

Brief Report

Relationship between Carotid-Femoral Pulse Wave Velocity and Diet-Induced Weight Loss

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Abstract: The global epidemic of obesity has increased over the past two decades, which has been attributed to a lack of physical activity and increased energy intake. Given the fact that obesity increases the risk of cardiovascular diseases, nutritional approaches to prevent cardiovascular diseases often target weight loss. Although many strategies are used to induce weight loss, the most common ones involve either total energy restriction, carbohydrate restriction, or dietary fat restriction. This report addresses the impact of each diet on improving carotid-femoral pulse wave velocity, a technique that is used to measure arterial stiffness—a surrogate marker of cardiovascular disease risk. Fourteen studies were included in the analysis and revealed that diet-induced weight-loss plateaus with increasing duration of the intervention ($p = 0.174$, $r = 0.455$). Weight loss was significantly associated with a reduction in cf-PWV ($p = 0.012$, $r = 0.591$). Further, when macronutrient composition was considered, weight loss was achieved through a low-carbohydrate diet ($p = 0.097$, $r = 0.626$), not total energy restriction ($p = 0.660$, $r = 0.204$) or low-fat diet ($p = 0.142$, $r = 0.975$), tended to reduce cf-PWV. These data suggest that weight loss achieved through a low-carbohydrate diet may have a greater impact on PWV over total energy restriction. More studies are needed to test the effect of weight loss achieved through a low-fat diet on cf-PWV.

Keywords: arterial stiffness; cf-PWV; low-carbohydrate diet; low-fat diet; weight loss



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1. Introduction

The global epidemic of obesity has increased over the past two decades [1], which has been attributed to poor lifestyle, i.e., lack of physical activity and increased energy intake [2]. Epidemiological data suggest that the prevalence of obesity would continue to increase in Australia, Canada, European countries, the United Kingdom, and the United States [3–8]. Importantly, obesity and cardiovascular diseases, which were the leading cause of death in Canada and other parts of the world [9,10], often occur concurrently. Indeed, Hingorani et al. suggest that obesity is the main cause of developing cardiovascular diseases [2]. The potential mechanism involves the increased flux of fatty acids and dyslipidemia, which eventually results in the accumulation of lipids in arterial walls, leading to atherosclerosis [11]. Identifying atherosclerosis at an early stage has been a beneficial strategy to prevent the risk of cardiovascular disease-related mortality.

The diagnosis of atherosclerosis at early stages has been challenging. Measuring arterial stiffness appears to be a promising tool to identify atherosclerosis noninvasively during early stages. Although the direct mechanism through which atherosclerosis influences arterial stiffness is unknown, several studies have shown a strong relationship between arterial stiffness and atherosclerosis and, therefore, arterial stiffness has been used as a surrogate marker for atherosclerosis [12–14]. Arterial stiffness can be measured using a technique called pulse wave velocity (PWV). PWV can be used to measure peripheral vascular stiffness, using brachial-ankle (ba-PWV), carotid-radial (cr-PWV), femoral-ankle (fa-PWV), and carotid-ankle vascular index (CAVI); and central vascular stiffness, using carotid-femoral (cf-PWV). Although several factors such as blood pressure and body weight

have been shown to affect cf-PWV in children [15], cf-PWV is still considered as a gold-standard measurement of arterial stiffness and has shown to predict mortality in end-stage renal disease patients [16,17].

The primary cause of the obesity-induced increased risk of atherosclerosis-related mortality appears to be excess energy intake, which is why the prevention strategy includes energy restriction. Several dietary interventions have been considered [18], and they primarily focus on either reducing total energy intake or reducing the intake of a particular macronutrient. Reducing total nutrient intake or dietary fats has been a classical approach; however, reducing carbohydrate intake has been relatively new due to the number of metabolic benefits, i.e., reduction in lipids such as triglycerides (TG), total cholesterol (TC), and low-density lipoproteins (LDL), and cardiovascular benefits (arterial stiffness) observed with low or very low-carbohydrate diets [19–22]. However, limited data have been published to differentiate between the effects of each macronutrient restriction (low-fat or low-carbohydrate diet). Indeed, regardless of the type of macronutrient restricted, all diets appear to induce weight loss effectively [19]. However, whether similar effects are observed with weight loss on cf-PWV or restriction of specific macronutrient-induced weight loss can have differential effects on cf-PWV is unknown. Therefore, the purpose of this brief report was to demonstrate the changes in cf-PWV in response to diet-induced weight loss. Furthermore, the secondary purpose of this report was to identify whether weight loss-induced changes in cf-PWV were related to any particular macronutrient restriction.

2. Methods

As discussed above, arterial stiffness measured using cf-PWV has been used as a gold-standard technique and has shown to be the strongest predictor of CVD-related mortality [16,17]; only studies that measured arterial or vascular stiffness using cf-PWV published until the year 2021 were included in this brief analysis. Studies utilizing exercise, drugs, or surgical intervention (alone or in combination with dietary intervention) were excluded from the analysis. Review articles were screened to confirm that all the studies were included in the analysis. Studies were searched using the following term “(“weight loss” [Title/Abstract] AND (pwv[Title/Abstract] OR “arterial stiffness” [Title/Abstract] OR “aortic stiffness” [Title/Abstract] OR “vascular stiffness” [Title/Abstract]))” in PubMed/NLM. A regression analysis was performed using IBM SPSS (v. 27, Armonk, NY, USA, IBM Corp.) to test the relationship between percent weight loss and study duration using study duration as the independent variable (x -axis) and weight loss as the dependent variable (y -axis). For the relationship between percent weight loss and change in cf-PWV, the percent change in weight loss as an independent variable (reported on the y -axis) and change in cf-PWV as a dependent variable (x -axis).

