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RESEARCH PAPER

Dietary inflammatory potential and arterial stiffness in a French cohort: Insights from the STANISLAS study

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Abstract *Background and aims:* Chronic inflammation plays a key role in arterial stiffness pathogenesis. Dietary components can display anti- or pro-inflammatory properties. Nonetheless, the association between the diet's overall inflammatory potential and arterial stiffness is unclear. This study aimed to assess the association between the diet's overall inflammatory potential and arterial stiffness assessed by carotid-femoral pulse wave velocity (cfPWV).

Methods and results: This cross-sectional study included 1307 participants from the STANISLAS family cohort study. Dietary data were collected using a validated food frequency questionnaire. The adapted dietary inflammatory index (ADII) score was calculated to assess the inflammatory potential of the participants' diet. The association of ADII score quartile with cfPWV was assessed using IPW-weighted linear mixed models with random family effect.

The median (Q1-Q3) ADII score was 0.45 (-1.57, 2.04). Participants exhibiting higher ADII scores demonstrated elevated energy intake, dietary saturated fat, and ultra-processed foods. Conversely, individuals with lower ADII scores exhibited higher vitamins and omega intakes, and a higher diet quality, as assessed by the DASH score. Despite these observations from the descriptive analyses, ADII score quartiles were not significantly associated with cfPWV (β(95% CI) were 0.01 (-0.02,0.04) for Q2, 0.02 (-0.01,0.05) for Q3, and 0.02 (-0.01,0.05) for Q4 compared to Q1).

Conclusion: In this cross-sectional study, participants had a relatively modest consumption of pro-inflammatory foods, no substantial associations were observed between the diet inflammatory potential and arterial stiffness. Further longitudinal studies in larger cohorts are needed to better understand the link between inflammatory diet and arterial stiffness.

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1. Introduction

Nutrition plays a pivotal role in human health. Recent findings suggest that diet may have a specific role in chronic or low-grade inflammation by modulating the levels of pro- and anti-inflammatory cytokines in the body [1]. The Western diet is characterized by foods with a high content of saturated fatty acids, sodium and added sugar, such as foods rich in refined cereals, red and processed meat, soft drinks, and processed products [2–5]. Consuming such a diet may lead to the activation of the innate immune system, leading to an excessive production of pro-inflammatory cytokines and a reduced production of anti-inflammatory cytokines [6]. On the other hand, healthy dietary patterns based on fruits, vegetables, and whole grains have shown the opposite effect. The Mediterranean diet is a notable example, given its richness in bioactive compounds including unsaturated fatty acids, polyphenols, fibers, phytosterols, vitamins, and minerals, which could exert an anti-inflammatory effect [7–10].

Inflammation is a well-recognized factor contributing to tissue damage and known to alter the structure and function of arterial walls, thereby playing a crucial role in the development of arterial stiffness [11,12], the latter of which is considered as a strong predictor of future cardiovascular disease [13]. Diet could represent a valuable means to modulate chronic or low-grade inflammation and limit the development of arterial stiffness. Rather than solely focusing on individual nutrients or specific diets, it would be insightful to assess the overall inflammatory impact of the diet. Several studies have used indices to evaluate diet quality in relation to inflammation. These indices were developed on the basis of a systematic review of available literature on diet and inflammation, and consist of several nutrients that have shown strong associations with inflammation such as vitamins, omegas 3 and 6 fatty acids, etc. Accordingly, several studies have demonstrated that a pro-inflammatory diet is associated with increased risks of cancer, all-cause mortality, and cardiovascular mortality [14–18]. Recently, two studies have explored the association between the overall inflammatory potential of the diet and arterial stiffness in specific populations. In the China National Nutrition and Health Survey involving 2644 women, the dietary inflammatory index was positively associated with arterial stiffness in women with diabetes and prediabetes, but not in women with normal glucose homeostasis [19]. In another study involving overweight/obese and sedentary Latin American individuals, a lower inflammatory index (indicating an anti-inflammatory diet) was correlated with lower aortic pulse wave velocity [20]. However, these two studies focused on specific populations, and the generalization of their findings to broader populations remains uncertain. Furthermore, in the study by Lin et al. [19] on a Chinese cohort, brachial ankle pulse wave velocity (PWV) was used as a measure of arterial stiffness rather than the gold standard, carotid femoral PWV [21].

