Abstract

Introduction: Sedentary behavior, specifically an acute bout of prolonged uninterrupted sitting is associated with heightened cardiovascular disease (CVD) risk, with increased arterial stiffness (AS) being implicated as a principal pathophysiological mechanism. The current systematic review, with meta-analysis, aimed to consolidate the AS response to: (1) prolonged uninterrupted- and (2) interrupted-sitting, as assessed by central- and peripheral pulse wave velocity (PWV). **Methods:** In total, 326 articles were identified, of which 11 and 7 met inclusion criteria for objectives: (1) and (2), respectively. Mean differences (MD) and 95% confidence intervals (CI) were calculated for all trials using a three-level random-effects model, with restricted maximum likelihood (REML) estimation. The amount of heterogeneity was estimated using Cochran's Q, and Higgins I^2 tests. **Results:** (1) Prolonged uninterrupted sitting resulted in a significant increase in carotid-femoral (cf) PWV (MD = 0.184 m/s, 95% CI = 0.098 to 0.270, $p < 0.0003$). (2) Interrupting bouts of prolonged sitting resulted in a significant increase in cf-PWV (MD = 0.127 m/s, 95% CI = 0.044 to 0.209, $p < 0.0026$), that was lower compared to the uninterrupted sitting. **Conclusion:** An acute bout of uninterrupted sitting appears to increase cf-PWV and whilst interrupting with brief physical activity it is of benefit; it does not fully mitigate the response.

Introduction

Repeated exposure to sedentary behavior, specifically, prolonged uninterrupted sitting, defined as waking behaviors consisting of <1.5 metabolic equivalents in seated, reclined, or supine postures [1] is associated with heightened cardiovascular disease (CVD) risk and all-cause mortality [2,3]. Previous reviews have identified this ubiquitous behavior is associated with impaired vascular endothelial function [4,5] as measured by flow-mediated dilation (FMD). FMD has been shown to be affected by prolonged periods of uninterrupted and interrupted sitting [4], and provides mechanistic insight into the early detection of subclinical atherosclerosis [6]. However, it suffers potential limitations that may limit its use in discerning vascular dysfunction related to prolonged sitting. For instance, FMD needs to be conducted on peripheral conduit arteries, and thus cannot assess central (aortic) vasculature dysfunction. requires high technical acumen [8,9], and has lengthier measurement and analysis duration, making it relatively challenging to perform, especially for large scale clinical trials.

Arterial stiffness (AS), defined as the resistance of the arterial wall to deformation [10,11], is a novel cardiovascular health biomarker that is increasingly recognized as a surrogate predictor of CVD and all-cause mortality, independent of other vascular risk factors [12]. As the stiffness of the arterial

system determines the rate at which the pressure pulse waveform propagates, its measurement — pulse wave velocity (PWV) – provides a reliable estimate of AS [13]. PWV measured in the carotid-femoral (cf-PWV) segment typically estimates aortic stiffness [13,12], with every 1 m/s increase purportedly augmenting CVD risk and mortality by 14% and 15% [12], respectively. In contrast, PWV measured at peripheral arterial sites (e.g., femoral-ankle [fa-PWV]) has limited and conflicting prognostic value [14]; however, it may provide insight into the acute vascular dysfunction over local arterial segments, specifically endothelial impairment [15]. Several factors, including measurement accuracy and precision, prospective validity, ease of use, and that it can assess vasculature, both centrally and at the periphery, favour its use over its contemporary biomarkers. Whilst structural dysfunction is least likely to happen post an acute bout of prolonged sitting, increasing evidence suggests that arterial function, specifically endothelial function can become impaired, as indicated by decreased FMD [4,5]. As such PWV might be a viable alternative to FMD to assess CVD dysfunction in acute sedentary behavior research. And although PWV has been used in numerous small-scale clinical trials, there is no collective synthesis of PWV data to determine whether there is an effect of sitting with and without interruptions on AS.

Whilst prolonged sitting appears to negatively impact CVD risk, recent literature alludes that simple sitting interruption strategies may off-set deleterious effects associated with it [16,17,18], presumably via an increase in the muscle pump, which drives a reduction in venous pooling, as well as a decrease in lower limb metabolic activity [19]. However, what is a sufficient stimulus to offset prolonged-sitting-induced-damage is unclear. If we are to develop sitting and sitting interruption guidelines, for which there are currently none, we need to synthesise the evidence. As such, the current systematic review, with meta-analysis, consolidates and quantifies the AS response to prolonged uninterrupted and interrupted sitting, as assessed by central and peripheral PWV.