3. Results

As shown in Figure 1, 112 studies were identified using the search and, of those, fourteen studies (five studies had two different dietary interventions and were reported separately) were selected for the analysis based on the criteria mentioned above. Information related to studies included in this report is presented in Table 1. Of the fourteen studies, one study was conducted on men (two different arms), three studies were conducted only on women, and eleven studies were conducted on both sexes. All the studies included middle-aged participants with an average age above 35 years who were overweight or obese. Two studies investigated low-fat diet intervention (one study had two arms) [23,24], four studies conducted low-carbohydrate diet intervention [24–27], four studies were very low-carbohydrate diet intervention [20,24,27,28], one study had two arms (one with low glycemic and one with high glycemic index diet) [29], and the remaining six studies had total energy restriction [30–35]. Study duration ranged from as short as two weeks to as long as 52 weeks. Given the limited number of studies available in the literature to identify differences between macronutrient-related effects on cf-PWV, average weight loss

from each intervention was recorded in kilograms (kg) and percent weight loss either was reported directly from the study itself or was estimated via calculations. Similarly, change in cf-PWV was recorded from each publication (not shown in table).

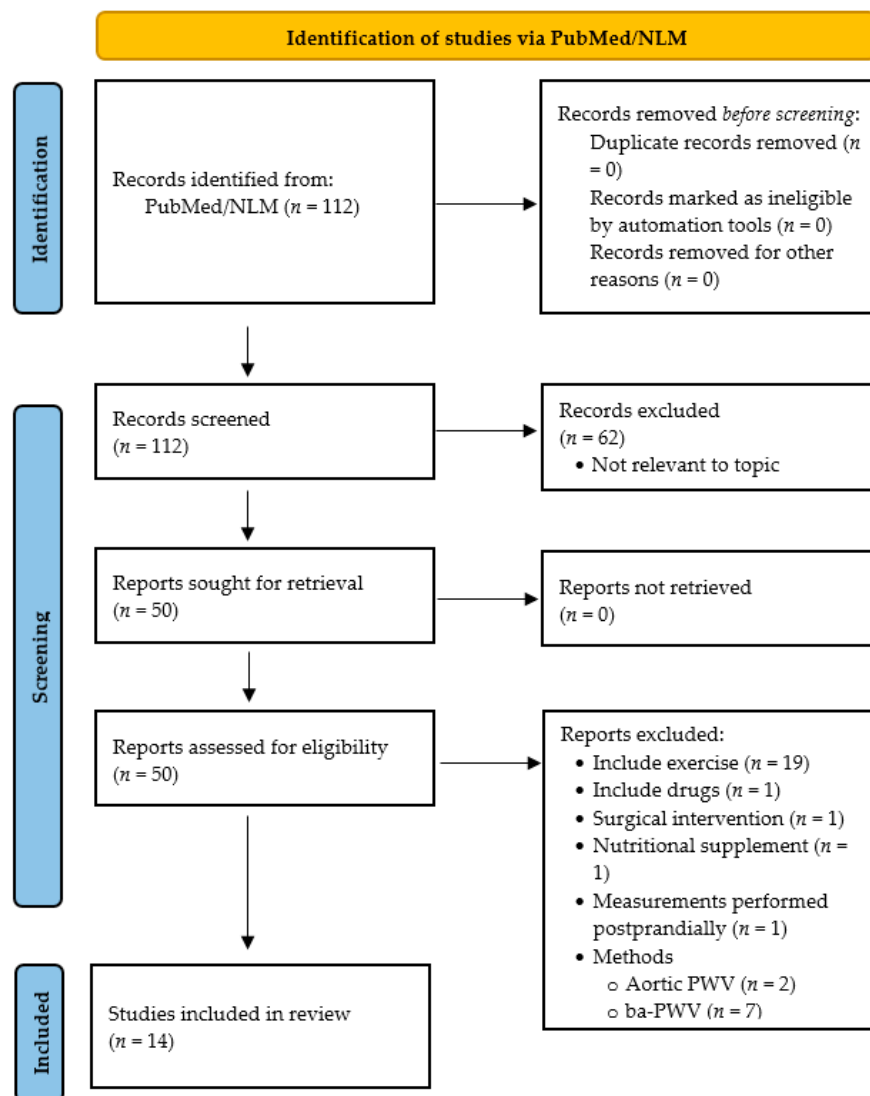


Figure 1. PRISMA 2020 flow diagram for studies included in this report.

Table 1. Details pertaining to the studies included in this report.

Study	N	Age	Sex	Study Population	Intervention	Duration	WL (kg)	WL (%)	Change in cf-PWV (cm/s)
Barinas-Mitchell et al. [30]	21	50 ± 2	Both	T2DM + Ob	ER ≤30 g fat ~500 kcal/d	52	-9.4	-9.2	-50
Clifton et al. [23]	26	49 ± 9	Both	OW	ER (LF-MR) 6026 kJ/d 1800 kJ in SlimFast replaced breakfast and lunch 3500 kJ/d of fruits and vegetables [57.2% carbs, 22.7% fat, 20.1% proteins]	12	-6.0	-6.4	-124

Table 1. Cont.