The aim of the present study was to assess the association between the overall inflammatory potential of the diet and arterial stiffness, estimated with carotid femoral pulse wave velocity, in a French population-based family cohort study.

2. Methods

2.1. Population

The STANISLAS cohort (*Suivi Temporaire Annuel Non-Invasif de la Santé des Lorrains Assurés Sociaux*) is a longitudinal single-center familial study that included 4295 volunteers (1006 families), between 1993 and 1995, at the Preventive Medicine Center during a routine annual examination. Only families of French origin, residing in the Lorraine region (northeastern France), free of acute and chronic disease, and composed of at least two parents and two biological children aged older than 6 years old, were included in this cohort. The detailed description of the STANISLAS cohort has been published elsewhere [22].

From 1993 to 2016, four medical visits were organized at intervals of 5–10 years. The present study focused on the 4th visit of the cohort (2011–2016) where food data and outcomes were collected. Of the initially included 4295 participants, 1705 attended the 4th visit. Excluded from the analysis were 10 participants with missing dietary data, 63 participants with aberrant energy intakes (daily energy intake either below 1000 or above 5000 kcal), 32 participants with missing data on outcome, and 293 participants without the covariates required for the IPW calculation (see Statistical Analysis section below). The present cross-sectional analysis included 1307 participants (Fig. 1).

2.2. Assessment of diet

2.2.1. Food frequency questionnaire

Dietary data collection was collected at the fourth visit and was based on a validated food frequency questionnaire (FFQ) [23]. Briefly, participants reported the frequency of consumption and portion size of 133 foods over the last 3 months. Frequency of consumption was reported in the questionnaire based on 6 levels, ranging from “never or rarely” to “twice or more per day.” For portion size of each food, an estimate was made using standard serving sizes and food models. Daily nutrient intakes were calculated on a grams-per-day scale by multiplying the frequency of consumption of each food by the nutrient content of French food composition database established by the French Food Quality Data Center (Ciqual, 2013 version). The omega-6 intake was the sum of linoleic acid and arachidonic acid, and the omega-3 intake the sum of alpha-linolenic acid + eicosapentaenoic acid + docosahexaenoic acid + docosapentaenoic acid.

2.2.2. Assessment of the dietary inflammatory index

The dietary inflammatory potential was calculated using the Adapted Dietary Inflammatory Index (ADII), as

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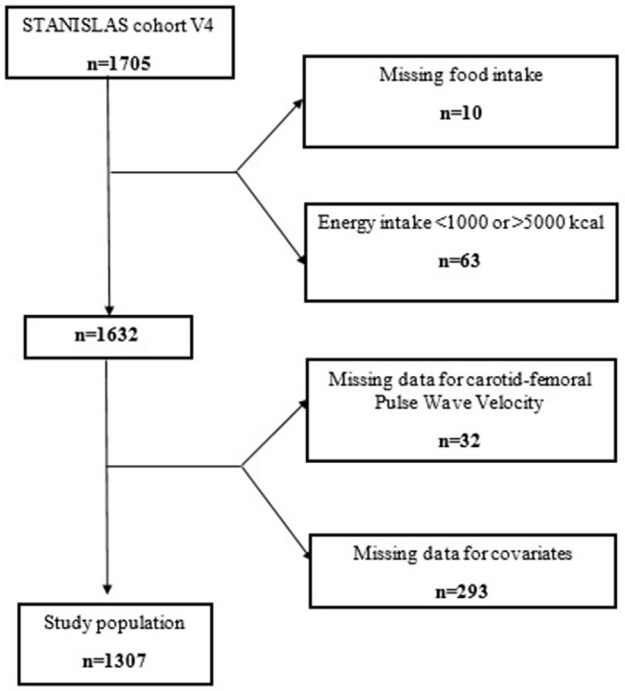

Figure 1 Study flow chart.

proposed by Woudenbergh et al. [24]. This score was derived from the literature-based score developed by Cavicchia et al. to reflect the inflammatory potential of an individual's diet [25].

