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Isotemporal Associations of Device-Measured Sedentary Time and Physical Activity with Cardiac-Autonomic Regulation in Previously Pregnant Women

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Abstract

Background: High sedentary time (ST) and low physical activity may increase cardiovascular risk, potentially though cardiac-autonomic dysregulation. This study investigated associations of statistically exchanging device-measured ST and physical activity with measures of cardiac-autonomic regulation in previously pregnant women.

Methods: This cross-sectional, secondary analysis included 286 women (age=32.6±5.7 yrs; 68% white) measured 7-15 years after delivery. ST and light (LPA), moderate (MPA), vigorous (VPA), and moderate-to-vigorous (MVPA) intensity physical activity were measured by ActiGraph GT3X. ST was further partitioned into long (≥30 minutes) and short (<30 minutes) bouts. MVPA

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Conflicts of interest declaration

The authors declare that they have no conflict of interest.

Informed consent

Informed consent was obtained from all individual participants included in the study.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

was also partitioned into long (≥10-minute) and short (<10-minute) bouts. Cardiac-autonomic regulation was assessed by heart rate variability (HRV) (resting heart rate, natural log transformed standard deviation of normal R-R intervals [lnSDNN], natural log-transformed root mean square of successive differences [lnRMSSD]) from a 5-minute seated ECG. Progressive isotemporal substitution models adjusted for confounders. Sensitivity analyses removed women with related underlying medical conditions and who did not meet respiration rate criteria.

Results: Initial analyses found no significant associations with HRV when exchanging 30 minutes of ST and physical activity ($p>0.05$). Yet, replacing long- and short-bout ST with 30 minutes of long-bout MVPA yielded significantly higher (healthier) lnRMSSD ($B=0.063\pm0.030$ and $B=0.056\pm0.027$, respectively; both $p<0.05$). Sensitivity analyses strengthened these associations and yielded further associations of higher lnSDNN and lnRMSSD when replacing 30 minutes of short-bout MVPA with equivalent amounts of long-bout MVPA ($B=0.074\pm0.037$ and $B=0.091\pm0.046$, respectively).

Conclusion: Replacing ST with long-bout MVPA is a potential strategy to improve cardiac-autonomic function in previously pregnant women.

Keywords

Physical behaviors; exercise; heart rate variability; vagal tone; isotemporal substitution

INTRODUCTION

Adults spend the majority of their waking time in sedentary behavior [1], defined as any activity that occurs in a lying, reclining, or seated posture and has an energy expenditure of ≤ 1.5 metabolic equivalents [2]. Sedentary behavior is emerging as a risk factor, independent of physical inactivity, for many unfavorable health outcomes including cardiovascular disease (CVD) [3-5]. Recently, quantitative and non-quantitative sedentary behavior guidelines have been established in response to this novel risk factor [6 7]. A common strategy recommended across these guidelines is to generally replace sedentary time (ST) with physical activity, i.e. to ‘sit less and move more,’ to improve health [6]. Yet, these guidelines often lack specific recommendations about whether intensity, duration, and types of physical activity is important when replacing ST and whether reducing long bouts of ST is more important than reducing short bouts of ST.

Heart rate variability (HRV), the measurement of healthy variation in time intervals between consecutive heartbeats in response to respiration, is a measure of cardiac autonomic regulation [8]. HRV is a subclinical marker of CVD where reduced HRV has consistently been associated with CVD events and mortality [9]. Reflecting some preliminary epidemiological and experimental studies finding that higher or increased ST was correlated to lower/reduced HRV [10-12], lower HRV is proposed as an important linking mechanism between ST and CVD [13]. Notably, the current epidemiological studies that have examined associations between ST and HRV have yielded inconsistent findings (i.e., negative, positive, or no associations) [12 14-18]. This inconsistency may be explained by important limitations in some of these studies, including not using gold standard methods for HRV measurement (i.e., ECG), different domains of physical activity (i.e.,

leisure, occupational, total), variable methods of classifying the intensity of device-measured activity (i.e., counts per minute, duration of MVPA), and self-reported measures of ST and physical activity that do not consider the interrelatedness of ST, light (LPA), and moderate-to-vigorous intensity physical activity (MVPA). The latter limitation is particularly crucial because experimental aerobic MVPA intervention studies have consistently found that exercise training improves HRV [19-21]. Research that considers these important limitations is needed to clarify associations between ST and HRV.

Emerging studies have demonstrated the importance of simultaneously considering all physical activity behaviors (i.e., MVPA, LPA, ST) to accurately understand the associations between behavior changes and better health. This approach recognizes that an increase in time spent performing one physical behavior must result in a decrease in time spent performing another physical behavior [22]. As such, determining which behavioral *exchanges* are associated with health benefits can most clearly inform translatable activity interventions. This issue can be statistically addressed by using isotemporal substitution analysis, a statistical framework that estimates the effect of reallocating time spent in one behavior for an equal amount of time spent in another behavior [23]. Specifically, utilization of this statistical technique can allow for estimation of the hypothetical effects of replacing overall, longer bouts of ST, or shorter bouts of ST with an equal amount of time in various intensities of physical activity on HRV. This is an important advantage of this statistical model because it could help inform more specific sedentary behavior guidelines to promote cardiovascular health.