Study	N	Age	Sex	Study Population	Intervention	Duration	WL (kg)	WL (%)	Change in cf-PWV (cm/s)
Clifton et al. [23]	29	47 ± 10	Both	OW	ER (LF) 6047 kJ/d [61.7% carbs, 16.8% fat, 21.5% proteins]	12	−6.6	−6.9	−102
Dengo et al. [31]	25	61 ± 1	Both	OW + Ob	ER 1200–1500 kcals [50 ± 3% carbs, 32 ± 2% fats, 18 ± 1% proteins]	12	−7.1	−8.4	−187
Figueroa et al. [32]	13	54 ± 4	Female	OW + Ob	ER ~1250 kcal/d [55–60% carbs, 20–25% fats, 20–25% proteins]	12	−5.6	−6.3	−50
Heiston et al. [25]	12	46 ± 4	Female	Ob	ER (LC) ~1000–1200 kcal/d [reduced total energy intake by −918 ± 221 kcal/d]	2	−2.2	−2.1	−30
Joris et al. [28]	23	52 ± X	Both	Ob	ER (VLCD) ~1500 kcal/d 500 kcal/d liquid meal (50 g carbs, 7 g fats, 52 g proteins) + 250 g/d of fruits and vegetables	8	−10.3	−10.5	−50
Keogh et al. [26]	13	50 ± 1	Both	OW + Ob	ER (LC) 6000 kJ/d [33% carbs, 27% fats, 40% proteins]	52	−4.6	−5.0	0
Keogh et al. [26]	13	47 ± 2	Both	OW + Ob	ER (LF) 6000 kJ/d [60% carbs, 20% fats, 20% proteins]	52	−5.5	−6.0	90
Keogh et al. [24]	52	51 ± 8	Both	OW + Ob	ER (VLCD) 6000–7000 kJ/d [4% carbs, 61% fats, 35% proteins]	8	−7.5	−8.0	−80
Keogh et al. [24]	47	49 ± 8	Both	OW + Ob	ER (LC) 6000–7000 kJ/d [46% carbs, 30% fats, 24% proteins]	8	−6.2	−6.4	−160
Miyaki et al. [33]	12	45 ± 7	Female	OW + Ob	ER 1680 kcal/d Meals containing 840 g carbs, 420 g fats, 420 g proteins [50% carbs, 25% fats, 25% proteins]	12	−8.0	−9.1	60
Nordstrand et al. [34]	91	42 ± 10	Both	M-Ob	ER ~900 kcal/d [43% carbs, 20% fats, 37% proteins]	28	−9.4	−6.8	−20
Philippou et al. [29]	22	NR	Male	Unknown	ER (Low GI) Reduced total energy intake by −447 ± 499 kcal/d [Carbs consumed 224 ± 50 g/d]	24	−2.2	NR	−40
Philippou et al. [29]	16	NR	Male	Unknown	ER (High GI) Reduced total energy intake by −236 ± 632 kcal/d [Carbs consumed 278 ± 7 g/d]	24	−3.0	NR	−30
Syed-Abdul et al. [20]	19	40 ± 8	Both	OW + Ob	ER (VLCD) 1500 kcal/d 20–25 g/d of carbs and 8 oz of non-caloric fluids [14% carbs, 58% fats, 28% proteins]	4	−5.7	−3.8	−48
Weiss et al. [35]	17	57 ± 7	Both	Ow	ER 1428 ± 85 kcal/d [47 ± 2% carbs, 33 ± 1% fats, 19 ± 1% proteins]	12–14 *	−5.4	−6.8	−10
Wycherley et al. [27]	26	50 ± 9	Both	OW + Ob	ER (VLCD) 6–7 MJ/d [4% carbs, 61% fats, 35% proteins]	52	−14.9	−16.0	−14
Wycherley et al. [27]	23	50 ± 7	Both	OW + Ob	ER (LC) 6–7 MJ/d [46% carbs, 30% fats, 24% proteins]	52	−11.5	−12.0	−15

Data are presented in mean ± SD for age. Body weight reduced after weight loss (WL) is reported in kg. X refers to value not reported. Abbreviations: ER: energy restriction, GI: glycemic index, LC: low-carbohydrate diet, LF: low-fat diet, M-Ob: morbidly obese, MR: meal replacement, NR: no report, Ob: obese, OW: overweight, T2DM: Type 2 diabetes mellitus, and VLCD: very low-carbohydrate diet. * Duration was extended if needed to achieve percent weight loss.