The scoring system is based on the inflammatory weights of dietary components obtained based on the literature whereby an inflammatory weight was assigned to each dietary component according to their effect on biological inflammatory markers such as interleukins (IL-1β, IL-6, IL-4 and IL-10), TNFα, and C-reactive protein (CRP) [25]. We used the most up-to-date weights proposed by Shivappa et al. [26], instead of those of Cavicchia et al., as previously done in another recent study [27]. The list of inflammatory weights of each component is described in Supplementary Table 1. The inflammatory weight of ethanol was assumed to be zero when ethanol consumption exceeded than 40 g/day, as ethanol consumption is unlikely to be anti-inflammatory beyond that threshold [28].

Of the 31 proposed dietary components, 24 were available in the STANISLAS cohort. In a first instance, intakes were energy-adjusted using the residual method [29] and subsequently standardized. These standardized energy-adjusted intakes were then multiplied by the inflammatory weights of each dietary components, and summed.

Positive ADII values indicate a pro-inflammatory diet while negative values correspond to an anti-inflammatory diet. In comparison to the score initially developed by Cavicchia et al. [25], and other score like the inflammatory score of the diet (ISD) [30] or the dietary inflammatory index (DII) [26], the ADII score presents the advantage of using standardized energy-adjusted intake to reduce between-person variation in dietary intake resulting from differences in physical activity, body size, and metabolic efficiency. Several components were also excluded from the score calculation, such as energy or total lipid intakes, because it is likely that the inflammatory effect of energy is the sum of the inflammatory effects of all energy-providing macronutrients, and because it was assumed that the inflammatory effect of total fat is the sum of the inflammatory effects of all separate fatty acids [24]. Data available in the STANISLAS cohort database fitted better the ADII requirement calculation. Moreover, ADII and DII scores were found strongly correlated in the SU.VI.MAX cohort [31].

3. DASH score and ultra-processed food intake

Also, in order to assess the global quality of the diet, the Dietary Approaches to Stop Hypertension (DASH)-derived diet quality score was calculated [32–34] based on 9 dietary components, namely: fruits; vegetables; grain products (total and whole grains); dairy products and alternatives; processed meat, fish; nuts, legumes, and seeds; oils and fats (total and saturated fats); sodium; sugar and sweets. A value from 0 to 10 was assigned for each food group on the basis of compliance to the DASH recommendations. The resulting component scores were summed to create the overall DASH score, which could range from 0 to 90. For each individual, serving sizes were individually expressed for a total energy intake of 2000 kcal.

Ultra-processed food intake was assessed through the NOVA classification, and reported as the number of portions of ultra-processed food consumed per day [35].

3.1. Carotid-femoral pulse wave velocity (cfPWV)

Carotid-femoral pulse wave velocity was measured using the Complior device (ALAM Medical, France) after a 10-min supine rest period. This measuring device allows the simultaneous recording of arterial pulse waves at the carotid and femoral sites, in accordance with the recommendations of the European Network for Noninvasive Investigation of Large Arteries [36]. For the recording, two sensors were placed simultaneously on the carotid and femoral arteries. Two measurements were made, with cfPWV calculated as their average. If the first two measurements differed by more than 0.5 m/s, then a third measurement was made and the cfPWV was calculated as the median of the three measurements. The transit time and the carotid-to-femoral, carotid-to-sternal notch, and sternal-to-carotid notch distances were also measured using the embedded foot-to-foot algorithm and a tape measure, respectively. CfPWV (m/s) was obtained by multiplying the direct distance by 0.8.

