggest that, similar to the increased “social bonding” effects of oxytocin on HF-HRV and on social-approach motivation, state loneliness may likewise be associated with increased HF-HRV, reflecting both greater PNS influence and greater motivation for social affiliation.

The physiological implications of state loneliness on PNS activity currently remain unclear, as no prior research has examined the direct effect of state loneliness on HF-HRV. Furthermore, the handful of studies that have investigated associations between loneliness and HF-HRV have exclusively employed measures of trait loneliness (Cacioppo et al., 2002; Gouin et al., 2015; Hawkley et al., 2003; Hawkley et al., 2006; Muhtadie et al., 2015; Norman et al., 2011). This circumscribed focus may present another possible explanation for the absence of observed effects. The lack of research on state loneliness is particularly concerning as it is unclear whether trait loneliness may be the result of multiple prolonged experiences of state loneliness (Heinrich & Gullone, 2006; Shaver et al., 1985).

Although loneliness did not predict SNS activation, Norman et al. (2011) note that their null findings may be due to low power as the relationship approaches significance, \( p = .10 \).
Potential Associations between State and Trait Loneliness

As suggested by the contradictory findings of Gardner et al. (2005) and Muhtadie et al. (2015), the potential conflation of state and trait loneliness may be obscuring critical insights into the antecedents, time course, and consequences of loneliness. Thus, in addition to investigating individual differences in trait loneliness on HF-HRV recovery to cognitive challenge, and examining the immediate physiological effects of state loneliness, there is a clear imperative for further research that examines both state and trait loneliness simultaneously.

Given the challenges associated with manipulating loneliness, prior research into potential moderating or mediating associations between state and trait loneliness has been sparse. Indeed, only a few studies have directly manipulated loneliness (Cacioppo et al., 2006; Hu, 2007; Lamster, Nittel, Rief, Mehl, & Lincoln, 2017; Wildschut, Sedikides, Arndt, & Routledge, 2006; Zhou, Sedikides, Wildschut, & Gao, 2008). By modifying existing methods used to induce loneliness and by applying rigorous manipulation checks, we developed a strengthened variant of a previously used loneliness-induction paradigm. The present study was thus able to more directly examine potential associations between state and trait loneliness than has previously been possible.

Overview and Hypotheses

The purpose of the present study was to address gaps in the literature on the effects of state loneliness and trait loneliness on vagally-mediated parasympathetic nervous system (PNS) functioning. Trait loneliness was measured using the UCLA Loneliness Scale (Version 3; Russell, 1996), and high-frequency heart rate variability (HF-HRV) was used to index vagally-mediated PNS activity. Under controlled conditions in the laboratory, individuals’ HF-HRV was recorded before, during, and after a cognitive challenge task, as well as before, during, and after
an acute experience of state loneliness. The main research objectives were to (a) replicate and extend the findings of Muhtadie et al. (2015) on trait loneliness and HF-HRV reactivity to cognitive challenge, (b) examine the direct effect of state loneliness on HF-HRV, and (c) conduct exploratory analyses on individual differences in trait loneliness both during and after an experience of state loneliness.

Based on evidence that trait loneliness is related to impaired cognitive processing (Cacioppo et al., 2014), and that blunted HF-HRV reactivity is linked to poorer cognitive task performance (Human & Mendes, 2018), it was expected that (H1a) higher trait loneliness predicts blunted HF-HRV reactivity to cognitive challenge, and that (H1b) higher trait loneliness predicts blunted HF-HRV recovery, or absence of HF-HRV recovery, following cognitive challenge. In line with the evolutionary model of loneliness (Cacioppo et al., 2014) and polyvagal theory (Porges, 2007), it was also hypothesized that (H2) state loneliness is associated with increased vagally-mediated PNS activity, indexed by greater HF-HRV during an acute experience of loneliness, compared to baseline HF-HRV. Furthermore, given the lack of physiological research distinguishing between trait and state loneliness, coupled with the finding that the effect of oxytocin on HF-HRV is moderated by trait loneliness (Norman et al., 2011), exploratory analyses were conducted to determine whether HF-HRV differs between high trait-lonely individuals and low trait-lonely individuals during an experience of state loneliness (H3a) and/or immediately following state loneliness (H3b).
Method

Participants

To control for age and sex differences in heart rate variability (Dart, Du, & Kingwell, 2002; Sammito & Böckelmann, 2016; Smith et al., 2011; Zhang, 2007), and to retain sufficient statistical power to detect an effect, we restricted recruitment to women between the ages of 18 and 30 years. Participants were excluded based on criteria established prior to data collection that were derived from recommended heart rate variability (HRV) research guidelines (Berntson et al., 1997; Laborde et al., 2017; Quintana, Alvares, & Heathers, 2016). Specifically, individuals were ineligible if they reported: having a heart condition, taking cardioactive medication, habitual smoking (defined as smoking once or more per day), or habitual marijuana use (defined as using marijuana once or more per day). Participants were recruited from the University of British Columbia Human Subject Pool and received course credit for participation. The study protocol was approved by the university’s research ethics board.

Using an effect size estimate from prior research on the association between loneliness and HF-HRV reactivity \((r = .25; \text{Muhtadie et al., 2015})\), expected power calculations with the \textit{fabs} package for R (\texttt{github\jbiesanz\fabs}; Biesanz, 2018) indicated that a sample of 173 participants was required for sufficient power \((1 - \beta = .80)\) to detect a similar effect. Between September 2018 and November 2018, a total of 177 participants were recruited. However, it was necessary to exclude data from 29 participants, leaving a final sample of 148 women \((M_{\text{age}} = 19.97 \text{ years}, SD = 1.70, \text{ age range: 18–27}; \text{see Table 1 for sample descriptives})\). Specifically, participants were excluded for the following: taking cardioactive medication \((n = 14)^3\), having

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3 Specifically, the cardioactive medications were: apraprazole (1), bupropion (3), citalopram (3), escitalopram (1), dextroamphetamine-amphetamine (1), methylphenidate (1), paroxetine (1), quetiapine (1), sertraline (1), and venlafaxine (1).
corrupted or missing ECG data ($n = 12$), reporting a chronic heart condition ($n = 1$), reporting daily marijuana use ($n = 1$), and being over age 30 ($n = 1$). Although this sample was smaller than planned, the expected power for the current study with 148 participants, after including the uncertainty of the initial effect size estimate ($r = .25$; Muhtadie et al., 2015) into the analysis, is $.77$ (refer to Biesanz & Schrager, 2017; McShane & Bockenholt, 2016; and the R.fabs package: github\jbiesanz\fabs).

**Procedure**

All participants provided written informed consent at the beginning of the study. Each participant first completed a series of online questionnaires assessing psychosocial, demographic, and health behaviour variables before attending an individual 90 min lab session within the following week. Participants were asked to avoid vigorous exercise and to refrain from consuming food, caffeine, and alcohol for 2 hr prior to the lab session. All sessions were scheduled between 11:30 a.m. and 5:30 p.m. to minimize the influence of diurnal fluctuations in HF-HRV (Bonnemeier et al., 2003).