Lastly, addressing these research gaps among women is important. Compared to men, women typically have lower levels of MVPA [24] and have greater increases in CVD risk development as they progress from young adulthood to middle age [25]. Emerging evidence also indicate that higher LPA is associated with improved cardiometabolic outcomes such as lipids and insulin level, which can lead to lower CVD risk in young and middle-aged women [26 27]. However, Isotemporal associations between ST, LPA, MVPA, and HRV have not been examined in this population, yet could explain CVD risk development and inform preventive interventional strategies during this critical period. Therefore, the primary aim of this study was to assess the effects of statistically substituting accelerometer-measured ST with LPA and MVPA on HRV in previously pregnant women. We hypothesized that replacing ST with both LPA and MVPA would be associated with higher (i.e., better) HRV. Additional aims evaluated whether associations differed when moderate (MPA) and vigorous physical activity (VPA) were considered separately or when ST and MVPA were separated into shorter and longer bouts.

Methods

Study design and population

This study was a secondary, cross-sectional analysis of data from the follow-up study of the Pregnancy Outcomes and Community Health (POUCH) Study [28]. Briefly, the POUCH Study was a multi-racial (white, African-American, and others) cohort that enrolled 3019 women during pregnancy to prospectively examine the pathophysiological pathways that lead to preterm delivery. The POUCH study oversampled women who had preterm delivery

(<37 weeks gestation) or who were at higher risk of preterm delivery due to increased CVD risk and compared them to women who had normal term delivery creating a sub-cohort focused on preterm birth and fetal growth issues. Because more detailed pregnancy-related data were collected on this sub-cohort, they were the target for recruitment in the follow-up POUCHmoms study; this follow-up study sought to perform in-depth investigations of early evidence of CVD risk after birth and obtained follow-up data 7 to 15 years after delivery (between 2011-2014) among a subset of these women (n = 1371) [29]. However, in our current analyses, we employed sampling weights to account for oversampling of these higher risk women and provide estimates that are representative of the original sample and, thus, a more general population [30].

To be included in POUCHmoms, women could not have been currently pregnant or pregnant within the past six months. Of 1371 women who were invited, 678 women participated in the follow-up assessment [28].

To be included in the current analysis, participants additionally had to have valid accelerometry data measuring ST and physical activity along with HRV measurement of sufficient quality defined as 1) at least 5 minutes HRV data, 2) artifacts of < 5 %, 3) and free of any abnormal signals (i.e., ectopic beats, arrhythmic events, missing data, and noise). All participants provided written informed consent. This follow-up study was approved by the Institutional Review Boards of the University of Michigan and the University of Pittsburgh.

Measurements

ST and physical activity—The POUCHmoms study used a daytime waist-wear protocol to measure daytime activity behavior. A subset of participants received a tri-axial accelerometer (ActiGraph GT3X+, ActiGraph LLC, Pensacola, FL, USA), elastic waist belt, and an activity diary. According to the current guidelines [31], the participants were instructed to wear the monitor around their waist using the elastic belt for 7 days. They were instructed to take the monitor off only for bathing or showering. If the monitor was removed for more than 5 minutes, participants were instructed to record the exact time of removal in the activity diary. ActiLife® software was used to initialize the monitor and to process the collected data. The sampling rate of the monitor was set at 30 Hz; data were integrated into 60-second epochs according to the current accepted standards [32]. Non-wear time was defined as consecutive periods of ≥ 60 minutes of zero counts per minute (cpm) [31].

Participants were required to have at least 3 days of 10 hours of waking wear time for the measurement to be considered valid [33]. ST and activity were calculated from accelerometry data using standard methods and cut points: epochs with < 100 cpm were considered “ST” [34]; epochs 101-2690 cpm were considered “LPA”; epochs 2691 - 6166 cpm were considered “MPA”; epochs ≥ 6167 cpm were considered “VPA”; thus, epochs with ≥ 2691 cpm were considered “MVPA” [32]. In addition, reflecting preliminary evidence that prolonged ST could be more harmful for cardiovascular health [35 36], ST was partitioned into long-bout ST (bouts lasting ≥ 30 minutes) and short-bout ST (< 30 minutes). To evaluate the potential influence of physical activity patterns [37], we also partitioned MVPA into long-bout MVPA (bouts lasting ≥ 10 minutes) and short-bout MVPA (< 10 minutes).

Resting HRV—HRV was measured using an electrocardiogram (ECG) at the POUCHmoms follow-up visit. Participants were instructed to fast for at least 8 hours prior to the study visit. Upon arriving, several assessments were conducted, including blood sample collection and self-reported questionnaires, followed by a 45–60-minute snack break. Thereafter, ECG measurements were obtained while participants were seated quietly in a chair with both feet flat on the floor. Two electrodes were placed on the participant's upper chest, and one electrode was placed on the participant's abdomen to record resting ECG signals using the Biopac MP36RWSW system (Goeta, CA). Sampling rate was set at 1000 Hz. Thereafter, 6 minutes of ECG signals were recorded and were later exported as AQC files (Biopac AcqKnowledge). AQC files were imported into Kubios Premium HRV analysis software (version 3.3.1, MATLAB, The MathWorks, Inc) for processing and deriving HRV indexes.