3.2. Covariates

At each visit, all volunteers underwent a complete clinical examination, including measurements of weight, height, waist circumference and blood pressure. Body mass index

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was calculated by dividing weight (kg) by height squared (m) [22]. A self-reported questionnaire was completed by the participants to collect demographic and sociodemographic information such as age, sex, education level (low, middle, and high), marital status, smoking status, as well as information on the occurrence of diseases during follow-up and treatments [22]. Physical activity was assessed using the International Physical Activity Questionnaire, and weekly energy expenditure was expressed in minutes of equivalent metabolic tasks per week. Blood samples were also taken and serum concentrations of biomarkers such as fasting blood glucose, HDL, LDL, and triglycerides were measured. Blood pressure was taken both in the office as well as on a 24-h ambulatory basis [22]. Briefly, STANISLAS participants underwent 24-h ambulatory blood pressure recordings using the Spacelabs 90207(Spacelabs Medical) monitor, with a monitoring cuff placed around the non-dominant arm. The blood pressure system was programmed to take measurements every 15 min from 6 a.m. to 10 p.m. and every 30 min from 10 p.m. to 6 a.m. [37].

3.3. Ethics

The study was conducted in accordance with the Declaration of Helsinki. The protocol was approved by the local ethics committee (*Comité de Protection des Personnes Est III, Nancy, France*). All participants signed a written informed consent.

3.4. Statistical analysis

First, participant characteristics are described according to ADII quartiles using mean ± standard deviation (SD) or median (Q1 - Q3, which represent 25th and 75 percentiles, respectively) in case of non-normal distribution for continuous variables and frequency (percentages) for categorical variables. Differences between the groups were assessed using the chi-square test for categorical variables and ANOVA or Kruskal-Wallis tests for continuous variables, as appropriate.

Second, the associations between continuous ADII score and ADII score quartiles and cfPWV were assessed using linear mixed models with random family effect. The cfPWV variable was log-transformed to obtain a more normal distribution, and the following adjustment variables were considered: age, sex, education level, smoking status, waist circumference, physical activity, SBP, antihypertensive treatments, antidiabetic treatments, and lipid-lowering treatments.

Third, given the large differences in participant characteristics, notably in terms of age and sex in ADII quartiles (Table 1), an inverse probability of weighting (IPW) analysis was performed using stabilized weights. Weights were calculated using propensity scores estimated from multinomial logistic regression model with ADII quartiles as dependent variable (Q1 as reference), and the following well-known risk factors as explanatory variables: age, sex, education level, smoking status, waist circumference, physical activity, SBP, antihypertensive treatments, antidiabetic treatments, and lipid-lowering treatments. The stabilized weights were then computed using the marginal probability of each group. The balance of covariates between the quartiles was assessed after weighting by calculating the mean of all pairwise absolute standardized mean differences (ASMDs). For each variable and the groups, the ASMD represents the absolute difference between the mean values in the groups divided by the common SD.

Table 1 Characteristics of the study population according to ADII score quartiles (n = 1307), before weighting on IPW.