On arrival at the lab, 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 spent approximately 5 min filling out a questionnaire that assessed behaviour in the preceding 24 hr (e.g., caffeine consumption prior to the lab visit, hours of sleep the previous night)\(^4\). This time also allowed participants to acclimate to the lab environment. All participants subsequently completed a 5-min baseline resting period

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\(^4\) This questionnaire was also used as a compliance check of study protocol requests prior to the lab session. Self-reported behaviours in the 24 hr preceding the lab session were tested for associations with the main study variables; no significant associations were found. Similarly, time of day and room temperature had no effect on any study analyses.
(“Baseline 1”), following which they performed a 5-min cognitive challenge task (“Cognitive task”). Next, participants completed a second 5-min resting period (“Baseline 2”), to allow HF-HRV to return to baseline levels before undergoing a 15-min loneliness manipulation. Participants were then asked to reflect on an experience of loneliness for 5 min (“Loneliness task”), after which they completed a final 5-min resting period (“Recovery”). At the end of the session, height and weight were recorded, and participants were debriefed. Participants completed all tasks (including resting 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 timeline for the study procedure is presented in Figure 1.

**Measures**

**Psychosocial variables.** Several psychosocial variables were assessed using online questionnaires prior to the lab session. The same measures of stress, anxiety, depression and trait loneliness that were used by Muhtadie et al. (2015) in their study of the association between loneliness and HF-HRV reactivity were employed. Also included were measures of psychosocial variables associated with loneliness, specifically: perceived social support, social engagement, social anxiety, self-esteem, and adult attachment style (Cacioppo et al., 2006; Heinrich & Gullone, 2006), to assess if these variables affected the relationship between loneliness and HF-HRV.

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5 In addition to the psychosocial measures reported in the main text, the social interaction subscale of the DSSI (Duke Social Support Index; Wardian, Robbins, Wolfersteig, Johnson, & Dustman, 2013) was used to assess objective social isolation. However, internal consistency for the subscale was unacceptable (Cronbach’s α = .42), and this measure was excluded from further analyses. The SELSA-S (short form Social and Emotional Loneliness Scale for Adults; DiTommaso, Brannen, & Best, 2004) was also administered to participants for use in exploratory analyses unrelated to the current thesis.
**Trait loneliness.** The UCLA Loneliness Scale (Version 3; Russell, 1996) assesses feelings of loneliness or subjective social isolation, and is commonly used as a measure of trait loneliness (Heinrich & Gullone, 2006; Hu, 2007; Marangoni & Ickes, 1989; Tam & Chan, 2019; van Roekel et al., 2018). 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 $\alpha = .93$).

**Stress.** The 10-item Perceived Stress Scale (PSS; Cohen & Williamson, 1988) 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 (Cronbach’s $\alpha = .82$).

**Anxiety.** Anxiety was assessed with the Burns Anxiety Inventory (BAI; Burns & Eidelson, 1998), 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*). This scale had excellent internal reliability in the present sample (Cronbach’s $\alpha = .95$).

**Depression.** The Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977) assesses current levels of depressive symptomology, with a focus on depressed mood. The CESD was designed for use in studies of the relationships between depression and other variables in the general population (Radloff, 1977). 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 was excellent in this sample (Cronbach’s $\alpha = .94$).

**Social support.** Perceived social support was measured using the short form Interpersonal Support Evaluation List (ISEL-12; Cohen, Memelstein, Kamarck, & Hoberman, 1985). The ISEL-12 assesses perceived availability of three types of social support: *tangible* (availability of material aid), *appraisal* (availability of someone to turn to for advice or guidance), and *belonging* (availability of people to spend time with) (Cohen et al., 1985). For each of 12 items, individuals are asked to rate the extent to which they agree with a self-descriptive statement on a 4-point scale from 0 (*definitely false*) to 3 (*definitely true*). Internal reliability for the overall scale was high in the current study (Cronbach’s $\alpha = .86$).

**Social engagement.** The short form Lubben Social Network Scale (LSNS-6; Lubben et al., 2006) is used to measure levels of social engagement. It is comprised of six questions that assess frequency of social interactions with family and friends on a 6-point scale from 0 (*zero*) to 5 (*nine or more*). In the present sample, internal consistency for the LSNS-6 was good (Cronbach’s $\alpha = .83$).

**Social anxiety.** The 20-item Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998), assesses concerns and distressing feelings related to meeting and talking with other people. For each item, respondents indicate the degree to which they feel that the statement is characteristic or true of themselves on a 5-point scale from 0 (*not at all*) to 4 (*extremely*). Internal reliability for the SIAS was excellent in this study (Cronbach’s $\alpha = .94$).

**Self-esteem.** The Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965) assesses overall perception of self-worth by measuring positive and negative feelings about the self. Individuals rate the extent to which they agree with each of 10 statements on a 4-point scale
ranging from 0 (strongly disagree) to 3 (strongly agree). This measure had high internal consistency in the present sample (Cronbach’s $\alpha = .90$).

**Adult attachment style.** The 12-item short form Experiences in Close Relationship Scale (ECR-S; Wei, Russell, Mallinckrodt, & Vogel, 2007) is a measure of adult attachment that contains six items to assess attachment avoidance (fear of interpersonal intimacy and dependence) and six items to assess attachment anxiety (fear of interpersonal rejection or abandonment). For each item, respondents rate their agreement with a self-descriptive statement on a 7-point scale from 1 (strongly disagree) to 7 (strongly agree). In the current sample, internal reliability was good for the attachment avoidance subscale (Cronbach’s $\alpha = .82$) and was acceptable for the attachment anxiety subscale (Cronbach’s $\alpha = .71$).

**State affect.** Participants completed an 8-item measure of state affect at three points during the lab session: following the first two resting periods and immediately after the 5-min loneliness experience. 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?”

**High-frequency heart rate variability (HF-HRV) measures.** Recommended HRV research guidelines were followed in the selection and implementation of all equipment and procedures (Berntson et al., 1997; Laborde et al., 2017). Electrocardiograph (ECG) activity was recorded continuously using the Bittium Faros 180 system (Oulu, Finland) at a sampling rate of 1000 Hz. Disposable Ag/AgCl wet gel electrodes (Ambu BlueSensor VLC; Copenhagen, Denmark) 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 (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014). A combination of automated and visual approaches to artifact correction was applied (as recommended by Berntson et al., 1997). In addition to identifying recording artifacts (e.g., ectopic beats, excessive movement) using an automated algorithm (Kubios HRV 3.1 automatic correction; Tarvainen, Lipponen, Niskanen, & Ranta-Aho, 2018), 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 0.08% (SD = 0.16%). 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 (Mateo & Laguna, 2000). The data were detrended using the smoothness priors method (smoothing parameter λ = 500; Tarvainen, Ranta-Aho, & Karjalainen, 2002), and submitted to a Fast Fourier Transform (FFT)\(^6\) to determine spectral power values (in ms\(^2\)/Hz). HF-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 (Berntson et al., 2008; Shaffer & Ginsberg, 2017). Respiration rate was obtained using ECG derived respiration (EDR), which estimates respiratory frequency from changes in R-wave amplitude (Tarvainen et al., 2018).