Established guidelines were followed to calculate HRV from ECG signals [8]. Among participants with at least 5 minutes of data, the automatic correction was employed to detect artifacts. Any files that had > 5 % artifacts were immediately excluded. Thereafter, files that had ≤ 5 % artifacts underwent further visual evaluation for noise, distortion, missing or premature R waves, ectopic beats, arrythmias, or irregular rhythms; abnormal samples were corrected if possible according to the guidelines [8] or otherwise excluded. To account for the potential effects of respiratory maneuvers, changes in or extremes of respiratory rate on HRV, Kubios Premium software estimated the respiration rate from ECG using the amplitude of R waves (ECG-derived respiration rate) [38] to use in sensitivity analyses (described below). We selected HRV indices that have a well-understood physiological and statistical basis and predict CVD outcomes. As such, heart rate, the standard deviation of normal R-R intervals (SDNN; representing the overall variability), and the root mean square of the successive differences (RMSSD; representing cardiac parasympathetic activity that is modulated by respiration) were selected as outcomes of interest.

Covariates—The POUCHmoms follow-up visit linked prospectively collected pregnancy data from the POUCH study and measured confounders of our hypothesized associations. Demographic, lifestyle, and health-related factors including age, race (i.e., non-Hispanic white, African American, or other), education (i.e., high school or less, some college, or college degree), type of health insurance (i.e., private, Medicaid, or none), and current smoking status (i.e., yes or no) were self-reported. In addition, waist and hip circumferences were measured in triplicate with a Gulick tape measure (85417, Creative Health Product, USA). The average of hip and waist measurements was used to calculate waist-to-hip ratio (WHR). Following five minutes of seated rest, systolic (SBP) and diastolic (DBP) blood pressures were measured three times using an Omron HEM-907 (Omron Healthcare, Inc.; Lake Forest, IL) with an appropriately sized cuff. The average of the second and third measurements was calculated as the resting blood pressure [39]. Women with SBP ≥ 140 mmHg or DBP ≥ 90 mmHg or who reported using anti-hypertensive medications were classified as hypertensive (HTN). The presence of diabetes (DM) and/or glucose-lowering medications were self-reported. Finally, important underlying medical conditions that can affect autonomic function and HRV (e.g., hypoglycemia, post-traumatic stress disorder

[PTSD], carpel tunnel syndrome, heart flutters, neuropathy, or cardiac problems) were reported by participants during an in-person interview.

Analytical method—Participant characteristics were summarized descriptively as means with standard deviations (for normally distributed variables), medians with 25th and 75th percentiles (for non-normally distributed variables), or numbers and percentages, as appropriate. Characteristics of included versus excluded women were compared using independent t-tests for continuous variables and χ^2 test for categorical variables. Outcome variables that were not normally distributed (i.e., SDNN and RMSSD) were natural log transformed. Confounders were defined *a priori* by constructing a directed acyclic graph (DAG) (see Figure, Supplemental Digital Content 1). Pearson's correlations between R-R intervals with heart rate, lnSDNN and lnRMSSD were also checked [40].

To address our aims, isotemporal substitution models were constructed to examine the associations of statistically exchanging ST and physical activity (i.e., LPA and MVPA) on heart rate and HRV, while holding wear time constant [23]. To elaborate, we used accelerometer measured time spent in each activity behaviors (i.e., ST, LPA, MPA, VPA, and MVPA) as well as total activity time (i.e., wear time) for each woman. Regression models were constructed by adding wear time and all activity behaviors except for one activity behavior at a time that was dropped out due to collinearity. As such, these models held the total activity time constant and allowed the included activity behaviors to increase at the expense of the dropped activity behavior.

To facilitate the interpretations, we rescaled the time unit of each behavior to 30 minutes/day. Models were specified, for example, as $\mathbf{heart\ rate\ or\ HRV_{index} = \beta_0 + \beta_1(LPA) + \beta_2(MVPA) + \beta_3(wear\ time),}$ where β_1 represented the effects of replacing 30 minutes of ST with the same amount of LPA, and (β_2 represented the effects of replacing 30 minutes of ST with the same amount of MVPA, while keeping the total wear time constant. Similar models replaced $\boldsymbol{\beta_1}$ with ST to additionally estimate the effect of replacing LPA with MVPA, and so forth. We repeated the isotemporal modelling strategy used above in expanded analyses to evaluate i) differential effects of VPA and MPA (i.e., separately considering ST, LPA, MPA, and VPA), and ii) patterns of ST and MVPA accumulation (i.e., by partitioning the overall duration of ST and MVPA into time spent in shorter and longer bouts). Consistent with previous studies [41 42], we have performed two levels of confounding control (i.e., level 1 controls for age, race, education, and medical insurance, and level 2 further controls for HTN, DM, antihypertensive medication, glucose-lowering medications, and WHR); as results were similar, only fully adjusted models (i.e., level 1 + level 2) are presented.

In sensitivity analyses, we excluded participants with underlying medical conditions that can affect autonomic function and HRV (e.g., hypoglycemia, PTSD, carpel tunnel syndrome, heart flutters, neuropathy, cardiac problems) and participants whose ECG-derived respiration rate was outside of the normal range (9-24 breaths/minute). Further, because most HRV indices have a positive correlation with heart period (i.e., as heart period increases, HRV indices also increase), some researchers have suggested that HRV should be adjusted for heart period or rate [40]. Therefore, adjusted HRV indices were calculated according to the current recommendations using the coefficient of variation (CV) technique as following:

$_{CV}$HRV index = 100 x HRV index / heart period [40]. Then, a final sensitivity analysis was conducted using the adjusted HRV indices to evaluate the potential influence.