Methods

This systematic review with meta-analysis was reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) guidelines [20]; however, the protocol was not pre-registered.

Data Sources and Searches

Three electronic databases (PubMed, Web of Science, and SPORTDiscus) were searched from inception to 5 January 2025. The search strategy combined the terms in the following domain: exposure (“prolonged sitting” OR “sedentary behav*” OR “sitting”), and outcome (“arterial stiff*” OR

"aortic stiff*" OR "PWV" OR "pulse wave velocity"). The search was limited to English language studies only.

Article Selection

Article screening and selection were performed using the *Covidence* software (Version 2), a screening and data extraction tool for conducting systematic reviews [21]. For the purpose of this meta-analysis, the terms 'article' and 'study' were used interchangeably; 'trial' will be the unit included in the meta-analysis. One article may result in more than one eligible trial as it could include more than one intervention group or PWV assessment site. Searches were conducted independently by two authors (AM and CP). First, duplicates were removed, then study titles and abstracts were screened for relevance, before finally reviewing full texts of potentially eligible articles. The reference lists of all relevant articles and reviews were also examined. Discrepancies in inclusion or exclusion were resolved by consensus (AM and CP), or through consultation with a third reviewer (SF). The inclusion criteria for the first objective of this review were: (i) Study design: randomised-controlled trials, randomised-crossover trials, and quasi-experimental trials (pre- versus post-test); (ii) Population: adult participants (≥ 18 years), free of autonomic or neuromuscular dysfunction and any other known chronic illness; (iii) Exposure: a prolonged sitting period of at least 1-hour; (iv) Outcome: assessment of any segmental measure of PWV; (v) PWV was assessed pre- and post-sitting in the supine or sitting posture. Additional inclusion criteria were used to address the second objective: (i) if a strategy was employed to disrupt the effects of sitting, (ii) there must have been a control (uninterrupted sitting) group or condition, and (iii) the interruption strategy must have involved the participants actively moving their limbs. If a study employed an intervention prior to, or after the sitting period, only data from the control (uninterrupted sitting) trial were included in the analysis.

Data Extraction and bias assessment

For each eligible trial, data were extracted relating to bibliographic information (author, publication year, country), sample characteristics (age, sex, body mass index [BMI], health status, ethnicity), PWV assessment (arterial segment, mean difference [MD], standard error [SE], duration of prolonged sitting, choice of device employed, duration of rest period, path length estimation, and co-variates), and details of any intervention aimed at interrupting sitting. Data were either unclear or not available for 7/11 included manuscripts, so the corresponding/first author was contacted (via email) to request the data. Of the 7 authors contacted, 6 responded within the stipulated timeline; however, we could not access or extract data from one study [30]. Data extractions were completed by the two authors (AM and CP) independently.

Study quality was assessed using the Cochrane Risk-of-Bias 2, which evaluates six domains: (1) selection bias, (2) performance bias, (3) detection bias, (4) attrition bias, (5) reporting bias, and (6) other bias, with each component rated as “low risk”; “some concerns”; or “high risk”. The 'intention-to-treat' model was assessed, per the review team’s study aim. Further, we counted blinding of the operator (to the outcome assessment) as a quality criterion since it’s challenging to blind participants for interruption interventions. Two reviewers (AM and LT) scored the studies independently, or in consultation with a third reviewer (SF) in case of disagreements.

Data Analysis

Data were analysed using the ‘metafor’ package (version 4.4.0) in R (version 4.3.1) [22]. As the primary aim of this meta-analysis was to determine the impact of sitting duration on PWV, data needed to be extracted at multiple timepoints from the same trial. So, a three-level random-effects model with REML estimation was used to determine the pooled effect sizes of cf-PWV (m/s), the primary outcome variable, and was reported as the MD. The choice of REML helped us account for sampling variance, study-level variance, and between-study variance and thus is able to deal with dependency between related data points [23]. We only meta-analysed cf-PWV, because there were sufficient number of trials available on this, thus a meta-analysis assessing peripheral PWV responses to uninterrupted sitting could not be performed. Central and peripheral PWV were not combined as they change differentially to perturbations and have variable prognostic values. Corresponding forest plots were generated for objective (1) and (2) independently. Heterogeneity was estimated using Cochran’s Q, and Higgins I^2 tests. The Higgins I^2 test interprets 25%, 25–75%, and >75% heterogeneity as small, moderate, and considerable, respectively [24]. The robustness of the pooled effect to identify potential outliers and/or influential trials was examined using the studentized residuals and Cook’s distance using previously described thresholds [25]. The rank correlation test and the regression test [26], using the standard error of the observed outcomes as predictor, were used to check for funnel plot asymmetry.