	Overall	Quartile 1 [-16.62, −1.57]	Quartile 2 [-1.57, 0.45]	Quartile 3 [0.45, 2.04]	Quartile 4 [2.04, 8.08]	p-value
Women, n (%)	667 (51.0)	202 (62.0)	168 (51.5)	165 (50.5)	132 (40.2)	<0.001
Age, years	55.0 [34.0, 60.0]	58.0 [42.2, 61.0]	56.5 [37.0, 61.0]	55.0 [33.0, 60.0]	37.0 [31.0, 58.0]	<0.001
Generation 2	546 (41.8)	85 (26.1)	122 (37.4)	146 (44.6)	193 (58.8)	<0.001
Education level						0.070
<High school degree	514 (39.3)	137 (42.0)	127 (39.0)	124 (37.9)	126 (38.4)	
>2 years after high school	355 (27.2)	91 (27.9)	95 (29.1)	98 (30.0)	71 (21.6)	
0−2 years after high school	438 (33.5)	98 (30.1)	104 (31.9)	105 (32.1)	131 (39.9)	
Physical activity (MET/min)	1786.8 [655.8, 4215.6]	1947.9 [821.3, 4220.8]	1776.9 [746.7, 4350.0]	1872.0 [648.5, 4158.0]	1576.5 [476.0, 4158.0]	0.171
BMI (kg/m^2)	25.0 [22.5, 28.2]	25.3 [22.8, 28.8]	24.7 [22.5, 27.9]	24.7 [22.5, 28.3]	25.0 [22.4, 27.8]	0.259
Waist circumference	89.3 (13.5)	89.8 (13.7)	88.8 (12.7)	89.1 (14.1)	89.8 (13.5)	0.709
Smoking status						<0.001
Current smoker	258 (19.7)	36 (11.0)	69 (21.2)	67 (20.5)	86 (26.2)	
Never smoker	623 (47.7)	162 (49.7)	159 (48.8)	158 (48.3)	144 (43.9)	
Past smoker	426 (32.6)	128 (39.3)	98 (30.1)	102 (31.2)	98 (29.9)	
Use of anti-diabetic drugs, n (%)	46 (3.5)	14 (4.3)	12 (3.7)	11 (3.4)	9 (2.7)	0.752
Use of antihypertensive drugs, n (%)	245 (18.7)	73 (22.4)	74 (22.7)	54 (16.5)	44 (13.4)	0.004
Use of lipid-lowering drugs, n (%)	189 (14.5)	63 (19.3)	51 (15.6)	47 (14.4)	28 (8.5)	0.001
Systolic blood pressure (mmHg)	125.3 (15.1)	125.2 (15.8)	125.5 (14.8)	125.7 (15.7)	124.7 (14.1)	0.859
Pulse Wave Velocity (m/s)	8.5 ± 1.7	8.5 ± 1.6	8.5 ± 1.7	8.5 ± 1.6	8.5 ± 1.6	0.31

ADII, adapted dietary inflammatory index; MET, metabolic equivalent minutes; BMI, body mass index; Continuous variables are expressed as the mean and standard deviation in the case of normal distribution, and median and quartiles in other cases. Categorical variables were described as numbers and percentages.

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Thereafter, the associations between the ADII score quartiles and cfPWV were assessed using IPW-weighted linear mixed models with random family effect. The cfPWV variable was log-transformed to obtain a more normal distribution.

Analyses were performed using SAS (version 9.4, SAS Institute Inc.). A two-sided p value of <0.05 was considered significant.

4. Results

4.1. Characteristics of the study population

The median (Q1-Q3) ADII score was 0.45 (−1.57, 2.04) and ranged from −16.63 to 8.08. The participants had a median age of 55 (34–60) years, and 51% were women. The mean (±SD) cfPWV was 8.5 ± 1.7 m/s, 4% had diabetes, mean blood glucose was 4.95 ± 0.88 mmol/L and median CRP was 1.4 (0.7–3.2) mg/L. Before weighting, participants with a higher ADII score (more pro-inflammatory) were more likely to be men, young, smokers, and have a lower education level compared to participants with a low ADII score. Participants with a low ADII score (more anti-inflammatory) were on more medications (Table 1). After weighting, all mean ASMD values were less than or equal to 0.10, indicating that the quartiles were well balanced (Supplementary Table 2).

Total energy intake, dietary intakes of SFAs, and ultra-processed food were higher in participants with higher ADII scores than in those with lower ADII scores. Intakes of vegetable protein, PUFAs, β-carotene, vitamin A, all vitamins B, vitamin C, vitamin D, vitamin E, fiber, iron, magnesium, tea, omega-3, and omega-6 were higher in participants with lower ADII scores than those with higher ADII scores. Participants with lower ADII scores had a higher DASH score, then higher diet quality, compared to those with a higher ADII score (Table 2).