A mean HF-HRV score for each of the five recording periods (Baseline 1, Cognitive task, Baseline 2, Loneliness task, Recovery; see Figure 1 for study timeline) was obtained for each participant by aggregating the HF-HRV values of 1-min epochs during each recording period.

\(^6\) Autoregressive modelling (AR) was also employed to obtain an additional measure of HF-HRV with which to compare against the main analyses that used HF-HRV derived by FFT. All FFT- and AR-derived measures were highly correlated (all rs > .95, all ps < .001), and produced almost identical results in all study analyses; thus only results using HF-HRV derived by FFT are reported here.
Change scores were calculated by subtracting a participant’s mean HF-HRV score for a preceding task from her mean HF-HRV score of the subsequent task. For example, HF-HRV recovery from the loneliness experience was calculated as a change score by subtracting the HF-HRV score obtained during the loneliness experience from the HF-HRV score obtained during the resting period that followed the loneliness experience.

**Cognitive challenge.** To replicate and extend earlier findings on HF-HRV reactivity and trait loneliness (Muhtadie et al., 2015), the same visual cognition task (Cavanagh & Alvarez, 2005) used to elicit HF-HRV reactivity in the prior research was employed in the current study. Importantly, this multiple-object tracking task: (a) has been previously demonstrated to elicit HF-HRV reactivity (specifically, vagal withdrawal, or a phasic decrease in HF-HRV; Hagan et al., 2017; Human & Mendes, 2018; Muhtadie et al., 2015), (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 the 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 after all the dots had ceased moving, the participant would attempt to identify the target dots by clicking on them with the computer mouse. The task became increasingly difficult as the trials progressed, with the number of target dots increasing after every four trials. On average, participants took
approximately 5 min to complete the cognitive challenge task ($M = 4.87$ min, $SD = 0.53$). This task was used to assess HF-HRV reactivity to, and HF-HRV recovery from, cognitive challenge.

**State loneliness manipulation.** State loneliness was induced using a variation on a loneliness manipulation paradigm that has been reported to have successfully induced state loneliness in previous studies (Hu, 2007; Lamster et al., 2017; Twenge, Baumeister, Tice, & Stucke, 2001; Wildschut et al., 2006; Zhou et al., 2008). 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, Booth, Boyd, and Francis (2015). 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 [the University of British Columbia] 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 a computer screen which prompted her to write about an experience of loneliness (see Appendix for screenshots and additional details of the loneliness manipulation). The participant was given 10 min to type a description of her loneliness experience into a text box, following which the experimenter re-entered the room and sat down opposite the participant (so that the computer screen was not
visible to the experimenter). Next, the experimenter prompted the participant to “submit” her writing sample, ostensibly for analysis by the LAWC program, by clicking a computer key. A pre-programmed page then appeared on the screen 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 experience of loneliness 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 HF-HRV during an acute experience of state loneliness. Immediately following this period, participants completed the measure of state affect previously described to ensure that the loneliness manipulation had been successful. In another manipulation check at the end of the lab session, no participants reported suspicion of the false loneliness feedback they had received. Finally, all participants were thoroughly and sensitively debriefed, and offered the opportunity to watch videos containing positive stimuli.
Demographics and health behaviours. Prior to the lab session, information on a number of demographic variables and health behaviours was collected from participants to allow these variables to be included as covariates in study analyses. In an online questionnaire, 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, whether they had any medical (including mental health) conditions (and if so, to name/describe the condition), and whether they were currently taking medication (and if so, to report the name of the medication).

In addition, participants reported their average hours of sleep per night, as well as their average alcohol consumption, caffeine consumption, smoking habits, and marijuana use. Exercise was assessed by asking participants the number of hours per week that they exercised enough to build up a sweat.
Results

All statistical analyses were conducted in SPSS (Version 24.0). Means and standard deviations for major study variables are presented in Table 2. To assess the effect of trait loneliness on vagal flexibility to cognitive challenge, HF-HRV reactivity to, and HF-HRV recovery from, the cognitive challenge task were examined. HF-HRV reactivity was calculated by subtracting the mean HF-HRV obtained during baseline (Baseline 1) from the mean HF-HRV obtained during cognitive challenge. Likewise, HF-HRV recovery was calculated by subtracting the mean HF-HRV obtained during the challenge task from the mean HF-HRV observed during the resting period immediately following the cognitive challenge (Baseline 2). All HF-HRV values are reported in natural log units (ln ms²).

As expected, mean HF-HRV reactivity to cognitive challenge was negative, indicating an overall pattern of vagal withdrawal across participants, $M = -0.42$ ln ms², $SD = 0.75$, 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 vagal withdrawal from baseline during the cognitive challenge, $t(147) = -6.86$, $p < .001$, $d = 0.56$, 95% CI [–0.55, –0.30]. Also as expected, mean HF-HRV recovery was positive, indicating an overall pattern of vagal augmentation across participants following cognitive challenge, $M = 0.46$ ln ms², $SD = 0.64$, but again, a considerable range of responses was observed: –1.04 to 2.28. A $t$ test confirmed that on average participants experienced significant HF-HRV recovery following cognitive challenge, $t(147) = 8.75$, $p < .001$, $d = 0.72$, 95% CI [0.36, 0.57]. Furthermore, HF-HRV during the recovery period (Baseline 2) was not significantly different from baseline HF-HRV (Baseline 1), $p = .361$, indicating that on average participants recovered to their earlier baseline HF-HRV levels.
Trait Loneliness

Associations between trait loneliness (UCLA total score) and HF-HRV measures were assessed (see Figure 2 for Pearson correlations). Trait loneliness was significantly correlated with HF-HRV reactivity to cognitive challenge, \( r(148) = .23, p = .005 \), such that higher loneliness scores were associated with smaller decreases in HF-HRV (i.e., blunted vagal reactivity) to cognitive challenge, whereas lower loneliness scores were associated with greater decreases in HF-HRV (i.e., vagal flexibility) to cognitive challenge. Trait loneliness was also correlated with HF-HRV recovery to cognitive challenge, \( r(148) = -.23, p = .005 \). Specifically, higher loneliness scores predicted less increase in HF-HRV following cognitive challenge, whereas lower loneliness scores predicted greater increase in HF-HRV following cognitive challenge. As expected, trait loneliness was not significantly associated with HF-HRV at baseline, during cognitive challenge, or following cognitive challenge (i.e., tonic HF-HRV), all \( ps > .263 \).