Because women who had preterm delivery or were at higher risk of preterm delivery were oversampled in POUCHmoms, sampling weights were applied to all analyses. Stata version 15.0 (StataCorp, College Station, TX) was used to conduct all statistical analyses. The significance level was set as $\alpha \leq .05$.

Results

A total of 678 women completed the POUCHmoms follow-up assessment visit (Figure 1). Of them, 604 women had sufficient ECG records for 5 minutes of HRV analysis. Of these women, 82 participants had invalid HRV records due to the following reasons that prevented HRV calculation: ECG distortion (n = 49), arrythmia/irregular ECG (n = 20), >5% artifacts (n = 10), excessive noise (n = 2), and file error (n = 1). Thus, 522 women had valid 5 minutes of HRV data. Also, only a subset of women received accelerometers due to limited devices (n=416). Of these, n=348 had sufficient wear time for inclusion. Overall, 286 women had both valid HRV records and accelerometer data and were included in the current analyses. Compared to included women, excluded women (n=392) tended to be younger, non-white, less likely to smoke, had higher SBP and DBP, and more frequent use of anti-hypertensive medications, and a higher HTN prevalence (see Table, Supplemental Digital Content 2).

Table 1 presents characteristics of the sample. The majority were white (67.8%), non-smoking (79.4%), and had private insurance (59.8%). On average, SBP and DBP values were in the normal range, though some participants had HTN (15.4%). Few participants (4.2%) had DM. Median accelerometer wear time was 15.0 hours/day, median LPA was 7.7 hours/day, median MVPA was 0.8 hours/day, and median ST was 6.3 hours/day The correlation between ST and LPA, ST with MVPA, and LPA with MVPA were $r=-0.634$ ($p<0.001$), $r=-0.403$ ($p<0.001$), and $r=0.113$ ($p=0.057$), respectively.

Though replacing 30 minutes/day of ST with LPA, MPA, VPA, and MVPA yielded associations in a generally favorable direction with heart rate (lower) and HRV (higher) in fully adjusted models (Table 2 and 3), none of these associations reached statistical significance ($p>0.05$). Therefore, we failed to support our primary hypothesis. Yet in most cases, the magnitude of these associations appeared to be higher as the intensity of physical activity increased. In addition, exchanging 30 minutes/day of lower intensity physical activity with higher intensity physical activity (i.e., LPA with VPA; MPA with VPA) tended to also have favorable, yet nonsignificant, associations with heart rate (lower) and HRV (higher). Similar results were observed when these models excluded women with potential underlying medical conditions that could have affected HRV and women who did not meet ECG-derived respiration rate criteria (see Tables, Supplemental Digital Content 3 and 4). Comparable associations were also observed when the adjusted cvHRV indices were utilized (data not shown).

Finally, to examine the role of activity patterns (our secondary objective), we also partitioned MVPA and ST into time accumulated in shorter and longer bouts and repeated the isotemporal substitution models (Table 4). Replacing 30 minutes/day of long-bout ST with other behaviors resulted in more favorable, but mostly not statistically significant, associations with heart rate (lower) and HRV (higher) compared to replacing short bouts of ST. Yet, replacing 30 minutes/day of long-bout ST and short-bout ST with long-bout MVPA resulted in statistically significant associations with greater lnRMSSD (B=0.060±0.029; p=0.038 and B=0.055±0.026; p=0.039, respectively), supporting our secondary hypothesis. Moreover, when we repeated analyses after excluding women with potential underlying medical conditions that could have affected HRV and women who did not meet ECG-derived respiration rate criteria, the statistically significant associations when exchanging long- and short-bout ST with long-bout MVPA persisted (B=0.063±0.030; p=0.036 and B=0.056±0.027; p=0.040, respectively) (Table 5). Further, replacing 30 minutes/day of short-bout MVPA with long-bout MVPA became significantly associated with greater lnSDNN (B=0.074±0.037; p=0.047) and lnRMSSD (B=0.091±0.046; p=0.050) in these sensitivity analyses. Comparable associations were also observed when the adjusted cvHRV indices were utilized (data not shown).

Discussion

This study examined the effects of statistically reallocating ST and various intensities of physical activity on cardiac-autonomic regulation, including resting heart rate, lnSDNN, and lnRMSSD, in previously pregnant women. Our main findings were that exchanging 30 minutes/day of ST, LPA, MPA, VPA, or MVPA was not significantly associated with HRV indices. However, once partitioned into short and long bouts, statistically significantly beneficial relationships with lnRMSSD were detected when short-bout and long-bout ST were replaced with long-bout MVPA. Moreover, sensitivity analyses excluding women with underlying medical conditions and non-standard respiration rates strengthened these associations and resulted in additional, favorable associations with lnSDNN and lnRMSSD when replacing short- with long-bout MVPA. Importantly, the changes of these beta coefficients were small in the magnitude, which makes the clinical relevance of these differences unclear.

Many epidemiological studies, in addition to meta-analyses of experimental interventions, have demonstrated beneficial effects of physical activity, especially MVPA, on various HRV indices in adults [43-45]. On the other hand, epidemiological studies associating ST with HRV indices are rare, do not often evaluate associations specifically in women and have reported inconsistent results (i.e., unfavorable, favorable, or no associations) [12 14-16]. The source of this discrepancy is not entirely clear and could potentially be due to differences in sample size and characteristics, differences in assessment methodology, potentially differing effects of occupational and leisure MVPA on HRV and cardiovascular health [46 47], and highly variable study designs and statistical approaches to evaluating associations.