4.2. Cross-sectional association between ADII score and arterial stiffness

In the crude models, no association was found between ADII, considered either continuously or in quartiles, and cfPWV (Supplementary Table 3). In the multi-adjusted models, cfPWV was significantly associated with continuous ADII score (Beta (95% CI):0.004 (0.001; 0.007), p = 0.005), as well as ADII score Q3 and Q4 (0.03 (0.008; 0.05), p = 0.01; 0.03 (0.008; 0.05), p = 0.009, respectively − see Supplementary Table 3). However, after IPW analyses, none of the ADII score quartiles was significantly associated with cfPWV. Beta (95% CI) and p-values were 0.01 (−0.02, 0.04), p = 0.32, for Q2, 0.02 (−0.01, 0.05), p = 0.14 for Q3, and 0.02 (−0.01, 0.05), p = 0.13 for Q4 compared to Q1 (Table 3). We also examined interactions between the ADII score and obesity, as well as waist circumference. However, none of these interactions were found to be significant (p = 0.37 and 0.30, respectively). Further adjustment for diet quality (DASH score and ultra-processed food intake) did not alter the results (beta (95% CI) and p-values were 0.01 (−0.02, 0.04), p = 0.46, for Q2, 0.02 (−0.01, 0.05), p = 0.23 for Q3, and 0.02 (−0.01, 0.07), p = 0.11 for Q4 compared to Q1).

5. Discussion

In this cross-sectional analysis of the STANISLAS cohort, which included both men and women, no significant association was observed between ADII score and cfPWV.

Previous research has indicated that diet may influence inflammation and have an impact on various health outcomes [1,18]. Until now, only two epidemiological investigations have delved into the relationship between the inflammatory nature of diet and arterial stiffness within specific populations. A study from the China National Nutrition and Health Survey showed a positive association between an inflammatory dietary score and brachial–ankle PWV in women with prediabetes and diabetes, but not in women with normal glucose [19]. In our French population, we had a limited number of diabetic participants and after weighting in which the ADII quartiles were well balanced, our findings did not reveal an association between ADII score and cfPWV. Our population was likely too healthy making it difficult to observe any association. Indeed, a cfPWV above 10 m/s is indicative of higher cardiovascular risk [38,39], in our case the Q3 (75th percentile) of PWV distribution is 9.31 m/s, less than 10% of our participants had a high PWV. Also, mean PWV was found to be lower in our study (8.5 m/s) compared to other studies involving for instance the French Parisian population, where the mean PWV was above 11 m/s [40,41].

In a study involving overweight and obese Latin American subjects, participants were categorized into two subgroups based on their diet scores: one with anti-inflammatory diet score, and another with pro-inflammatory diet score. A negative correlation between the DII score and PWV was found in the subgroup with anti-inflammatory diet only [20]. The range of ADII scores (−16.62 to 8.09) observed in our study was similar, albeit less anti-inflammatory, to that reported in a recent case-control study of another French metropolitan study involving another disease (−23.56 to 12.38) [16], using the same score and updated weights. It is possible that the present study lacked sufficient statistical power and variability in the anti-inflammatory scores to detect any significant associations. In addition, while the median ADII score remains at around 0 in several French studies [16,42,43] including the current analysis, thus denoting a relatively neutral diet with regard to inflammation, the median ADII in the American NHANES cohort was 1.4 indicating a slight pro-inflammatory diet [44]. Indeed, the American diet tends to lean more toward the Western diet model, characterized by high intakes of processed and refined foods, red and processed meats, added sugars, and saturated and trans fats along with low intakes of fruits, vegetables, whole grains, and nuts [45]. These disparities in dietary choices could explain the divergent inflammatory profiles observed, which in turn could shed light on the almost neutral ADII score

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Table 2 Food intake of the study population (n = 1307).