Trait loneliness predicted HF-HRV reactivity to cognitive challenge (H1a) in a hierarchical regression analysis controlling for factors that may influence vagal responses (Laborde et al., 2017; Quintana & Heathers, 2014), specifically: respiration, age, BMI, and tonic HF-HRV (i.e., baseline HF-HRV). In Step 1 of the model, covariates were entered, and the overall model significantly predicted HF-HRV reactivity, \( F(2, 145) = 37.46, p < .001, R^2 = .34, \) adjusted \( R^2 = .33 \). Importantly, the inclusion of trait loneliness in Step 2 significantly improved the accuracy of the model, \( \Delta R^2 = .03, \Delta F(1, 144) = 7.62, p = .007 \). Trait loneliness was a

\[^{7}\text{Although age and BMI were initially included as covariates, neither variable was related to HF-HRV reactivity, HF-HRV recovery, or loneliness, and were consequently removed from the regression models.}\]
significant predictor of HF-HRV reactivity to cognitive challenge, \( \beta = .18, t(144) = 2.76, p = .007 \), even after controlling for covariates.

The associations of HF-HRV reactivity to cognitive challenge and each of the self-reported psychosocial measures were also assessed (see Table 3). HF-HRV reactivity was not significantly correlated with anxiety (BAI total score), social engagement (LSNS-6 total score), social anxiety (SIAS total score), attachment avoidance (ECR-S subscale score), or attachment anxiety (ECR-S subscale score), all \( rs < .10, all \ p s > .209 \), and was marginally correlated with depression (CESD total score; \( r = .15, p = .073 \)) and self-esteem (RSES total score; \( r = -.14, p = .097 \)). HF-HRV reactivity was significantly correlated with stress (PSS total score; \( r = .19, p = .021 \)) and perceived social support (ISEL-12 total score, \( r = -.17, p = .041 \)). However, when stress, social support, depression, and self-esteem were individually added to the regression model, the association between trait loneliness and HF-HRV reactivity was not significantly altered, and the associations between HF-HRV reactivity and stress, social support, depression, and self-esteem became nonsignificant.

In addition, a number of demographic and health-related variables were examined as potential confounds (see Table 4 for correlations with HF-HRV). Of these, only average alcohol consumption and average hours of sleep were found to correlate with HF-HRV reactivity to cognitive challenge. However, the association between trait loneliness and HF-HRV reactivity remained essentially unchanged when including these variables as covariates, so the more parsimonious models without these variables are reported here\(^8\).

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\(^8\) Average alcohol consumption was no longer significantly associated with HF-HRV reactivity when included as a covariate in the regression model. Fewer average hours of sleep remained a significant predictor of blunted HF-HRV reactivity but did not improve the model fit and was not retained in the model.
Trait loneliness also predicted HF-HRV recovery after cognitive challenge (H1b) in a hierarchical regression analysis controlling for covariates. As before, in Step 1 of the model, respiration and tonic HF-HRV (i.e., HF-HRV during cognitive challenge) were entered as covariates, and the overall model significantly predicted HF-HRV recovery, $F(2, 145) = 28.43, p < .001, R^2 = .28$, adjusted $R^2 = .27$. Inclusion of trait loneliness in Step 2 significantly improved the accuracy of the model, $\Delta R^2 = .03, \Delta F(1, 144) = 7.24, p = .008$, such that trait loneliness was a significant predictor of HF-HRV recovery from cognitive challenge, $\beta = -.19, t(144) = -2.69, p = .008$. However, when HF-HRV reactivity to cognitive challenge was included in the regression model, the association between trait loneliness and HF-HRV recovery was no longer significant, suggesting that average change in HF-HRV during recovery from cognitive challenge was mainly a function of the average change in HF-HRV from Baseline 1 to cognitive challenge (HF-HRV recovery was highly correlated with HF-HRV reactivity, $r = -.72, p < .001$).

**State Loneliness**

Changes in state loneliness were evaluated using participants’ self-reports following baseline (Baseline 1), as well as immediately prior to the loneliness induction (Baseline 2), and immediately after the 5-min loneliness experience (Loneliness). Using a repeated-measures ANOVA with Greenhouse-Geisser correction, average state loneliness was found to differ significantly between time points, $F(1.30, 191.13) = 98.66, p < .001, \eta^2 = .40$. Bonferroni corrected follow-up comparisons indicated that each pairwise difference was significant (all $p$s < .001). Importantly, self-reported loneliness following the loneliness experience (Loneliness; $M = 3.64, SD = 2.70$) was significantly higher than self-reports immediately preceding the loneliness induction (Baseline 2; $M = 1.50, SD = 1.98$), and at baseline (Baseline 1; $M = 1.86, SD = 2.23$),
indicating that the loneliness manipulation was successful\textsuperscript{9}. The significant increase in self-reported loneliness ($M = 2.14, SD = 2.22$) observed after the loneliness experience (compared to immediately before the loneliness induction), was accompanied by significant increases in self-reported sadness ($M = 2.59, SD = 2.43$), anger ($M = 0.76, SD = 1.67$), worry ($M = 0.73, SD = 1.95$), and tension ($M = 1.14, SD = 1.78$), along with significant decreases in happiness ($M = -1.30, SD = 1.72$) and feelings of relaxation ($M = -1.80, SD = 2.22$). This broader pattern of emotional changes is associated with states of loneliness (Cacioppo et al., 2006; Heinrich & Gullone, 2006) providing further evidence that participants were truly experiencing loneliness, rather than merely exhibiting demand characteristics.

To assess the effect of state loneliness on HF-HRV ($H2$), a repeated-measures ANOVA with a Greenhouse-Geisser correction was conducted. Lab task was entered as a within-subjects factor (three levels: Baseline 1, Baseline 2, and Loneliness). The analysis revealed a significant main effect of lab task on HF-HRV, $F(1.61, 236.54) = 8.11, p = .001, \eta^2 = .05$. Pairwise comparisons using Bonferroni correction indicated that mean HF-HRV during loneliness (Loneliness; $M = 7.20 \ln \text{ms}^2, SD = 0.89$) was significantly higher than mean HF-HRV at baseline (Baseline 1; $M = 7.01 \ln \text{ms}^2, SD = 1.07$) ($p = .006$), and was also significantly higher than mean HF-HRV immediately preceding the loneliness induction (Baseline 2; $M = 7.05 \ln \text{ms}^2, SD = 0.92$) ($p = .002$). The difference between HF-HRV at Baseline 1 and HF-HRV at Baseline 2 was not significant ($p = .361$). Figure 3 depicts change in HF-HRV across lab tasks.