The most comparable studies to ours are those using a compositional and/or isotemporal substitution analysis approach to evaluate how ST and physical activity are associated with HRV. In one example of a cross-sectional study, a compositional analysis of data from

Canadian adults (n = 7,776; age: 18 – 79 years old) found that a higher proportion of time spent in MVPA relative to ST, LPA, or sleep was associated with lower resting heart rate [17]. Comparably, a significant favorable association with resting heart rate was detected when 60 minutes/day of ST was reallocated to MVPA but not to LPA measured using a thigh-worn accelerometer among 93 older adults [18]. Furthermore, a recent publication involving middle-aged adults (n = 1,668) from the Coronary Artery Risk Development in Young Adults (CARDIA) study revealed significant favorable associations with lnRMSSD when one standard deviation of waist-accelerometer-measured ST was replaced with LPA and VPA, but not with MPA. In the same study, only replacement with VPA resulted in a significant favorable correlation with lnSDNN [48]. Herein, we found nonsignificant associations with HRV indices when 30 minutes/day of ST was reallocated to LPA, MPA, VPA, or MVPA. Thus, research has typically associated MVPA with favorable HRV while studies associating ST with HRV have reported mixed and inconclusive results. Future longitudinal and experimental investigations are needed to draw stronger conclusions about the relationship between ST and HRV.

We also considered the intensity of the reallocated physical activity as a potentially important factor for healthier HRV. Indeed, this hypothesis is supported by several randomized controlled trials that reported greater HRV improvements following higher vs. lower intensity of physical activity programs [19 49]. In harmony with this, the previously mentioned CARDIA study observed higher favorable associations with lnRMSSD and lnSDNN when ST was replaced with VPA compared to MPA or LPA [48]. Although nonsignificant, potentially due to our smaller sample size, we found similar results where the associations with HRV indices were of their greatest magnitude when ST was reallocated to VPA. Altogether, these findings may indicate that the potential benefit to HRV when reallocating ST to physical activity is intensity dependent. Yet, further research that experimentally replaces ST with different intensities of physical activity is needed to confirm this hypothesis.

In addition to the total time spent in these behaviors, emerging evidence suggests that different patterns of activity behaviors may differentially influence health outcomes [50 51]. Noteworthy is that the 2018 Physical Activity Guidelines for Americans recommended, for the first time, that ‘any bout of MVPA counts’ and to ‘sit less and move more’ to improve health [6]. Yet, the lack of evidence regarding the role of ST bout length and the importance of breaking up prolonged sitting, along with the importance of comparing long- and short-bout physical activity in future research, were also highlighted in the Guidelines Committee final report as areas in need of future research [52].

Herein, we found significant favorable associations with lnRMSSD when replacing either type of ST (long-bout [≥30 minutes] and short-bout [<30 minutes]) specifically to long-bout (≥10 minutes) MVPA. Furthermore, our sensitivity analyses revealed additional significant favorable associations with both lnSDNN and lnRMSSD when exchanging short- (<10 minutes) for long-bout MVPA. We are aware of only one other study in older adults (n = 93) that examined the role of bouts that found a significant favorable association with resting heart rate when ST was reallocated to short-bout, but not long-bout, MVPA [18]; this later finding might reflect that older adult population being studied accumulated almost

all of their MVPA as short-bout MVPA, with very little long-bout MVPA. Together, these findings suggest that different patterns and bouts of activity behaviors may be differently associated with HRV indices. Further research applying emerging statistical approaches (e.g., compositional data analysis, exposure variation analysis) could provide more specific MVPA and ST recommendations.

Several physiological mechanisms have been proposed to explain the influences of ST and MVPA on HRV. MVPA is believed to improve HRV mainly through increasing cardiac vagal activity [53]. This vagal improvement may be ascribed to increased nitric oxide (NO) bioavailability, heightened oxytocin concentration, and/or suppressed angiotensin II, all of which can exert direct and/or indirect favorable effects on the vagal nerve [53-55]. MVPA can also enhance blood/plasma volume, which may induce baroreflex-mediated increased vagal activity [56]. However, one hypothesis suggests that these physiological benefits may be reversed by excessive ST. To elaborate, frequent exposure to sedentary behavior is suggested to cause chronic reductions in shear stress and, eventually, decreased NO bioavailability [57]. In addition, sedentary behavior, especially prolonged sitting, may lead to decreased blood/plasma volume [58]. These reductions in NO bioavailability and blood/plasma volume may lead to attenuated vagal activity and, thus, lower HRV. Our significant results are consistent with these proposed mechanisms, where reallocating long- and short-bout ST specifically with long-bout MVPA was associated with higher resting vagal activity (i.e., higher lnRMSSD). Thus, our findings support future experimental research examining and confirming these physiological mechanisms that link ST to unfavorable HRV.

Our study has several strengths that are worth highlighting. Our unique sample of women come from a multi-racial cohort, which improves the generalizability of our findings. Thus, our results are most relevant to young-to-middle aged women. Decreasing ST and increasing MVPA are potential behavioral targets to improve cardio-autonomic health in this population. Moreover, we used the gold standard field-based measurement of physical activity (i.e., accelerometer), allowing us to evaluate associations across various intensities of activity, to compare total, long-bout, and short-bout ST and MVPA, and to account for the interrelation between ST and physical activity via isotemporal substitution. Lastly, we also used gold standard assessment of HRV by ECG and carefully implemented robust guidelines to process the ECG data.

