Pulse

Pulse 2015;3:134–140
DOI: 10.1159/000435792
Published online: July 18, 2015

2235–8676/15/0032–0134$39.50/0
www.karger.com/pls

Mini-Review

Weight Loss, Dietary Intake and Pulse Wave Velocity

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Key Words
Pulse wave velocity · Arterial stiffness · Weight loss · Diet

Abstract
We have recently conducted a meta-analysis to determine the effect of weight loss achieved by an energy-restricted diet with or without exercise, anti-obesity drugs or bariatric surgery on pulse wave velocity (PWV) measured at all arterial segments. Twenty studies, including 1,259 participants, showed that modest weight loss (8% of the initial body weight) caused a reduction in PWV measured at all arterial segments. However, due to the poor methodological design of the included studies, the results of this meta-analysis can only be regarded as hypothesis generating and highlight the need for further research in this area. In the future, well-designed randomised controlled trials are required to determine the effect of diet-induced weight loss on PWV and the mechanisms involved. In addition, there is observational evidence that dietary components such as fruit, vegetables, dairy foods, sodium, potassium and fatty acids may be associated with PWV, although evidence from well-designed intervention trials is lacking. In the future, the effect of concurrently improving dietary quality and achieving weight loss should be assessed in randomised controlled trials.

Introduction

Poor diet, including a high intake of energy-dense, nutrient-poor foods, is a leading cause of ill health in Australia and around the world [1]. Worldwide, the prevalence of obesity has more than doubled since 1980 [2]. Obesity increases the risk of cardiovascular disease (CVD) by approximately two fold [3]. At present, the effect of weight loss on cardiovascular endpoints is not clear [4], and due to the methodological difficulties associated with conducting these types of studies, a large body of research has looked at measures of vascular function that

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predict cardiovascular outcomes. We recently conducted a meta-analysis to determine the effect of weight loss achieved with an energy-restricted diet with or without exercise, anti-obesity drugs or bariatric surgery on pulse wave velocity (PWV) measured at all arterial segments [5].

Carotid femoral PWV (cfPWV) is considered the gold standard for determining arterial stiffness because the propagation of the forward pressure is measured at the aorta [6]. It is an independent predictor of cardiovascular events and mortality, and addition of cfPWV to the conventional Framingham risk factors improves CVD risk prediction [7]. A systematic review of observational studies found that age and blood pressure [systolic blood pressure: n = 43 (43%) or mean arterial pressure: n = 30 (31%)] were independent predictors of cfPWV in 91 and 90% of all studies, respectively [8]. The systematic review conducted by Cecelja and Chowienczyk [8] concluded that other than age and blood pressure, cfPWV is largely independent of traditional risk factors for CVD. Therefore, it is possible that dietary factors, including diet-induced weight loss and overall dietary intake, could predict PWV.

Weight Loss and PWV

The meta-analysis of 20 studies, including data from 1,259 participants, showed that modest weight loss (8% of the initial body weight) caused a reduction in PWV measured at all arterial segments [5] [fig. 1]. Subgroup analysis showed that there was not a statistically significant difference in the change in PWV achieved with an energy-restricted diet alone (10 studies) or an energy-restricted diet plus exercise (8 studies). Meta-regression showed that the change in PWV achieved with weight loss was positively correlated with the change in systolic and diastolic blood pressure. However, from the research that has been conducted it is unclear whether the blood pressure reduction precedes PWV change or is a result of a lowered PWV. Another possible mechanism to explain the effect of weight loss on PWV is a reduction in inflammation. Current evidence suggests that arterial stiffness is largely driven by inflammatory processes that impair endothelial nitric oxide production, generate reactive oxygen species and the release of neutrophils, macrophages and cytokines [9]. It is known that obesity is associated with an increase in inflammatory cytokines [10], and weight loss reduces inflammation [11]. A limitation of the meta-analysis conducted is that we did not extract data on inflammation molecules from the included trials.

This meta-analysis suggests that weight loss may be an effective strategy to reduce arterial stiffening. However, the methodological quality of the studies included was poor, and therefore, well-designed randomised controlled trials are required to determine the effect of weight loss on PWV. In particular, the macro-nutrient composition of the diet needs to be investigated further, as the studies included in this meta-analysis were too heterogeneous with respect to the diet to determine the effect of dietary composition on PWV. Furthermore, future research should aim to determine the mechanism by which weight loss has an effect. The results of this meta-analysis may not be generalizable to certain population groups. For example, only 3 studies (all uncontrolled) included in this meta-analysis were conducted in cohorts with diabetes (all type 2) or impaired fasting glucose. Two of the 3 studies did not achieve a statistically significant reduction in cfPWV with weight loss, although subgroup analysis showed that there was no statistically significant difference in the change in PWV by the diabetes status of the subjects. However, given the small number of studies and the low methodological quality, further research is required to determine whether weight loss is an effective strategy to reduce arterial stiffness in patients with diabetes. This is an important question because subjects with type 2 diabetes have a heightened risk of CVD compared with the general population [12].