\textsuperscript{9} Interestingly, self-reported loneliness immediately prior to the loneliness induction showed a significant decrease from baseline. It is speculated that the mere act of participating in the study and interacting with the experimenter may have reduced state loneliness in participants.
Exploratory Analyses

Exploratory analyses of potential differences in HF-HRV between high trait-lonely women and low trait-lonely women both during state loneliness (H3a) and following state loneliness (H3b) were also conducted. Trait loneliness was not found to be associated with HF-HRV reactivity to the loneliness manipulation or with HF-HRV during the loneliness manipulation. However, in a two-step hierarchical regression controlling for covariates, trait loneliness was significantly associated with HF-HRV recovery following state loneliness. In Step 1 of the model, respiration and tonic HF-HRV (i.e., HF-HRV during acute loneliness) were entered as covariates, and the overall model significantly predicted HF-HRV recovery, $F(2, 145) = 11.07, p < .001, R^2 = .13$, adjusted $R^2 = .12$. Inclusion of trait loneliness in Step 2 significantly improved the accuracy of the model, $\Delta R^2 = .04, \Delta F(1, 144) = 6.47, p = .012$, indicating that trait loneliness was a significant predictor of HF-HRV recovery from state loneliness after controlling for covariates, $\beta = -.19, t(144) = -2.54, p = .012$. Specifically, among low trait-lonely women, HF-HRV continued to increase further away from baseline following state loneliness, whereas among high trait-lonely women, HF-HRV decreased following state loneliness (see Figure 4).
Discussion

The aim of this investigation was to examine differential associations between trait loneliness and state loneliness with vagally-mediated PNS activity, indexed by HF-HRV. Among women between ages 18 to 27 years, higher trait loneliness predicted blunted HF-HRV reactivity to cognitive challenge (H1a). Higher trait loneliness also predicted blunted HF-HRV recovery from cognitive challenge (H1b), however, this association was no longer significant after accounting for HF-HRV reactivity, suggesting that HF-HRV increase during recovery was mainly a function of how much HF-HRV had initially decreased in reaction to the cognitive challenge task. In line with the evolutionary model of loneliness (Cacioppo et al., 2014) and polyvagal theory (Porges, 2007), an acute experience of state loneliness was found to be associated with increased HF-HRV (H2). Trait loneliness did not predict tonic HF-HRV during state loneliness (H3a), or HF-HRV reactivity to state loneliness. Trait loneliness predicted HF-HRV recovery from state loneliness (H3b)—specifically, following an experience of state loneliness, HF-HRV decreased in women reporting higher trait loneliness, whereas HF-HRV increased in women reporting lower trait loneliness. Taken together, these findings suggest that trait loneliness and state loneliness have different implications for parasympathetic nervous system functioning.

In a successful replication of the findings by Muhtadie et al. (2015), higher trait loneliness was correlated with blunted HF-HRV reactivity to cognitive challenge. Furthermore, the present study extends the prior research by showing that this relationship held in a larger sample after accounting for psychosocial variables associated with loneliness (i.e., stress, anxiety, depression, social support, frequency of social interactions, self-esteem, social anxiety, attachment avoidance, attachment anxiety) and health behaviours (e.g., exercise, sleep, alcohol...
use, caffeine consumption); controlling for these factors represents a strength of the present research.

Of note, higher stress predicted blunted HF-HRV reactivity to cognitive challenge in the present study (although this association became nonsignificant when accounting for loneliness), whereas stress was not associated with HF-HRV reactivity in the study by Muhtadie and colleagues (2015). Self-reported stress in the current sample was quite high\(^{10}\), possibly because participants were female university undergraduates, as younger age and female sex are both independently associated with higher stress (Cohen & Janicki-Deverts, 2012). Therefore, higher perceived stress in the current sample may account for why stress was related to HF-HRV reactivity in the present study, but not related to HF-HRV reactivity in the older\(^ {11}\) mixed-sex community sample recruited by Muhtadie et al. (2015).

Furthermore, the fact that higher trait loneliness predicted blunted HF-HRV reactivity among the present sample of young women suggests that, at least for lower-order cognitive processes, the deleterious effects of loneliness may be incurred much earlier in life than was previously suspected (Cacioppo et al., 2014). It is unclear whether such cognitive impairments can develop after a single prolonged experience of situational loneliness (e.g., a university semester), or whether they may be driven by more stable personality factors. For example, neuroticism is strongly correlated with loneliness (e.g., \(r = .61\) in females; Boomsma, Willemsen, Dolan, Hawkley, & Cacioppo, 2005), and has been associated with impaired cognitive functioning, including lower general cognitive performance in childhood (Gale, Deary, Kuh,

\(^{10}\) Mean self-reported stress in the current sample was 20.50, \(SD = 5.95\). For comparison, mean self-reported stress in a mixed-sex sample of 18–25 years olds (\(N = 223\)) in 2009 was 16.78, \(SD = 6.86\) (Cohen & Janicki-Deverts, 2012). Mean trait loneliness in the current sample was 43.39, \(SD = 10.38\). As a comparison, mean trait loneliness in a mixed-sex sample of college students (\(N = 487\)) reported in 1996 was 40.08, \(SD = 9.50\) (Russell, 1996).

\(^{11}\) Mean age = 44.5 years, \(SD = 20.6\), age range: 20–74 (Muhtadie et al., 2015).
Huppert, & Richards, 2010), and greater response time inconsistency in younger and older adults (Munoz, Stawski, Sliwinski, Smyth, & MacDonald, 2018). However, it should also be noted that, in studies of young adults, associations between loneliness and cardiovascular activity (e.g., higher TPR [total peripheral resistance] and lower CO [cardiac output]), as well as associations between loneliness and negative affect, anxiety, anger, pessimism, self-esteem, and social support, remain robust even after accounting for neuroticism (Cacioppo et al., 2006; Hawkley et al., 2003). Further research in this area is clearly warranted.

From an evolutionary perspective, unpleasant feelings of loneliness are thought to have promoted survival by activating motivation for social affiliation and reconnection, in response to perceptions of social isolation (Cacioppo et al., 2015; Qualter et al., 2015). By demonstrating that HF-HRV is increased during an experience of loneliness, the present study provides the first evidence that acute loneliness may prompt a physiological state that facilitates social affiliation and reconnection. Furthermore, the present findings are consistent with research indicating that loneliness and parasympathetic function operate on common neural substrates (Wilson et al., 2018). Importantly, this increase in vagally-mediated parasympathetic influence does not preclude a concurrent increase in sympathetic activity or the heightened vigilance for social threat proposed to be triggered by loneliness (Berntson et al., 2008; Cacioppo et al., 2015; Norman et al., 2011).

Loneliness may not be unique in eliciting a physiological state conducive to social affiliative behaviours. Higher average daily sadness has been associated with higher HF-HRV in women (Verkuil et al., 2015), and individuals with higher HF-HRV have been found to be more likely to seek social support in response to an experience of sadness (Geisler, Kubiak, Siewert, & Weber, 2013). However, both increases and decreases in HF-HRV have been documented during
acute sadness (Kreibig, 2010; Stellar, Cohen, Oveis, & Keltner, 2015). One proposed explanation for this inconsistency is that different types of sadness (e.g., crying versus non-crying sadness, other-oriented versus self-oriented sadness) may provoke different patterns of autonomic responses (Kreibig, 2010; Stellar et al., 2015). A further possibility, suggested by the current findings, is that sadness linked to personal social loss (as opposed to watching, for example, a sadness-inducing film clip) is related to parasympathetic activation, and that the component of sadness inherent to loneliness experiences, may drive the observed increase in HF-HRV during state loneliness.