As expected, shown in Figure 2 is the weight loss over 52 weeks, a trend commonly observed, i.e., the weight loss is greatly achieved with shorter studies; whereas, when the intervention is long, weight loss plateaus ($p = 0.067$, $r = 0.454$). The average weight loss with energy-restricted diet was 7.8 kg (7.5%), low-carbohydrate diet was 8.0 kg (7.9%), and low-fat diet was 6.4 kg (6.0%) with no differences between them. The average reduction in cf-PWV with energy-restricted diet was 4.3 cm/s, low-carbohydrate diet was 8.3 cm/s, and low-fat diet was 4.5 cm/s with no significant differences between them. Regression analysis revealed no relationship between change in cf-PWV and change in percent weight loss ($p = 0.152$, $r = 0.363$) or absolute weight loss ($p = 0.140$, $r = 0.351$) for all studies combined (data not shown). Similar results were observed for studies that restricted total energy intake (percent weight loss: $p = 0.978$, $r = 0.015$, absolute weight loss: $p = 0.957$, $r = 0.023$), and low-fat diets (percent weight loss: $p = 0.434$, $r = 0.777$, absolute weight loss: $p = 0.426$, $r = 0.784$, data not shown). However, for studies that utilized carbohydrate-restricted diets, a trend was observed (percent weight loss: $p = 0.098$, $r = 0.625$, absolute weight loss: $p = 0.101$, $r = 0.620$, data not shown). However, the duration of these studies was highly variable; therefore, to control for the effect of study duration, both the percent weight loss and change in cf-PWV were divided by the number of weeks to obtain the percent change in body weight per week and change in cf-PWV per week. Interestingly, as shown in Figure 3, a significant relationship was observed between percent weight loss per week and change in cf-PWV (black line in Figure 3, $p = 0.015$, $r = 0.579$). When the regression analysis was performed for studies that utilized total energy restriction (orange line, $p = 0.683$, $r = 0.215$) or low-fat diet (yellow line, $p = 0.143$, $r = 0.975$), no relationships were observed. However, when studies that included only low-carbohydrate diets were included, a trend was observed (grey line, $p = 0.099$, $r = 0.623$). Lack of significance was likely due to smaller sample size. Similar results were observed with absolute change in weight loss per week and change in cf-PWV per week (data not shown).

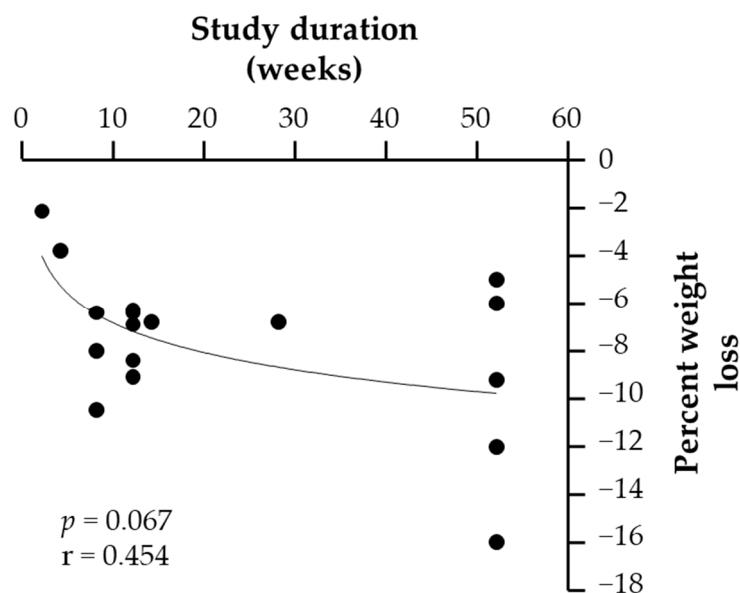


Figure 2. Percent weight loss achieved and duration of dietary intervention. Legend: Each data point represents each study included in the analysis. Two studies were excluded because they did not report percent weight loss / were not able to make calculations because of missing baseline body weight. A quadratic regression analysis was performed to test the relationship between study duration and percent weight loss achieved.