Still, several limitations should be considered when interpreting our results. Our study was observational and cross-sectional, making it susceptible to biases such as reverse causality and residual confounding. Future studies with longitudinal designs that establish temporality or experimental studies that manipulate ST, LPA, and MVPA are needed. Furthermore, most of the women included in these analyses exceeded MVPA guidelines; thus, our results may not apply to less active populations. Further, though the original study (i.e., the POUCH Study) was a community-based sample of pregnant women, only a small subsample was included in this analysis. Despite the use of weighting to address the sampling scheme in POUCHmoms, losses to follow-up or due to poor quality data could have affected the representativeness of our sample. Our small sample size might have also limited our ability to detect small associations.

Although the waist-worn GT3X accelerometer that we utilized is a highly validated measure of MVPA, it has been found to be less accurate to measure ST as compared to a thigh-worn monitor (i.e., activPAL) [59]. This could have resulted in misclassification of activity and attenuated results; future studies with more precise measurement of ST are needed. In addition, future research of associations between device-measured activity and HRV could use emerging methods such as pattern recognition to improve interpretation and reduce misclassification across the activity spectrum that can occur using the standard approaches we used herein [31]. Lack of a precise, direct measure of respiration rate is another limitation to our study and should be considered in future research [8]. Lastly, HRV only reflects overall and cardiac-parasympathetic activity when resting. As such, the associations of reallocating total and short- and long-bout of ST to LPA and MVPA with cardiac-sympathetic activity remain to be evaluated.

Conclusion

Altogether, our results provide some evidence suggesting that ST unfavorably affects HRV indices in previously pregnant women. Replacing ST with long-bout MVPA may counteract these effects and elicit a beneficial influence on HRV. Moreover, additional benefit to HRV may be achieved by reallocating short- to long-bout MVPA (e.g., replacing sporadic walking with more continuous brisk walking of at least 10 minutes in duration) in healthy previously pregnant women without existing cardiovascular or other conditions that could impact HRV. Our findings may contribute mechanistic insight into the pathway between high ST, low MVPA, and CVD risk development as cardiac-autonomic dysregulation may be a potential linking mechanism between ST, MVPA, and CVD in previously pregnant women. Lastly, our study also provides insight into specific MVPA and ST prescriptions. In addition to the current recommendations, women, particularly previously pregnant ones, may reduce ST and replace it with MVPA accumulated in bouts ≥10 minutes in length to achieve better cardiac-autonomic health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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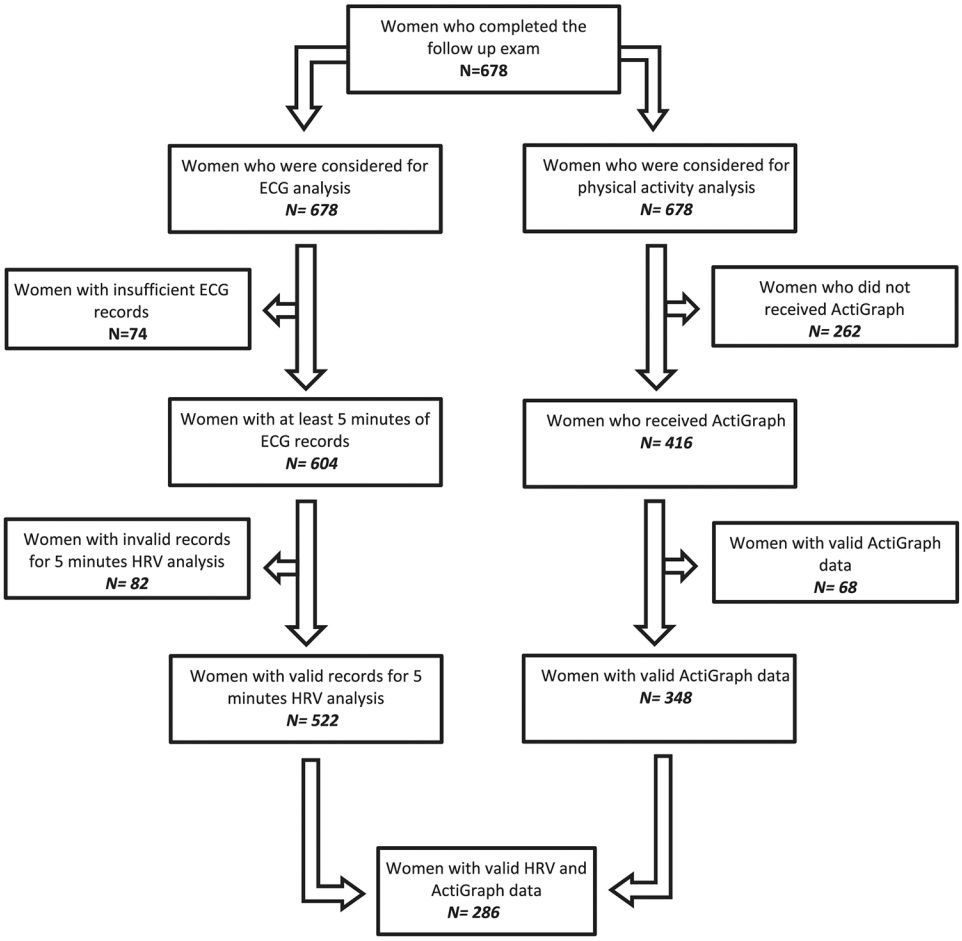

Figure 1.
Flowchart of women who completed ECG and accelerometer measurements.