Whether loneliness is experienced as transient, without long-lasting adverse consequences, or develops into a prolonged and aversive condition with severe health implications, is argued to depend on whether the reaffiliation motive (RAM) fails to lead to social reconnection after it has been triggered (Qualter et al., 2015). Exploratory analyses in the present study revealed that high trait loneliness predicted a decrease in HF-HRV following an experience of state loneliness. This association was not explained by level of tonic HF-HRV during the loneliness experience12. Intriguingly, this observation may suggest that chronically lonely individuals display a physiological state that is less conducive to social affiliation immediately following acute feelings of loneliness that may hinder their ability to reconnect. Thus, how people respond immediately after state loneliness may represent a critical period in determining whether loneliness is reduced or perpetuated. Research on how lonely individuals respond to opportunities for social engagement, and to acute feelings of loneliness, has been inconsistent (Vanhalst et al., 2015). The present findings suggest that it may be profitable to

12 Furthermore, there was no association between baseline HF-HRV and the change in HF-HRV following state loneliness, $r = .002, p = .977$. 
include measures of parasympathetic functioning in studies examining the social behaviours of lonely individuals.

A main strength of the present study is that it was conducted under controlled laboratory conditions. Numerous factors that can influence HF-HRV were controlled for, including: time of day, room temperature, age, sex, BMI, cardioactive medication use, medical condition, average hours of sleep, weekly exercise, and habitual intake of alcohol, caffeine, nicotine, and marijuana. Furthermore, factors that are not often controlled for, but that are known to cause autonomic responses—namely, participants’ sleep routine and consumption of food and drink prior to the lab session—were also accounted for (Quintana & Heathers, 2014). Another strength of the study was the use of a loneliness induction paradigm with high face validity. In addition to measuring loneliness at the start of the lab session, and immediately before and after the loneliness manipulation, the assessment of a broader pattern of emotional changes associated with loneliness provided strong evidence that the loneliness manipulation was successful. The present study also measured HF-HRV recovery, enabling a more complete picture of parasympathetic responding before, during, and after both the cognitive challenge and the loneliness induction. In the past, studies have typically measured HF-HRV before and during emotion induction, but not after emotion induction (Balzarotti et al., 2017). The assessment of HF-HRV recovery in this study exposed individual differences in HF-HRV following state loneliness, and also revealed the important observation that a 5-min recovery period can be sufficient for HF-HRV to return to baseline levels following a lower-order cognitive challenge, but is unlikely to be sufficient following an emotional challenge.

Several limitations to this study should be acknowledged. First, the correlational approach used to examine the associations between HF-HRV and trait loneliness prohibits any
definite conclusions on the direction of the observed associations. Second, because there was no control condition for the loneliness manipulation, alternate explanations cannot be ruled out as to why HF-HRV increased during state loneliness. Also, for practical reasons, the baseline HF-HRV measures did not immediately precede the 5-min state loneliness HF-HRV measure, as there was a 15-min loneliness induction paradigm before the state loneliness measure. Although this timeline is unlikely to have influenced the present findings, it would have been ideal for the baseline HF-HRV measure to have immediately preceded the state loneliness HF-HRV measure. Therefore, one possibility for future research in this area would be the use of ambulatory measures of HF-HRV combined with experience sampling of state loneliness using ecological momentary assessments (EMA). Another limitation is that sympathetic nervous system (SNS) activity was not measured. As HF-HRV only indexes parasympathetic activity, no conclusions can be drawn regarding SNS functioning during loneliness. Finally, although restricting study participation to young adult women reduced variability in the sample, it limits the generalizability of the results. As such, future research is needed to determine whether the observed effects in the present study apply to older adults and to men, especially as there is evidence to suggest that there may be sex differences in brain function during the experience of sadness (Verkuil et al., 2015). Specifically, self-reported sadness has been associated with increased right amygdala activity in men, but not in women (Schneider, Habel, Kessler, Salloum, & Posse, 2000). As no similar research has been conducted on state loneliness, it is not possible

13 PNS influence operates on a timescale of < 1 s (Nunan et al., 2010; Shaffer et al., 2014), thus any PNS fluctuations to facilitate—for example—the action of typing on the computer keyboard during the loneliness induction paradigm would have been restored once typing ceased, which was ~3 min before the measure of HF-HRV during state loneliness was taken.
to say whether sex differences corresponding to those observed during sadness will be present for loneliness.

Furthermore, the present finding that state loneliness is associated with increased HF-HRV raises important questions about the process by which loneliness may exert a cumulative impact on parasympathetic functioning over the life span. For example, if repeated and frequent experiences of loneliness have a detrimental effect on parasympathetic functioning, state loneliness may not elicit the same pattern of autonomic activity in old age as it does in youth. This hypothesis could be tested by administering the loneliness paradigm employed in the current study to a sample of older adults, and observing the elicited parasympathetic and sympathetic responses (if any). Other applications of the current loneliness paradigm include testing whether inducing state loneliness can temporarily improve accuracy in detecting social cues (e.g., facial expressions), as well as investigating individual differences in social affiliative behaviours following loneliness induction.
Conclusion

Loneliness is now a well-established risk factor for numerous adverse health outcomes, including early death. However, it can also be an important motivating force that drives individuals to initiate and repair fragile social bonds. The present study provides the first evidence that high-frequency heart rate variability (HF-HRV) is increased during an acute state of loneliness, consistent with theorizing that loneliness promotes a physiological state that facilitates social affiliation and reconnection. The current findings also indicate that trait loneliness and state loneliness may have starkly different implications for parasympathetic nervous system functioning. In a replication and extension of prior work, trait loneliness was found to predict blunted HF-HRV reactivity to cognitive challenge, an association that remained robust after accounting for known covariates. Manipulation checks during the study revealed that a new, strengthened loneliness induction paradigm developed from existing methods was successful. Following an experimental induction of state loneliness, higher levels of trait loneliness predicted decreased HF-HRV. The application of a successful loneliness induction paradigm in psychological research, used responsibly and considerately, can be a valuable tool to begin disentangling the concepts underlying the terms of state and trait loneliness.
**Table 1**

*Sample Descriptive Statistics*

<table>
<thead>
<tr>
<th>Variable</th>
<th>% or M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>19.97 (1.70)</td>
</tr>
<tr>
<td>Body mass index(^a)</td>
<td>22.81 (3.97)</td>
</tr>
<tr>
<td>% in a committed relationship</td>
<td>25.6%</td>
</tr>
<tr>
<td>% living in Vancouver for one year or less</td>
<td>21.6%</td>
</tr>
<tr>
<td>% first year university students</td>
<td>29.7%</td>
</tr>
<tr>
<td>% report medical condition</td>
<td>11.5%</td>
</tr>
<tr>
<td>% use medication</td>
<td>8.8%</td>
</tr>
<tr>
<td><strong>Health behaviours</strong></td>
<td></td>
</tr>
<tr>
<td>Average hours of sleep per night</td>
<td>6.87 (1.03)</td>
</tr>
<tr>
<td>% exercise less than one hour per week</td>
<td>31.8%</td>
</tr>
<tr>
<td>% drink caffeine once per week or less</td>
<td>33.8%</td>
</tr>
<tr>
<td>% drink alcohol once per week or less</td>
<td>88.5%</td>
</tr>
<tr>
<td>% smoke less than one cigarette per week or never</td>
<td>98.0%</td>
</tr>
<tr>
<td>% use marijuana less than once per month or never</td>
<td>87.8%</td>
</tr>
</tbody>
</table>