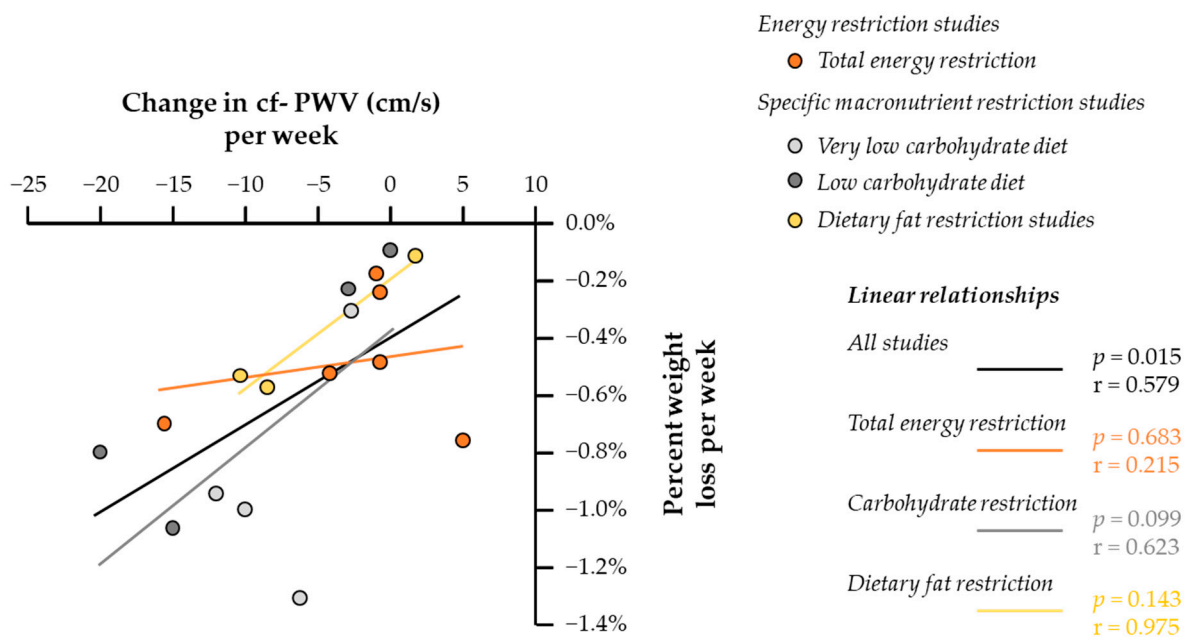


Figure 3. Relationship between diet-induced percent weight loss and arterial stiffness calculated per week. Legend: Each data point represents each study included in the analysis. Two studies were excluded because they did not report percent weight loss/were not able to make calculations because of missing baseline body weight. The change in cf-PWV was divided by the duration of the study to estimate the cf-PWV per week. Similar calculations were performed for weight loss. Blackline represents all studies included in the analysis. Orange line and dots represent studies with total energy restriction; grey line, and dots represent studies that restricted only carbohydrates, darker grey dots represent moderate restriction and lighter grey dots represent very low-carbohydrate diets. Yellow line and dots represent studies that restricted dietary fat. Abbreviations: cf-PWV, carotid-femoral pulse wave velocity.

4. Discussion

The primary purpose of this report was to identify studies and analyze the changes in cf-PWV and body weight in response to dietary restrictions. The secondary purpose was to investigate the relationship between change in weight loss and change in cf-PWV and whether this effect was related to particular macronutrient restrictions. The analysis from this report showed that the reduction in arterial stiffness is associated with diet-induced weight loss. Wildman et al. conducted a longitudinal study in 152 participants with an average of 30 years, and measured arterial PWV annually [36]. The goal was to observe changes in PWV with changes in body weight observed in the absence of dietary intervention. Interestingly, the increase in PWV was strongly associated with an increase in body weight; similarly, a decrease in body weight was associated with a decrease in PWV [36]. In the present report, similar findings were observed when weight loss was achieved through dietary intervention. These data suggest that reduction in arterial stiffness is primarily associated with weight loss alone. In contrast, in a study conducted by Desamericq et al., no relationship was observed between BMI and cf-PWV [37]. In this study, the authors measured cf-PWV in 2304 patients referred for ambulatory blood pressure monitoring. No differences were observed in cf-PWV with regard to body weight or BMI. However, Desamericq et al. did not consider sex differences that could play a significant role in cf-PWV. Indeed, Zuo et al. conducted a study on 834 participants and reported a relationship between cf-PWV and BMI in females only [38]. With an increment of 10 units of BMI, only females but not males showed a significant increase in PWV. However, in the present study, sex differences were not evaluated due to a lack of studies on both sexes.

Furthermore, the secondary purpose of this study was to identify whether it would make a difference to the cf-PWV if weight loss was achieved through different dietary interventions (i.e., total energy restriction, low-fat diet, or low-carbohydrate diet). Indeed, when total energy-restricted studies were included in the analysis, no differences were observed even though weight loss was achieved. In contrast, when low-carbohydrate diets were utilized to achieve weight loss, cf-PWV tended to relate to weight loss. The lack of significance was likely due to the smaller sample size. These findings suggest that low-carbohydrate diet-induced weight loss may have a greater impact on improving cf-PWV compared to weight loss achieved through total energy restriction. Given that low-carbohydrate diets can reduce systolic blood pressure [39]; systolic blood pressure has been previously associated with PWV [15]. The improvement in cf-PWV observed with a low-carbohydrate diet could be either a consequence of or the product of decreased cf-PWV. The other major mechanism through which the low-carbohydrate diets may affect cf-PWV is likely through an established insulin resistance pathway. Further, individuals with Type 2 diabetes (who are hyperglycemic and insulin resistant) have exhibited increased cf-PWV compared to non-diabetic individuals [40,41]. Insulin resistance is a common feature of obesity and has been shown to induce pathological vascular stiffening [42]. Indeed, in a study conducted by Grunewald et al. [43], mice fed with high sucrose and high fructose diets resulted in increased body weight. The diet-induced obesity resulted in the increased insulin resistance of the vasculature and worsening of arterial stiffness through endothelial dysfunction. In contrast, when carbohydrates were restricted in the diet of patients taking statins, insulin sensitivity was improved as well as vascular endothelial function (a marker of vascular stiffness) [44]. This is likely due to the nature of the low-carbohydrates diets that improve glycemic control and insulin sensitivity—factors that may eventually lead to improved arterial stiffness [45].

With regard to the low-fat diet, only two studies were found that met the inclusion criteria, and, therefore, it is difficult to speculate the effects of low-fat diet on cf-PWV. Findings from the report present new insights into weight loss-induced improvements in cf-PWV. Despite the fact that this report indicated a stronger relationship between low-carbohydrate diet-induced weight loss and cf-PWV compared to total energy-restricted diet-induced weight loss, more studies should be conducted to establish a definitive conclusion.

One of the limitations of this study was that only fourteen studies were included in the analysis due to stringent inclusion criteria. The goal was to identify if diet-induced weight loss changes cf-PWV, and if these changes are associated with particular macronutrient restriction. Therefore, studies including exercise, drugs, or in combination with diet were excluded. Lastly, despite other valid methods of measuring PWV are available, only studies that utilized cf-PWV were included in the analysis. The primary reason was that cf-PWV is considered a gold-standard technique over the other techniques and the secondary reason was to be able to compare studies without the effect of confounding factors.