Results:

Literature Search and Trial Selection

Figure 1 depicts the PRISMA flow diagram. Preliminary database searches identified 326 potential articles. Most (304, including 121 duplicates) were removed following initial title and abstract screening as they failed to meet the inclusion criteria. Twenty-three articles were screened for full text with 11 studies included in the systematic review. The final meta-analyses for objective (1) and (2) included 8 trials (8 articles) and 6 trials (6 articles), respectively.

Characteristics of Included Studies

Individual trial characteristics are summarized in **Table 1**. Of the 11 studies included, 7 were conducted in the USA [16, 18, 27, 28, 30, 32, 33], 1 in the UK [29], 2 in Japan [19,31], and 1 in Kingdom of Saudi Arabia (KSA) [7]. The number of participants in each study ranged from 13.0 to 26.0 and were aged between 21.0 and 53.4 years. Most studies assessed both males (n = 102 [58%]) and females (n = 74 [42%]), two [29,33] (only male participants), and one [7] (only female participants) with mean body mass index (BMI) between 21.5 and 31.9 kg/m^2. All studies reported participants' health status (8 studies recruited healthy subjects whilst 3 studies recruited overweight or obese subjects). Most studies were published in the previous 6-years. Bouts of prolonged sitting ranged from 2.5 hours to 7.3 hours, with the median duration of 3 hours. For the uninterrupted sitting studies, cf-PWV was assessed in 8/11 studies [7, 16, 18, 27, 28, 29, 30, 32, 33], carotid-radial (cr-PWV) in 3/11 studies [16, 18, 30], femoral-ankle (fa-PWV) in 2/11 studies [29,32], and only one study assessed brachial-femoral (bf-PWV) [32]. In addition to an uninterrupted sitting condition, 7 studies explored the effects of breaking up of prolonged sitting via strategies such as standing (n = 2) [16,33], simple resistance activities such as calf raises, knee raises, squats (n = 3) [18,19,31], seated leg fidgeting (n = 1) [28], and moderate aerobic exercise prior to sitting (n=1) [7]. Of the studies that explored the effects of breaking up of prolonged sitting, 5 of the 7 assessed cf-PWV [7, 16,18,28,33], and 2 assessed cr-PWV [16,18], and brachial-ankle (ba-PWV) [19,31].

Methodological Quality Assessment

The risk of bias outcomes as assessed using the Cochrane Risk-of-Bias 2 Tool for each study is summarised in **Figure 2**. Most studies included in the systematic review were randomised cross-over trials [7,16,18,19,29,28,30,31,32] with the exception of 2 studies that were randomized control [27], and pseudo-randomized [33] trials. Overall, 4 out of the 11 included studies had 'some concern' [16,19,31,33] while the remaining 7 were classified as 'low risk' of bias [7,18,27,28,29,30,32]. Specifically, 6/11 studies provided details regarding the randomisation process [7,19,27,28,29,32] and 8/11 reported blinding of the outcome assessor [7,19,27,28,29,30,32,33]. While 4/11 studies do not report any sort of blinding for the assessment of outcome measures raising 'some concerns'

[16,19,27,31]. All studies were deemed as 'low risk' of bias, both for missing data and reporting of outcome.

Synthesis of results

Sitting without interruption

Cf-PWV was assessed in 8/10 uninterrupted trials, of which 4 found it significantly increased by between 0.20 and 0.30 m/s [16,27,28,33], while in the remaining 4 trials [7,29,30,32] no significant changes were reported. 3/10 studies assessed and found no significant changes in cr-PWV [16,18,30]. One study reported a significant increase in both bf-PWV (0.36 m/s) and fa-PWV (0.55 m/s) [32], the other found no change in fa-PWV [29]. Meta analytically, prolonged sitting resulted in a significant increase in cf-PWV (MD = 0.184 m/s, 95% CI = 0.098 to 0.270, $p < 0.0003$) as depicted in the forest plot (**Figure 3**). There was no significant amount of heterogeneity in the model: $\tau^2 = 0.003$, (Q (df=7) = 10.212, $p = 0.177$, $I^2 = 22.19\%$). Examination of studentized residuals identified no potential outliers, and Cook's distances reported none of the studies were considered overly influential. Neither the rank correlation ($p = 0.381$) nor the regression test ($p = 0.789$) indicated any funnel plot asymmetry (**Figure 4**).