*Note. N = 148. All participants were female. The present sample excluded individuals who reported a heart condition, current use of cardioactive medication, or daily use of marijuana or cigarettes. \(^a\)Body mass index calculated as kg/m\(^2\).*
Table 2

*Means and Standard Deviations for Measures of High-Frequency Heart Rate Variability, Psychosocial Variables, and Experiment Characteristics*

<table>
<thead>
<tr>
<th></th>
<th>M (SD)</th>
<th>Possible range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High-frequency heart rate variability† (HF-HRV)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline 1</td>
<td>7.01 (1.07)</td>
<td></td>
</tr>
<tr>
<td>Cognitive Challenge</td>
<td>6.59 (0.93)</td>
<td></td>
</tr>
<tr>
<td>Baseline 2</td>
<td>7.05 (0.92)</td>
<td></td>
</tr>
<tr>
<td>State Loneliness</td>
<td>7.20 (0.89)</td>
<td></td>
</tr>
<tr>
<td>Recovery</td>
<td>7.21 (0.87)</td>
<td></td>
</tr>
<tr>
<td><strong>Psychosocial measures</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loneliness (UCLA)</td>
<td>43.39 (10.38)</td>
<td>[20 – 80]</td>
</tr>
<tr>
<td>Perceived stress (PSS)</td>
<td>20.50 (5.95)</td>
<td>[0 – 40]</td>
</tr>
<tr>
<td>Anxiety (BAI)</td>
<td>22.24 (16.80)</td>
<td>[0 – 99]</td>
</tr>
<tr>
<td>Depression (CESD)</td>
<td>19.26 (14.51)</td>
<td>[0 – 80]</td>
</tr>
<tr>
<td>Social support (ISEL-12)</td>
<td>24.70 (6.89)</td>
<td>[0 – 36]</td>
</tr>
<tr>
<td>Social engagement (LSNS-6)</td>
<td>16.72 (5.13)</td>
<td>[0 – 36]</td>
</tr>
<tr>
<td>Social anxiety (SIAS)</td>
<td>31.86 (15.83)</td>
<td>[0 – 80]</td>
</tr>
<tr>
<td>Self-esteem (RSES)</td>
<td>18.95 (5.69)</td>
<td>[0 – 30]</td>
</tr>
<tr>
<td>Attachment avoidance (ECR-S subscale)</td>
<td>19.00 (7.44)</td>
<td>[6 – 42]</td>
</tr>
<tr>
<td>Attachment anxiety (ECR-S subscale)</td>
<td>23.43 (6.38)</td>
<td>[6 – 42]</td>
</tr>
<tr>
<td><strong>Experiment characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Room temperature (Celsius)</td>
<td>21.94 (1.31)</td>
<td></td>
</tr>
<tr>
<td>Time of day (24 hr clock)</td>
<td>13:40 (1:41)</td>
<td></td>
</tr>
</tbody>
</table>

*Note.†HF-HRV is reported in natural log units (ln ms², FFT metric). UCLA = UCLA Loneliness Scale; PSS = Perceived Stress Scale; BAI = Burns Anxiety Inventory; CESD = Center for Epidemiological Studies Depression Scale; ISEL = Interpersonal Support Evaluation List; LSNS = Lubben Social Network Scale; SIAS = Social Interaction Anxiety Scale; RSES = Rosenberg Self-Esteem Scale; ECR-S = Experiences in Close Relationship Scale.*
Table 3

Correlations between Measures of High-Frequency Heart Rate Variability⁴ (HF-HRV) and Psychosocial Variables

<table>
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<tr>
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<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Baseline HF-HRV (before cognitive challenge)</td>
<td>-.52**</td>
<td>.19*</td>
<td>-.09</td>
<td>-.04</td>
<td>-.05</td>
<td>-.14</td>
<td>.14</td>
<td>.01</td>
<td>.02</td>
<td>.04</td>
<td>-.01</td>
<td>.02</td>
</tr>
<tr>
<td>2</td>
<td>HF-HRV reactivity (to cognitive challenge)</td>
<td>-.72**</td>
<td>.23**</td>
<td>.19*</td>
<td>.07</td>
<td>.15</td>
<td>-.17*</td>
<td>-.10</td>
<td>.03</td>
<td>-.14</td>
<td>.08</td>
<td>.02</td>
<td></td>
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<tr>
<td>3</td>
<td>HF-HRV recovery (from cognitive challenge)</td>
<td>-.23**</td>
<td>-.27**</td>
<td>-.10</td>
<td>-.17*</td>
<td>.15</td>
<td>.17*</td>
<td>-.02</td>
<td>.17*</td>
<td>-.06</td>
<td>.03</td>
<td></td>
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</tr>
<tr>
<td>4</td>
<td>Trait loneliness (UCLA)</td>
<td></td>
<td>.43**</td>
<td>.45**</td>
<td>.47**</td>
<td>-.76**</td>
<td>-.57**</td>
<td>.59**</td>
<td>.54**</td>
<td>.46**</td>
<td>.25**</td>
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<tr>
<td>5</td>
<td>Stress (PSS)</td>
<td></td>
<td>.63**</td>
<td>.74**</td>
<td>-.27**</td>
<td>-.10</td>
<td>.36**</td>
<td>-.52**</td>
<td>.17*</td>
<td>.06</td>
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<tr>
<td>6</td>
<td>Anxiety (BAI)</td>
<td></td>
<td>.77**</td>
<td>-.36**</td>
<td>-.12</td>
<td>.43**</td>
<td>-.44**</td>
<td>.34**</td>
<td>.16</td>
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<td>7</td>
<td>Depression (CESD)</td>
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<td></td>
<td></td>
<td>-34**</td>
<td>-.12</td>
<td>.42**</td>
<td>-.57**</td>
<td>.30**</td>
<td>.12</td>
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<td>8</td>
<td>Social support (ISEL-12)</td>
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<td></td>
<td>.54**</td>
<td>-.58**</td>
<td>.37**</td>
<td>-.46**</td>
<td>-.27**</td>
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<td>9</td>
<td>Social engagement (LSNS-6)</td>
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<td>-.38**</td>
<td>.23**</td>
<td>-.25**</td>
<td>-.06</td>
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<td>10</td>
<td>Social anxiety (SIAS)</td>
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<td>-.28**</td>
<td>-.23**</td>
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<tr>
<td>12</td>
<td>Attachment avoidance (ECR-S)</td>
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<td>.33**</td>
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<td>13</td>
<td>Attachment anxiety (ECR-S)</td>
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</table>