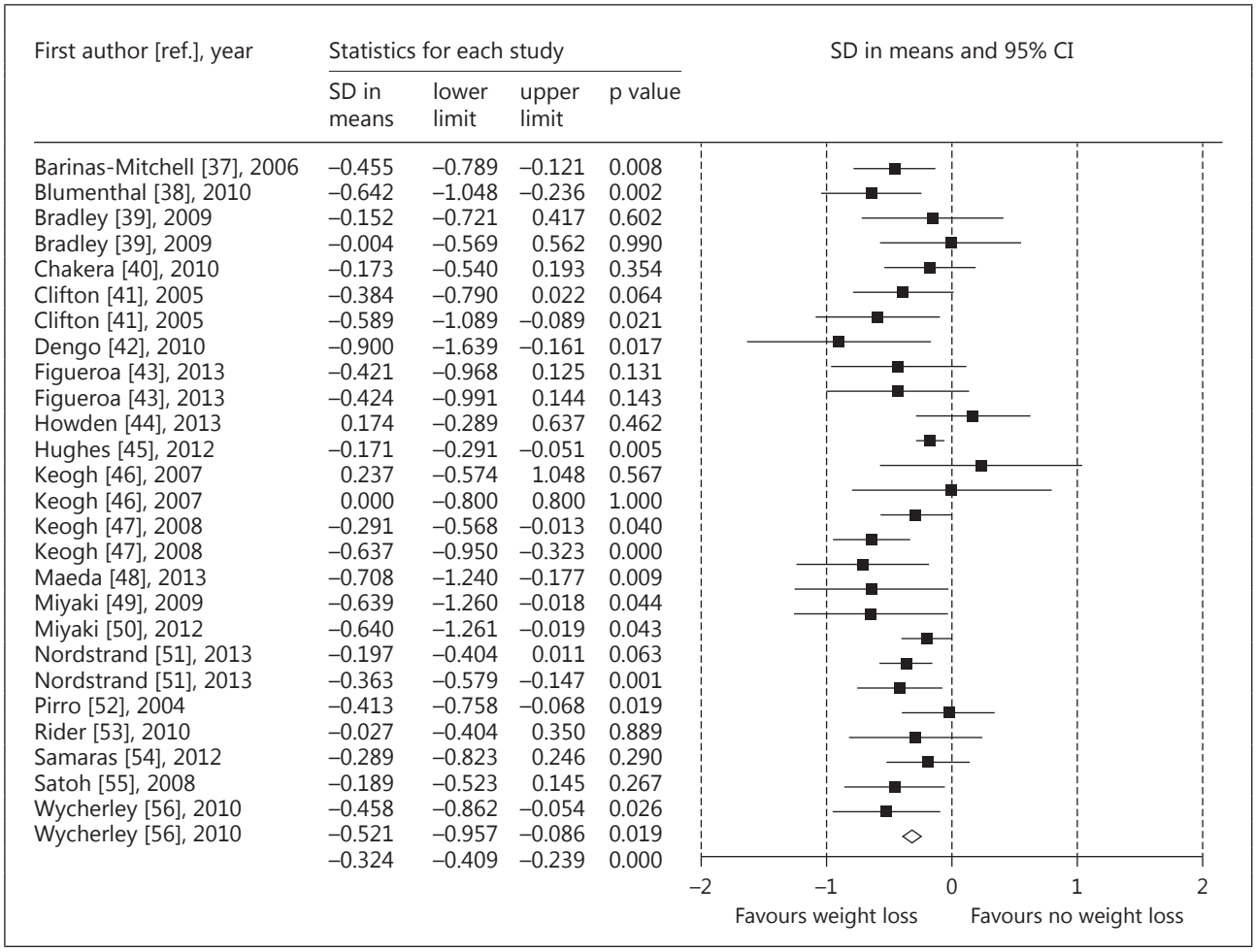

Fig. 1. The effect of weight loss on PWV measured at all arterial segments (adapted from Petersen et al. [5]).