Sitting with interruption

Four out of six studies assessed the effects of breaking up prolonged sitting on cf-PWV only [16,18,28,33], 2 on cr-PWV [16,18] and cf-PWV, whilst 2 assessed only ba-PWV [19,31]. Meta analytically, interrupting sitting resulted in a smaller but significant increase in cf-PWV (MD = 0.127 m/s, 95% CI = 0.044 to 0.209, $p < 0.0026$) as depicted in the forest plot (**Figure 5**). Although one study [33] had a relatively large weight (at least 3 times) compared to the rest of the studies, there was no significant amount of heterogeneity in the model: $\tau^2 = 0.000$, (Q(df=5) = 6.523, $p = 0.256$, $I^2 = 1.69\%$). Examination of studentized residuals identified no potential outliers, and Cook's distances reported that none of the studies were considered overly influential. Neither the rank correlation ($p = 0.817$) nor the regression test ($p = 0.487$) indicated any funnel plot asymmetry (**Figure 6**).

Discussion

The aim of this meta-analysis was to determine the effect of prolonged sitting with and without interruption strategies on central and peripheral PWV. We found that cf-PWV significantly increased following acute bouts of uninterrupted sitting. Whilst interrupting sitting also caused a significant increase in cf-PWV, the effect was smaller compared to uninterrupted sitting.

Sitting without interruption

Cf-PWV is the gold standard measure of large elastic AS (e.g., the aorta) and it has demonstrated prognostic value in predicting CVD outcomes [12]. So, it is perhaps unsurprising that most trials reported cf-PWV as their primary outcome measure [7,16,18,27,28,29,30,32,33], and only two studies assessed ba-PWV [19,31], the other clinical predictor of CVD (popular across Asia) [36] (**Table 1**). A 1 m/s chronic increase in cf-PWV has been associated with a 14%, 15%, and 15% increase in the risk of future CV events, CV mortality, and all-cause mortality, respectively [12]. Whilst our pooled cf-PWV data (MD = 0.184 m/s) appears moderate **(Figure 3)**, it is likely reflective of the acute changes to singular sitting periods. However, repeated exposure to these short acute uninterrupted sitting bouts may compound the PWV response over time. This may be even more so in populations which have pre-existing CVD risk factors (hypertension, diabetes, obesity, and menopause), although this is speculation and beyond the scope of this paper [59]. The acute 0.3 – 0.6 m/s increase in cf-PWV, reported in **Table 1** seems more prominent in young and healthy compared to middle-aged adults, and those with metabolic dysfunction (**Figure 3**). This could be in part due to a blunted BP response to the renin-angiotensin-aldosterone system (RAAS) seen with aging [16,37], as reported in a recent meta-analysis which found increases in SBP and MAP were more pronounced in younger age groups in response to prolonged sitting [38]. This together with structural arterial stiffening a priori, may be an insufficient stimulus to elicit a meaningful response. Longitudinal data from the Whitehall II study on older adults reported an increase in aortic stiffness of 0.76 m/s (95% CI: 0.69, 0.83) over a five-year period. However, increased levels of moderate-to-vigorous physical activity and a reduction in sedentary behavior were linked with a slower progression of aortic stiffness, independent of traditional cardiovascular risk factors. This may be in part due to the protective effects of physical activity on arterial stiffening, which can help mitigate the structural changes due to age.

Unlike most exercise science studies that primarily focus on male participants [58], the majority of the trials in our analysis [16, 18, 19, 27, 28, 30, 31, 32] recruited equal number of young and healthy female participants (**Table 1**). Previously, biological sex has been shown to alter AS, with higher stiffness reported in men than women. Whilst higher estrogen concentration provides a direct protective arterial effect in women than men, particularly through nitric oxide (NO) production, elevated testosterone concentrations in men potentially increase AS via increases in the sympathetic nervous system and RAAS activation [60]. Whilst the menstrual cycle phase may affect AS due to fluctuations in sex hormones (estrogen and progesterone) across a cycle, the evidence is still inconclusive. This is in part because of differences in both the methods of AS assessment, and the

arterial segments used. Studies that have assessed changes in central AS (cf-PWV) between difference phases of the menstrual cycle, found no significant changes [61, 62]. Contradicting this, carotid artery stiffness was higher in the follicular phase and at ovulation, compared to the luteal phase and during menses [63]. The paucity of comprehensive evidence in local and central AS not only highlights the importance of assessing them between men and women but also needs to be assessed during menstrual cycle phases in future investigations. Given the well-documented influence of hormones on AS in women [39,40], it is imperative that future research should explore potential sex differences in order to provide a wider, and more inclusive public health message.