In summary, the analysis from this report revealed a trend with diet-induced weight loss and the duration of the intervention; that was expected. The novel findings from this report presents a relationship between weight loss achieved through low-carbohydrate diets and cf-PWV which was not observed when weight loss was achieved through total energy intake. Mechanistic studies are needed to identify the mechanism through which dietary carbohydrate restriction improves arterial stiffness.

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References

1. Hruby, A.; Hu, F.B. The Epidemiology of Obesity: A Big Picture. *Pharmacoeconomics* **2015**, *33*, 673–689. [[CrossRef](#)] [[PubMed](#)]
2. Hingorani, A.D.; Finan, C.; Schmidt, A.F. Obesity causes cardiovascular diseases: Adding to the weight of evidence. *Eur. Heart J.* **2020**, *41*, 227–230. [[CrossRef](#)] [[PubMed](#)]
3. Pineda, E.; Sanchez-Romero, L.M.; Brown, M.; Jaccard, A.; Jewell, J.; Galea, G.; Webber, L.; Breda, J. Forecasting Future Trends in Obesity across Europe: The Value of Improving Surveillance. *Obes. Facts.* **2018**, *11*, 360–371. [[CrossRef](#)] [[PubMed](#)]
4. Webber, L.; Divajeva, D.; Marsh, T.; McPherson, K.; Brown, M.; Galea, G.; Breda, J. The future burden of obesity-related diseases in the 53 WHO European-Region countries and the impact of effective interventions: A modelling study. *BMJ Open* **2014**, *4*, e004787. [[CrossRef](#)]
5. Janssen, F.; Bardoutsos, A.; Vidra, N. Obesity Prevalence in the Long-Term Future in 18 European Countries and in the USA. *Obes. Facts.* **2020**, *13*, 514–527. [[CrossRef](#)]
6. Hayes, A.J.; Lung, T.W.; Bauman, A.; Howard, K. Modelling obesity trends in Australia: Unravelling the past and predicting the future. *Int. J. Obes.* **2017**, *41*, 178–185. [[CrossRef](#)]
7. Keaver, L.; Xu, B.; Jaccard, A.; Webber, L. Morbid obesity in the UK: A modelling projection study to 2035. *Scand. J. Public Health* **2020**, *48*, 422–427. [[CrossRef](#)]
8. O'Neill, M.; Kornas, K.; Rosella, L. The future burden of obesity in Canada: A modelling study. *Can. J. Public Health* **2019**, *110*, 768–778. [[CrossRef](#)]
9. Viorela, D.; Ouellette, N.; Camarda, C.G.; Bourbeau, R. Insight on 'typical' longevity: An analysis of the modal lifespan by leading causes of death in Canada. *Demogr. Res.* **2016**, *35*, 471–504.
10. Lippi, G.; Plebani, M. Biomarker research and leading causes of death worldwide: A rather feeble relationship. *Clin. Chem. Lab. Med.* **2013**, *51*, 1691–1693. [[CrossRef](#)]
11. Zhang, T.; Chen, J.; Tang, X.; Luo, Q.; Xu, D.; Yu, B. Interaction between adipocytes and high-density lipoprotein: new insights into the mechanism of obesity-induced dyslipidemia and atherosclerosis. *Lipids. Health Dis.* **2019**, *18*, 223. [[CrossRef](#)] [[PubMed](#)]
12. Glasser, S.P.; Arnett, D.K.; McVeigh, G.E.; Finkelstein, S.M.; Bank, A.J.; Morgan, D.J.; Cohn, J.N. Vascular compliance and cardiovascular disease: A risk factor or a marker? *Am. J. Hypertens.* **1997**, *10*, 1175–1189. [[CrossRef](#)]
13. Laurent, S.; Boutouyrie, P. Arterial stiffness: A new surrogate end point for cardiovascular disease? *J. Nephrol.* **2007**, *20* (Suppl. S12), S45–S50. [[PubMed](#)]