Note. *p < .05; **p < .01; †HF-HRV is reported in natural log units (ln ms², FFT metric). UCLA = UCLA Loneliness Scale; PSS = Perceived Stress Scale; BAI = Burns Anxiety Inventory; CESD = Center for Epidemiological Studies Depression Scale; ISEL = Interpersonal Support Evaluation List; LSNS = Lubben Social Network Scale; SIAS = Social Interaction Anxiety Scale; RSES = Rosenberg Self-Esteem Scale; ECR-S = Experiences in Close Relationship Scale.
Table 4

Correlations between Measures of High-Frequency Heart Rate Variability† (HF-HRV), Demographics, and Health Behaviours

<table>
<thead>
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<tbody>
<tr>
<td><strong>Baseline HF-HRV</strong> (before cognitive challenge)</td>
<td></td>
<td>-52**</td>
<td>.19*</td>
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<td>.30**</td>
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<td>.20*</td>
<td>-05</td>
<td>.03</td>
<td>.08</td>
<td>.12</td>
<td>.05</td>
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<tr>
<td><strong>HF-HRV reactivity</strong> (to cognitive challenge)</td>
<td></td>
<td>-72**</td>
<td>-05</td>
<td>-03</td>
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<td>-09</td>
<td>-18*</td>
<td>.13</td>
<td>-.21*</td>
<td>.01</td>
<td>-.12</td>
<td></td>
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<tr>
<td><strong>HF-HRV recovery</strong> (from cognitive challenge)</td>
<td></td>
<td>.07</td>
<td>-07</td>
<td>-08</td>
<td>.12</td>
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<td>.13</td>
<td>-.06</td>
<td>.15</td>
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<td><strong>Age</strong></td>
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<td>.21**</td>
<td>.08</td>
<td>.16</td>
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<td>.13</td>
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<td>-06</td>
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<tr>
<td><strong>Body mass index</strong>a</td>
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<td>.08</td>
<td>.04</td>
<td>.06</td>
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<td>.07</td>
<td>.13</td>
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<td><strong>Weekly exercise</strong></td>
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<td>.11</td>
<td>.19*</td>
<td>.22**</td>
<td>-.11</td>
<td>-.03</td>
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<td><strong>Average hours of sleep</strong></td>
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<td>.22**</td>
<td>-.12</td>
<td>-.09</td>
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<tr>
<td><strong>Average caffeine intake</strong></td>
<td></td>
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<td>.23**</td>
<td>.25**</td>
<td>.11</td>
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<td><strong>Average alcohol intake</strong></td>
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<td>.14</td>
<td>.05</td>
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<td><strong>Smoking</strong>c</td>
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<td><strong>Marijuana use</strong>c</td>
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Note. *p < .05, **p < .01; †HF-HRV is reported in natural log units (ln ms², FFT metric). aBody mass index calculated as kg/m². b1 = single, 2 = dating, 3 = in committed relationship, not cohabiting with partner, 4 = in committed relationship, cohabiting with partner. cThe present sample excluded individuals who reported a heart condition, current use of cardioactive medication, daily smoking, or daily use of marijuana.
Figure 1. Timeline of study procedure.
Figure 2. Pearson correlations between (a) trait loneliness and high-frequency heart rate variability (HF-HRV) reactivity to cognitive challenge, and (b) trait loneliness and HF-HRV recovery from cognitive challenge. HF-HRV is displayed in natural log units (ln ms\(^2\), FFT metric).
Figure 3. Within-subjects differences in mean high-frequency heart rate variability (HF-HRV) across study conditions. Error bars represent 95% confidence intervals. HF-HRV is displayed in natural log units (ln ms², FFT metric).
Figure 4. Change in high-frequency heart rate variability (HF-HRV) following state loneliness in low trait-lonely women (below the sample median) and in high trait-lonely women (above the sample median). HF-HRV is displayed in natural log units (ln ms², FFT metric).
References


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Mateo, J., & Laguna, P. (2000). Improved heart rate variability signal analysis from the beat occurrence times according to the IPFM model. *IEEE Transactions on Biomedical Engineering, 47*(8), 985–996. doi: 10.1109/10.855925


the differential reactivity hypothesis. *Journal of Clinical Child and Adolescent Psychology, 47*(6), 888–899. doi: 10.1080/15374416.2016.1146993


Appendix: State Loneliness Manipulation Materials

Screen 1

Linguistic Analysis Word Count
LAWC2015

Participant Demographics

Please enter your age. Please use a whole numeral instead of words.

Are you:

☐ Male
☐ Female
For this next task, we would like for you to write about your very deepest thoughts and feelings about a time in your past when you felt lonely. This may have been a time when you were alone or when you were with others but you felt alone—and you did not want to be alone. You may have felt lonely after you moved to a new city, or when you wished you were part of a study group. Perhaps you felt lonely when you wished that you had someone to make plans with on a Friday night. There are many other situations when you may have felt lonely.

In your writing, we’d like you to really let go and explore your very deepest lonely feelings and thoughts.

You might write about one lonely event, or several—not everyone experiences loneliness in the same way, so write about the feeling of loneliness as it is true for you. Try to really take yourself back to how you felt at that time.

All of your writing will be completely anonymous. You may find it easiest to type as if you are speaking to another person. Don’t worry about spelling, sentence structure, or grammar. The only rule is that once you begin typing, continue to do so until your time is up. If you get stuck on what to write, it can help to retype the last sentence that you wrote.

You do not need to keep track of the time. The experimenter will knock on the door and re-enter the room after 10 minutes has passed. Do not hit the 'Submit' button until the experimenter has returned and instructed you to do so.

Please now take a moment to recall a time when you felt lonely.

When you are ready to begin writing, simply start typing in the space below.
LAWC Results
Scale: LON
Details of Writer: 18-30 year old Female

SUMMARY
Score: 52.98

High

<table>
<thead>
<tr>
<th>LAWC Dimension</th>
<th>Score</th>
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<tbody>
<tr>
<td>Self-reference</td>
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<tr>
<td>Displacement</td>
<td>8.62</td>
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<tr>
<td>Positive affect</td>
<td>1.12</td>
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<tr>
<td>Negative affect</td>
<td>9.85</td>
</tr>
<tr>
<td>Social connection</td>
<td>2.26</td>
</tr>
<tr>
<td>Social isolation</td>
<td>16.39</td>
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</tbody>
</table>

LAWC2015 overall loneliness score: 52.98
Average score (based on aggregate): 40.02

The LAWC2015 Loneliness scale has been administered to a large number of students over the last 3 years. Based on the responses of over 18,000 students, we have developed a way of scoring your answers that allows us to provide you with valid and specified feedback regarding your level of loneliness.

Please refer to the researcher for further information regarding your loneliness score in comparison to your local population.

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