Table.1

Characteristics of Participants (n=286)

Characteristic	Mean (SD), Median (25^{th}– 75^{th}), or n (%)
Age (years)	32.6 (5.7)
Race	
White	194 (67.8)
African American	77 (26.9)
Other	15 (5.2)
Education	
High School or Less	53 (18.5)
Some College	132 (46.2)
College Degree	101 (35.3)
Insurance	
Private	171 (59.8)
Medicaid	87 (30.4)
None	28 (9.8)
Currently Smoking	
No	227 (79.4)
Yes	59 (20.6)
Waist-to-Hip Ratio	0.8 (0.1)
Systolic Blood Pressure (mmHg)	112.7 (12.9)
Systolic Blood Pressure ≥140 mmHg	12 (4.2)
Diastolic Blood Pressure (mmHg)	74.5 (10.8)
Diastolic Blood Pressure ≥90 mmHg	21 (7.3)
Hypertension	
No	242 (84.6)
Yes	44 (15.4)
Using Medication	24 (54.6)
Not Using Medication	20 (45.5)
Diabetes	
No	274 (95.8)
Yes	12 (4.2)
Using Medication	5 (41.7)
Not Using Medication	7 (58.3)
Resting Heart Rate (beats/minute)	75.8 (9.4)
SDNN ln(ms)	3.6 (0.4)
RMSSD ln(ms)	3.4 (0.6)
Wear Time (min/day)	901.8 (847.5 – 950.8)
ST (min/day)	376.8 (324.5 – 435.3)
Short-bout ST (<30 min/day)	264.3 (157.8 – 333.3)
Long-bouts ST (≥30 min/day)	95.2 (57.6 – 213.8)

Characteristic	Mean (SD), Median (25^{th}– 75^{th}), or n (%)
LPA (min/day)	459.8 (395.2 – 528.0)
MPA (min/day)	40.2 (28.5 – 61.8)
VPA (min/day)	4.0 (2.0 – 8.8)
MVPA (min/day)	46.2 (32.0 – 69.2)
Short-bout MVPA (<10 min/day)	30.1 (16.7 – 43.2)
Long-bout MVPA (≥10 min/day)	16.0 (0.0 – 31.2)

ln: natural logarithm, mmHg: millimeters of mercury, ms: milliseconds, LPA: light physical activity, MPA: moderate physical activity, MVPA: moderate-to-vigorous physical activity, ms: millisecond, RMSSD: root mean square of successive differences, SDNN: standard deviation of normal R-R intervals, ST: sedentary time, VPA: vigorous physical activity.

Table 2.

Isotemporal Associations of Replacing 30 Minutes/Day of ST, LPA, and MVPA with Heart Rate and HRV in Women (n=286)

Outcomes	Heart Rate (beats/minute)	SDNN ln(ms)	RMSSD ln(ms)
	B±SE (p-value)	B±SE (p-value)	B±SE (p-value)
Replacing ST with LPA	−0.150±0.225 (0.504)	0.006±0.012 (0.596)	0.014±0.009 (0.125)
Replacing ST with MVPA	−0.309±0.365 (0.398)	0.018±0.023 (0.418)	0.007±0.018 (0.676)
Replacing LPA with MVPA	−0.158±0.418 (0.705)	−0.006±0.021 (0.759)	0.012±0.026 (0.644)

Bold indicates significant difference (p≤0.05). B: beta coefficient; ln: natural logarithm; LPA: light physical activity; ms: milliseconds, MVPA: moderate-to-vigorous physical activity; RMSSD: root mean square of successive differences; SDNN: standard deviation of normal R-R intervals; SE: standard error; ST: sedentary time.

All models adjusted for age, race, education, insurance, smoking, hypertension, diabetes, antihypertensive medication, glucose-lowering medications, and waist-to-hip ratio.

Table 3.

Isotemporal Associations of Replacing 30 Minutes/Day of ST, LPA, MPA, and VPA with Heart Rate and HRV in Women (n=286)

Outcomes	Heart Rate (beats/minute)	SDNN ln(ms)	RMSSD ln(ms)
	B±SE (p-value)	B±SE (p-value)	B±SE (p-value)
Replacing ST with LPA	−0.167±0.231 (0.471)	0.018±0.010 (0.075)	0.009±0.013 (0.475)
Replacing ST with MPA	−0.168±0.787 (0.831)	−0.031±0.045 (0.491)	−0.007±0.056 (0.902)
Replacing ST with VPA	−0.624±1.377 (0.651)	0.094±0.066 (0.153)	0.075±0.088 (0.391)
Replacing LPA with MPA	−0.002±0.863 (0.998)	−0.049±0.051 (0.330)	−0.016±0.062 (0.796)
Replacing LPA with VPA	−0.457±1.343 (0.734)	0.076±0.061 (0.219)	0.066±0.084 (0.432)
Replacing MPA with VPA	−0.455±2.026 (0.822)	0.125±0.106 (0.239)	0.082±0.137 (0.549)

Bold indicates significant difference (p≤0.05). B: beta coefficient; ln: natural logarithm; LPA: light physical activity; ms: milliseconds, MPA: moderate physical activity; RMSSD: root mean square of successive differences; SDNN: standard deviation of normal R-R intervals; SE: standard error; ST: sedentary time; VPA: vigorous physical activity.