14. van Popele, N.M.; Grobbee, D.E.; Bots, M.L.; Asmar, R.; Topouchian, J.; Reneman, R.S.; Hoeks, A.P.; van der Kuip, D.A.; Hofman, A.; Witteman, J.C. Association between arterial stiffness and atherosclerosis: The Rotterdam Study. *Stroke* **2001**, *32*, 454–460. [[CrossRef](#)]
15. Genovesi, S.; Salvi, P.; Nava, E.; Tassistro, E.; Giussani, M.; Desimone, I.; Orlando, A.; Battaglino, M.; Lieti, G.; Montemerlo, M.; et al. Blood Pressure and Body Weight Have Different Effects on Pulse Wave Velocity and Cardiac Mass in Children. *J. Clin. Med.* **2020**, *9*, 2954. [[CrossRef](#)]
16. Van Bortel, L.M.; Laurent, S.; Boutouyrie, P.; Chowienzyk, P.; Cruickshank, J.K.; De Backer, T.; Filipovsky, J.; Huybrechts, S.; Mattace-Raso, F.U.; Protogerou, A.D.; et al. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity. *J. Hypertens.* **2012**, *30*, 445–448. [[CrossRef](#)]
17. Pannier, B.; Guerin, A.P.; Marchais, S.J.; Safar, M.E.; London, G.M. Stiffness of capacitive and conduit arteries: Prognostic significance for end-stage renal disease patients. *Hypertension* **2005**, *45*, 592–596. [[CrossRef](#)]
18. Lanier, J.B.; Bury, D.C.; Richardson, S.W. Diet and Physical Activity for Cardiovascular Disease Prevention. *Am. Fam. Physician.* **2016**, *93*, 919–924.
19. Sackner-Bernstein, J.; Kanter, D.; Kaul, S. Dietary Intervention for Overweight and Obese Adults: Comparison of Low-Carbohydrate and Low-Fat Diets. A Meta-Analysis. *PLoS ONE* **2015**, *10*, e0139817. [[CrossRef](#)]
20. Syed-Abdul, M.M.; Hu, Q.; Jacome-Sosa, M.; Padilla, J.; Manrique-Acevedo, C.; Heimowitz, C.; Parks, E.J. Effect of carbohydrate restriction-induced weight loss on aortic pulse wave velocity in overweight men and women. *Appl. Physiol. Nutr. Metab.* **2018**, *43*, 1247–1256. [[CrossRef](#)]
21. Gjuladin-Hellon, T.; Davies, I.G.; Penson, P.; Amiri Baghbadorani, R. Effects of carbohydrate-restricted diets on low-density lipoprotein cholesterol levels in overweight and obese adults: A systematic review and meta-analysis. *Nutr. Rev.* **2019**, *77*, 161–180. [[CrossRef](#)] [[PubMed](#)]
22. Ge, L.; Sadeghirad, B.; Ball, G.D.C.; da Costa, B.R.; Hitchcock, C.L.; Svendrovski, A.; Kiflen, R.; Quadri, K.; Kwon, H.Y.; Karamouzian, M.; et al. Comparison of dietary macronutrient patterns of 14 popular named dietary programmes for weight and cardiovascular risk factor reduction in adults: Systematic review and network meta-analysis of randomised trials. *BMJ* **2020**, *369*, m696. [[CrossRef](#)] [[PubMed](#)]
23. Clifton, P.M.; Keogh, J.B.; Foster, P.R.; Noakes, M. Effect of weight loss on inflammatory and endothelial markers and FMD using two low-fat diets. *Int. J. Obes.* **2005**, *29*, 1445–1451. [[CrossRef](#)] [[PubMed](#)]
24. Keogh, J.B.; Brinkworth, G.D.; Noakes, M.; Belobrajdic, D.P.; Buckley, J.D.; Clifton, P.M. Effects of weight loss from a very-low-carbohydrate diet on endothelial function and markers of cardiovascular disease risk in subjects with abdominal obesity. *Am. J. Clin. Nutr.* **2008**, *87*, 567–576. [[CrossRef](#)]
25. Heiston, E.M.; Gilbertson, N.M.; Eichner, N.Z.M.; Malin, S.K. A Low-Calorie Diet with or without Exercise Reduces Postprandial Aortic Waveform in Females with Obesity. *Med. Sci. Sports Exerc.* **2021**, *53*, 796–803. [[CrossRef](#)]