Given the known interactions of peripheral (cr-, ca-, fa-, bf-PWV) to central (cf-PWV) AS, a number of articles [16,18,29,30,32] explored the impact of sitting on both, despite the limited clinical predictability of peripheral AS on CVD outcomes [14]. Whilst the two studies assessing lower limb arteries (fa-PWV) had contrasting outcomes (0.55 m/s significant increase [32]) and no significant change [29]), none of the upper limb arteries (cr-PWV) seem affected by prolonged sitting (**Table 1**).

Sitting with interruption

Seven of the included eleven studies investigated the effect of sitting interruption strategies (standing breaks [n = 2] [16,33], simple resistance exercises [n = 4]) [18,19,28,31], and moderate aerobic exercise prior to sitting (n=1) [7] on cf-PWV, of which only one by Wright et al. [33] found a significant decrease (strategy: standing breaks), while the remaining five found no effect (**Table 1**). Previous trials suggested that an increase in endothelium-mediated shear stress [16,28,41,59], a reduction in venous pooling [16,28] and a reduced interdependence on BP [42, 59] are the cornerstone mechanisms that may explain reduced PWV with interrupted sitting. Contrary to these hypotheses, our pooled sitting interruption data [16, 18, 28, 32, 33] showed a marginally less (compared to prolonged uninterrupted sitting) yet significant increase in cf-PWV (MD = 0.127) **(Figure 5)**. This is similar to Whitehall II study which found an increased moderate to vigorous physical activity resulted in a smaller but significant increase in PWV of 0.16 m/s (95% CI: 0.32, 0.002) compared to individuals who did not change their activity levels. Importantly, these models were adjusted for physical activity levels and were independent of cardiovascular risk factors [34]. The differing results could be in part due to variability in the interruption strategies employed **[Table 1]** and that a greater level of activity (duration and frequency) may be needed to offset the time spent in prolonged sitting.

Mechanistic insight

Sitting without interruption

Several mechanisms could have contributed to the observed increase in cf-PWV (**Figure 3, 5**). With acute prolonged sitting, a merger of contorted arteries (hip- and knee-joint) [43], elevated

gravitational pressure, and lack of skeletal muscle pump activity [44], increases lower extremity venous blood pooling [45], which limit venous return and stroke volume [45,46]. A decreased stroke volume reduces shear stress in the arterial tree – promoting oxidative stress and endothelial dysfunction – consequently, augmenting acute AS [11,16,28,32]. Another mechanism may be a subsequent drop in the renal perfusion pressure that activates RAAS, thus increasing both BP and AS [16]. Another plausible mechanism is the baroreceptor-mediated increase in systemic vascular resistance that stiffens the arterial segments [47]. Importantly, the speculated mechanism of prolonged-sitting-induced microvascular blunting in calf muscles, as assessed by near-infrared spectroscopy (NIRS), is recently challenged by an artificial intelligence (AI) based study [48], positing, it is the calf muscle-metabolic-rate (MMR) that becomes attenuated in response to lower oxygen demand (during prolonged uninterrupted sitting) rather than the proxy measure of microvascular oxygenation [48]. Together, these insights highlight that vascular dysfunction associated with acute sitting occurs not only in larger arteries, but advances across the local microvasculature, warranting future work to explore these mechanisms in chronic settings.

Sitting with interruption

As summarized in a recent review by Stoner et al. [11], two physiological mechanisms may help to explain the protective effect that sitting interruption strategies confer on AS: (1) *Direct hemodynamic effects:* Physical activities through the autonomic and hormonal-driven pathways increase blood flow to the exercising muscles [49]. An increased blood flow [50], plus a reduction in vascular resistance [50], triggers the hyperaemia-induced arterial sheer stress response [51], presumably via stimulation of endothelial mechanoreceptors [52]. This eventually leads to an exacerbated NO release, a molecule considered key in regulating arterial modelling, endothelial function and health [53]. (2) *Constrained energy theory:* Recent work suggests that sitting interruption strategies increase activity energy expenditure [54], to which the body responds by conserving energy dissipation made by other bodily processes [54,55]. This constrained energy expenditure presumably blocks proton leak from the mitochondria [55,56], thus limiting free radical generation, which subsequently reduces oxidative stress [56] and thus confers a protective effect on the extracellular matrix-related AS [57].