Dietary Intake and PWV

The results of this meta-analysis suggest that weight loss may help improve vascular function, although improving dietary quality may also be another strategy to improve PWV. In 2011, Pase et al. [13] conducted a systematic review and meta-analysis of randomised controlled trials looking at the effect of dietary and nutrient interventions to improve measures of arterial stiffness (including PWV). It was found that omega 3 and soy isoflavone supplementation improved PWV, and there was limited evidence for a beneficial effect of vitamin and micro-nutrient supplementation. Epidemiological studies conducted in cohorts of healthy, hypertensive or high-risk CVD subjects suggest that the consumption of fruit and vegetables [14, 15], dairy foods [15–17] and flavonoids including soy [18] may be inversely associated with PWV, while the consumption of saturated fat [19] has a detrimental effect. Observational data for the effect of sodium on PWV is mixed. Lee et al. [20] showed an inverse association between predicted 24-hour sodium excretion (Tanaka's equation) and PWV, and García-Ortiz et al. [21] reported a non-significant J-shaped relationship between 24-hour sodium excretion and PWV. Dietary patterns such as the DASH diet [22] and the Mediterranean diet [23] seem to improve PWV; however, nutrient-poor, energy-dense patterns of

intake have a detrimental effect [24]. Intervention studies to support these findings are lacking or inconsistent.

In a randomised controlled trial of subjects at high risk of CVD with a habitually low intake of fruit and vegetables, the consumption of +2, +4 and +6 portions of high-flavonoid fruit and vegetables or low-flavonoid fruit and vegetables per day for 6 weeks (above habitual intake) did not affect cfPWV after 18 weeks once the data was adjusted for heart rate [25]. In the study by Macready et al. [25], the number of serves of fruit per day derived from fruit juice was not reported; however, the participants were allowed to include fruit juice, and this may help explain their finding, as it has been shown that fruit juice consumption is positively associated with arterial stiffness because of the high sugar content and lack of fibre [26]. Therefore, further research is required from well-designed randomised controlled trials to determine the effect of fruit and vegetable consumption on PWV and the mechanisms responsible. There is evidence to indicate that polyphenols and nitrates contained in fruit and vegetables may contribute to the beneficial effect on PWV [27]. A high potassium intake reduces PWV in subjects with hypertension in a dose-dependent manner, which may also explain the effect of fruit and vegetables [28].

Intervention studies looking at the effect of sodium on PWV have shown that in normotensive populations, sodium supplementation/a high-sodium diet for 2–6 weeks has no effect on PWV [29–31]. In hypertensive populations, a high-sodium diet (>90 mmol/24 h) compared to a lower sodium intake increases PWV [32–34]. Pimenta et al. [35] observed no reduction in PWV when the sodium intake was reduced to 50 mmol/24 h compared to 250 mmol/24 h for 7 days in a cohort of subjects with resistant hypertension, but blood pressure fell significantly. In summary, in subjects with hypertension, either unmedicated or responsive to antihypertensive therapy, sodium intake is positively associated with arterial stiffness. In normotensive populations, an increase in the sodium intake for up to 6 weeks does not seem to change PWV; however, it is not clear what the long-term effect is.

Epidemiological studies have indicated that a higher consumption of dairy products is associated with less arterial stiffening [15–17]. In the Maine-Syracuse Longitudinal Study, the frequency of the overall dairy consumption was inversely associated with cfPWV [16]. In a study of healthy Spanish subjects, Recio-Rodriguez et al. [17] found that only the consumption of low-fat dairy was associated with a reduced cfPWV, such that per 100 g/day increase in low-fat dairy, cfPWV was 0.101 m/s lower (95% CI –0.178, –0.023; p = 0.011). However, consuming whole-fat dairy was associated with a 0.109-m/s increase (95% CI 0.006, 0.213) in cfPWV per 100 g/day [17]. There have been limited randomised controlled trials conducted in this area. A recent randomised, cross-over study showed that an additional 4 servings of non-fat dairy per day reduced cfPWV compared with the no-dairy condition (4 servings of fruit juice) [36]. As previously mentioned, fruit juice has been shown to have a detrimental effect on arterial stiffness, and therefore, the result of this study may be a reflection of this rather than the effect of dairy. In summary, data from observational and intervention studies are not consistent with regard to the effect of dietary intake on PWV, and this is largely due to the heterogeneity between the studies and the lack of well-designed randomised controlled trials.

Conclusion

The most promising dietary strategy for improving PWV seems to be weight loss. However, concomitantly improving the dietary quality and achieving weight loss may improve vascular function to a greater extent. In the future, well-designed randomised controlled trials are required to determine the optimal macro-nutrient composition of weight loss diets

to reduce PWV, and the mechanisms that explain the effect of weight loss on PWV need to be explored. In addition, greater evidence is required for the effect of individual dietary components and patterns of intake on PWV.

Disclosure Statement

The authors have no disclosures.

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