26. Keogh, J.B.; Brinkworth, G.D.; Clifton, P.M. Effects of weight loss on a low-carbohydrate diet on flow-mediated dilatation, adhesion molecules and adiponectin. *Br. J. Nutr.* **2007**, *98*, 852–859. [[CrossRef](#)]
27. Wycherley, T.P.; Brinkworth, G.D.; Keogh, J.B.; Noakes, M.; Buckley, J.D.; Clifton, P.M. Long-term effects of weight loss with a very low carbohydrate and low fat diet on vascular function in overweight and obese patients. *J. Intern. Med.* **2010**, *267*, 452–461. [[CrossRef](#)]
28. Joris, P.J.; Plat, J.; Kusters, Y.H.; Houben, A.J.; Stehouwer, C.D.; Schalkwijk, C.G.; Mensink, R.P. Diet-induced weight loss improves not only cardiometabolic risk markers but also markers of vascular function: A randomized controlled trial in abdominally obese men. *Am. J. Clin. Nutr.* **2017**, *105*, 23–31. [[CrossRef](#)]
29. Philippou, E.; Bovill-Taylor, C.; Rajkumar, C.; Vampa, M.L.; Ntatsaki, E.; Brynes, A.E.; Hickson, M.; Frost, G.S. Preliminary report: The effect of a 6-month dietary glycemic index manipulation in addition to healthy eating advice and weight loss on arterial compliance and 24-hour ambulatory blood pressure in men: A pilot study. *Metabolism* **2009**, *58*, 1703–1708. [[CrossRef](#)]
30. Barinas-Mitchell, E.; Kuller, L.H.; Sutton-Tyrrell, K.; Hegazi, R.; Harper, P.; Mancino, J.; Kelley, D.E. Effect of weight loss and nutritional intervention on arterial stiffness in type 2 diabetes. *Diabetes Care* **2006**, *29*, 2218–2222. [[CrossRef](#)]
31. Dengo, A.L.; Dennis, E.A.; Orr, J.S.; Marinik, E.L.; Ehrlich, E.; Davy, B.M.; Davy, K.P. Arterial destiffening with weight loss in overweight and obese middle-aged and older adults. *Hypertension* **2010**, *55*, 855–861. [[CrossRef](#)] [[PubMed](#)]
32. Figueroa, A.; Vicil, F.; Sanchez-Gonzalez, M.A.; Wong, A.; Ormsbee, M.J.; Hooshmand, S.; Daggy, B. Effects of diet and/or low-intensity resistance exercise training on arterial stiffness, adiposity, and lean mass in obese postmenopausal women. *Am. J. Hypertens.* **2013**, *26*, 416–423. [[CrossRef](#)] [[PubMed](#)]
33. Miyaki, A.; Maeda, S.; Yoshizawa, M.; Misono, M.; Saito, Y.; Sasai, H.; Endo, T.; Nakata, Y.; Tanaka, K.; Ajisaka, R. Effect of weight reduction with dietary intervention on arterial distensibility and endothelial function in obese men. *Angiology* **2009**, *60*, 351–357. [[CrossRef](#)] [[PubMed](#)]
34. Nordstrand, N.; Gjevestad, E.; Hertel, J.K.; Johnson, L.K.; Saltvedt, E.; Roislien, J.; Hjelmessaeth, J. Arterial stiffness, lifestyle intervention and a low-calorie diet in morbidly obese patients—a nonrandomized clinical trial. *Obesity* **2013**, *21*, 690–697. [[CrossRef](#)]
35. Weiss, E.P.; Albert, S.G.; Reeds, D.N.; Kress, K.S.; McDaniel, J.L.; Klein, S.; Villareal, D.T. Effects of matched weight loss from calorie restriction, exercise, or both on cardiovascular disease risk factors: A randomized intervention trial. *Am. J. Clin. Nutr.* **2016**, *104*, 576–586. [[CrossRef](#)]
36. Wildman, R.P.; Farhat, G.N.; Patel, A.S.; Mackey, R.H.; Brockwell, S.; Thompson, T.; Sutton-Tyrrell, K. Weight change is associated with change in arterial stiffness among healthy young adults. *Hypertension* **2005**, *45*, 187–192. [[CrossRef](#)]
37. Desamericq, G.; Tissot, C.M.; Akakpo, S.; Tropeano, A.I.; Millasseau, S.; Macquin-Mavier, I. Carotid-femoral pulse wave velocity is not increased in obesity. *Am. J. Hypertens.* **2015**, *28*, 546–551. [[CrossRef](#)]
38. Zuo, J.; Chao, H.; Tang, B.; Avolio, A.P.; Schlaich, M.P.; Nolde, J.M.; Adji, A.; Carnagarin, R. Female Gender Is Associated with Higher Susceptibility of Weight Induced Arterial Stiffening and Rise in Blood Pressure. *J. Clin. Med.* **2021**, *10*, 3479. [[CrossRef](#)]
39. Santos, F.L.; Esteves, S.S.; da Costa Pereira, A.; Yancy, W.S., Jr.; Nunes, J.P. Systematic review and meta-analysis of clinical trials of the effects of low carbohydrate diets on cardiovascular risk factors. *Obes. Rev.* **2012**, *13*, 1048–1066. [[CrossRef](#)]
40. Theofilis, P.; Oikonomou, E.; Lazaros, G.; Vogiatzi, G.; Anastasiou, M.; Mystakidi, V.C.; Goliopoulou, A.; Christoforatos, E.; Bourouki, E.; Vavouranaki, G.; et al. The association of diabetes mellitus with carotid atherosclerosis and arterial stiffness in the Corinthia study. *Nutr. Metab. Cardiovasc. Dis.* **2021**, *32*, 567–576. [[CrossRef](#)]
41. Wang, M.; Huang, J.; Wu, T.; Qi, L. Arterial Stiffness, Genetic Risk, and Type 2 Diabetes: A Prospective Cohort Study. *Diabetes Care* **2022**, *45*, 957–964. [[CrossRef](#)] [[PubMed](#)]
42. Hill, M.A.; Yang, Y.; Zhang, L.; Sun, Z.; Jia, G.; Parrish, A.R.; Sowers, J.R. Insulin resistance, cardiovascular stiffening and cardiovascular disease. *Metabolism* **2021**, *119*, 154766. [[CrossRef](#)] [[PubMed](#)]
43. Grunewald, Z.I.; Ramirez-Perez, F.I.; Woodford, M.L.; Morales-Quinones, M.; Mejia, S.; Manrique-Acevedo, C.; Siebenlist, U.; Martinez-Lemus, L.A.; Chandrasekar, B.; Padilla, J. TRAF3IP2 (TRAF3 Interacting Protein 2) Mediates Obesity-Associated Vascular Insulin Resistance and Dysfunction in Male Mice. *Hypertension* **2020**, *76*, 1319–1329. [[CrossRef](#)] [[PubMed](#)]
44. Ballard, K.D.; Quann, E.E.; Kupchak, B.R.; Volk, B.M.; Kawiecki, D.M.; Fernandez, M.L.; Seip, R.L.; Maresch, C.M.; Kraemer, W.J.; Volek, J.S. Dietary carbohydrate restriction improves insulin sensitivity, blood pressure, microvascular function, and cellular adhesion markers in individuals taking statins. *Nutr. Res.* **2013**, *33*, 905–912. [[CrossRef](#)] [[PubMed](#)]
45. de Oliveira Alvim, R.; Mourao-Junior, C.A.; de Oliveira, C.M.; de Faria Lima, R.; Horimoto, A.R.; Hong, V.A.; Bortolotto, L.A.; Krieger, J.E.; Pereira, A.C. Glycemic control and arterial stiffness in a Brazilian rural population: Baependi Heart Study. *Diabetol. Metab. Syndr.* **2015**, *7*, 86. [[CrossRef](#)] [[PubMed](#)]