Methodological considerations

To translate meaningful inferences from the data included in this meta-analysis, it is worth noting a few methodological considerations. Foremost is the participant posture during which the PWV assessment is conducted. Ideally, researchers wish to perform this in a seated position being the posture of interest; however, only 2/10 studies did PWV assessment in seated position [16, 30], the rest used the standard PWV measurement guidelines and performed PWV assessment in supine

position. Thus, moving from seated to supine may result in masking some of the effect of sitting on PWV itself, given the muscle pump is an important stimulus of increasing shear stress through blood flow. Further, the varying 5–20 minutes post-seated posture transition (seated-to-supine) duration is not without consequences. Given that the purported stimulus for sitting-induced cardiovascular dysfunction is blood pooling in the lower limbs, more extended post-stimulus periods of 20 minutes may mask the actual effect of prolonged sitting as blood pooling dissipates, whilst shorter post-stimulus periods of 5 minutes may capture the hemodynamic impact of a posture transition with greater validity. Future work is needed to address these key methodological inconsistencies.

Strengths and limitations

To our knowledge, this is the first known study to assess the pooled effect of prolonged sitting (with and without interruption) on AS assessed using PWV. Whilst our meta-analytic model is robust, without any heterogeneity or outliers, and we included studies which collected PWV in a supine posture (pre- and post-sitting), we refrain from drawing definitive conclusions owing to the limited available data. Whilst 50% (n=5) of the included 10-trials performed statistical correction for mean arterial pressure/blood pressure (BP) and heart rate (HR), this might not fully account for individual differences in PWV-pressure relationships because both HR and BP are known to confound PWV due to their interspecific interaction and intrinsic dependence on AS [33]. We extracted PWV data from published studies (excluding registers and other sources of grey literature), but there is limited data reporting PWV as an outcome measure, which in turn constrains our ability to draw firm conclusions. However, this is an important first study in determining the potential effect of prolonged sitting on PWV and will likely be influential for the field moving forwards. Our representative data were exclusively from the US [16,18,27,28,30,32,33], UK [29], Japan [19,31], and Kingdom of Saudi Arabia (KSA) [7], chiefly comprising of young, healthy, and white populations, which means it was more homogenous (in terms of risk factors, and societal demographics) and thus more likely captures the true effects of prolonged sitting on PWV across these populations. However, future research should focus on other populations and geographical locations. Further, our acute findings cannot be extrapolated to long-term effects of prolonged sitting, and we recommend that future research should consider replicating these findings in longitudinal settings. We have limited our meta-analytic data to healthy subjects who do not have any known chronic illnesses to avoid potential confounding effects that non-communicable diseases (e.g., hypertension, diabetes mellitus) might have on AS independent of prolonged sitting. Individuals with high systolic/diastolic- and controlled/uncontrolled- hypertension and hyperglycaemia tend to show elevated AS and BP responses to sitting. If used, these participants may have skewed our estimated AS response during prolonged sitting. Furthermore, despite equal distribution of men and women in our chosen sample,

the paucity of a disaggregated sex-stratified data refrained us from performing sub-group analyses. This study is the first to show that breaking up prolonged sitting marginally reduces the increased cf-PWV response seen during uninterrupted sitting. Future work needs to be done to explore the effects of prolonged sitting in patient populations, as well as exploring the efficacy of a variety of different strategies such as aerobic, body weight and calisthenics in all populations.

Conclusion

To date, there has been no consolidation of data to show the impact of an acute bout of prolonged uninterrupted sitting on AS, and whether regular interruption strategies may moderate any negative effect. We found that acute bouts of prolonged sitting resulted in a significant increase in cf-PWV, and that breaking-up sitting conferred a lesser yet significant increase in cf-PWV; however, our findings cannot be extrapolated to long-term effects of prolonged sitting. Our conclusions are strengthened by our robust meta-analytic model, adherence to standardized PWV assessment guidelines, and prominent female representation (~50%). We recommend future research aims to: 1) inflate sample sizes; 2) recruit older adults, and individuals with pre-existing cardiometabolic risk factors; 3) longitudinally assess the effects of prolonged sitting on PWV, and 4) determine the efficacy of different interruption strategies to improve vascular function.

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Data availability: The data contained within will be made available upon reasonable request.

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