All models adjusted for age, race, education, insurance, smoking, hypertension, diabetes, antihypertensive medication, glucose lowering medications, and waist-to-hip ratio.

Table 4.

Isotemporal Associations of Replacing 30 Minutes/Day of Long- and Short-bout ST, LPA, and Short- and Long-bout MVPA with Heart Rate and HRV in Women (n=286)

Outcomes	Heart Rate (beats/minute)	SDNN ln(ms)	RMSSD ln(ms)
	B±SE (p-value)	B±SE (p-value)	B±SE (p-value)
Replacing Long-bout ST with Short-bout ST	−0.102±0.137 (0.456)	0.006±0.007 (0.369)	0.006±0.009 (0.523)
Replacing Long-bout ST with LPA	−0.264±0.284 (0.352)	0.021±0.011 (0.070)	0.013±0.015 (0.382)
Replacing Long-bout ST with Short-bout MVPA	−0.019±0.507 (0.970)	−0.013±0.029 (0.647)	−0.011±0.036 (0.768)
Replacing Long-bout ST with Long-bout MVPA	−0.863±0.529 (0.104)	0.041±0.022 (0.060)	**0.060±0.029 (0.038)**
Replacing Short-bout ST with LPA	−0.162±0.222 (0.465)	0.015±0.009 (0.095)	0.007±0.012 (0.522)
Replacing Short-bout ST with Short-bout MVPA	0.121±0.497 (0.808)	−0.019±0.029 (0.518)	−0.016±0.037 (0.663)
Replacing Short-bout ST with Long-bout MVPA	−0.761±0.475 (0.111)	0.035±0.020 (0.082)	**0.055±0.026 (0.039)**
Replacing LPA with Short-bout MVPA	0.283±0.571 (0.620)	−0.034±0.032 (0.287)	−0.023±0.039 (0.50)
Replacing LPA with Long-bout MVPA	−0.598±0.491 (0.224)	0.021±0.023 (0.360)	0.047±0.029 (0.109)
Replacing Short-bout MVPA with Long-bout MVPA	−0.881±0.648 (0.175)	0.054±0.037 (0.139)	0.071±0.046 (0.123)

Bold indicates significant difference (p≤0.05). B: beta coefficient; ln: natural logarithm; LPA: light physical activity; ms: milliseconds, MVPA: moderate-to-vigorous physical activity; RMSSD: root mean square of successive differences; SDNN: standard deviation of normal R-R intervals; SE: standard error; ST: sedentary time.

All models adjusted for age, race, education, insurance, smoking, hypertension, diabetes, antihypertensive medication, glucose-lowering medications, and waist-to-hip ratio.

Table 5.

Isotemporal Associations of Replacing 30 Minutes/Day of Long- and Short-bout ST, LPA, and Short- and Long-bout MVPA with Heart Rate and HRV in Women without Chronic Conditions and who Met the Respiration Rate Criteria (n=264)

Outcomes	Heart Rate (beats/minute)	SDNN ln(ms)	RMSSD ln(ms)
	B±SE (p-value)	B±SE (p-value)	B±SE (p-value)
Replacing Long-bout ST with Short-bout ST	−0.139±0.144 (0.335)	0.008±0.007 (0.274)	0.007±0.009 (0.431)
Replacing Long-bout ST with LPA	−0.291±0.290 (0.317)	0.022±0.011 (0.057)	0.014±0.015 (0.356)
Replacing Long-bout ST with Short-bout MVPA	0.201±0.505 (0.691)	−0.029±0.027 (0.285)	−0.028±0.034 (0.416)
Replacing Long-bout ST with Long-bout MVPA	−1.057±0.540 (0.051)	0.044±0.023 (0.055)	**0.063±0.030 (0.036)**
Replacing Short-bout ST with LPA	−0.152±0.226 (0.502)	0.014±0.009 (0.112)	0.007±0.012 (0.570)
Replacing Short-bout ST with Short-bout MVPA	0.340±0.495 (0.493)	−0.037±0.028 (0.194)	−0.035±0.036 (0.326)
Replacing Short-bout ST with Long-bout MVPA	−0.918±0.479 (0.056)	0.037±0.021 (0.084)	**0.056±0.027 (0.040)**
Replacing LPA with Short-bout MVPA	0.492±0.571 (0.390)	−0.051±0.031 (0.095)	−0.042±0.038 (0.274)
Replacing LPA with Long-bout MVPA	−0.766±0.504 (0.130)	0.022±0.024 (0.347)	0.049±0.030 (0.107)
Replacing Short-bout MVPA with Long-bBout MVPA	−1.258±0.641 (0.051)	**0.074±0.037 (0.047)**	**0.091±0.046 (0.050)**

Bold indicates significant difference (p≤0.05). B: beta coefficient; ln: natural logarithm; LPA: light physical activity; ms: milliseconds, MVPA: moderate-to-vigorous physical activity; RMSSD: root mean square of successive differences; SDNN: standard deviation of normal R-R intervals; SE: standard error; ST: sedentary time.

All models adjusted for age, race, education, insurance, smoking, hypertension, diabetes, antihypertensive medication, glucose-lowering medications, and waist-to-hip ratio and participants with diseases (n = 12) and who did not meet breathing frequency (n = 10) were excluded.