	ADII Score					
	Overall	Quartile 1	Quartile 2	Quartile 3	Quartile 4	p-values
		[-16.62, −1.57]	[-1.57, 0.45]	[0.45, 2.04]	[2.04, 8.08]	
N	1307	325	336	328	318	
Energy (kcal/d)	2355.09 ± 815.83	2402.33 ± 839.67	2246.86 ± 779.81	2218.56 ± 735.95	2561.98 ± 862.11	<0.0001
Total protein (g/d)	84.77 ± 16.24	86.99 ± 17.52	86.97 ± 17.41	84.55 ± 15.42	80.41 ± 13.31	<0.0001
Vegetable protein (g/d)	26.12 ± 6.40	28.56 ± 6.68	26.05 ± 6.12	25.51 ± 5.98	24.31 ± 6.07	<0.0001
Animal protein (g/d)	57.53 ± 18.36	57.17 ± 19.41	59.39 ± 19.68	58.08 ± 18.30	55.36 ± 15.49	0.040
Total fat (g/d)	82.18 ± 15.76	80.55 ± 17.32	81.80 ± 15.26	82.92 ± 14.28	83.49 ± 15.97	0.086
SFAs (g/d)	31.43 ± 7.21	28.43 ± 6.63	30.63 ± 6.61	32.47 ± 6.72	34.28 ± 7.56	<0.0001
MUFAs (g/d)	32.58 ± 8.00	31.88 ± 8.25	32.99 ± 8.21	32.83 ± 7.78	32.62 ± 7.72	0.29
PUFAs (g/d)	12.45 ± 4.37	14.62 ± 5.88	12.59 ± 3.84	11.87 ± 3.26	10.67 ± 2.87	<0.0001
Cholesterol (mg/d)	317.68 ± 93.25	303.19 ± 100.23	323.90 ± 97.34	324.18 ± 93.05	319.19 ± 79.42	0.011
Carbohydrates (g/d)	215.32 ± 40.18	219.27 ± 41.32	212.51 ± 41.43	214.25 ± 38.25	215.37 ± 39.48	0.17
Fiber (g/d)	20.69 ± 6.35	26.98 ± 7.05	21.29 ± 4.36	18.78 ± 3.73	15.61 ± 3.24	<0.0001
Vitamin A (μg/d)	578.11 ± 405.39	627.57 ± 448.37	637.61 ± 474.24	553.89 ± 375.62	489.66 ± 271.40	<0.0001
β-Carotene (μg/d)	4730.11 ± 3726.74	7808.98 ± 5290.90	4947.50 ± 2616.72	3749.69 ± 1905.55	2365.01 ± 1118.50	<0.0001
Vitamin B-1 (mg/d)	1.24 ± 0.27	1.44 ± 0.31	1.26 ± 0.22	1.19 ± 0.20	1.07 ± 0.18	<0.0001
Vitamin B-2 (mg/d)	1.67 ± 0.43	1.86 ± 0.48	1.73 ± 0.42	1.61 ± 0.35	1.48 ± 0.35	<0.0001
Vitamin B-3 (mg/d)	17.91 ± 4.57	19.95 ± 4.85	18.64 ± 4.53	17.41 ± 4.05	15.56 ± 3.59	<0.0001
Vitamin B-6 (mg/d)	1.89 ± 0.53	2.35 ± 0.61	1.97 ± 0.41	1.76 ± 0.33	1.48 ± 0.26	<0.0001
Vitamin B-9 (μg/d)	315.77 ± 105.25	428.38 ± 116.01	327.13 ± 65.79	280.69 ± 49.59	224.87 ± 45.55	<0.0001
Vitamin B-12 (μg/d)	6.13 ± 3.07	6.98 ± 3.55	6.88 ± 3.36	5.78 ± 2.62	4.83 ± 1.94	<0.0001
Vitamin C (mg/d)	115.58 ± 73.47	175.14 ± 95.89	120.55 ± 56.27	97.12 ± 46.47	68.48 ± 32.04	<0.0001
Vitamin D (μg/d)	3.56 ± 2.54	4.64 ± 3.49	4.06 ± 2.47	3.17 ± 1.68	2.32 ± 1.28	<0.0001
Vitamin E (mg/d)	11.08 ± 3.37	13.34 ± 3.72	11.40 ± 2.92	10.44 ± 2.78	9.08 ± 2.43	<0.0001
Iron (mg/d)	12.33 ± 2.37	13.92 ± 2.70	12.88 ± 2.18	11.78 ± 1.56	10.70 ± 1.47	<0.0001
Magnesium (mg/d)	319.72 ± 64.74	369.36 ± 74.13	331.55 ± 54.44	303.64 ± 42.77	273.07 ± 38.84	<0.0001
Caffeine (g/d)	0.18 ± 0.20	0.19 ± 0.22	0.19 ± 0.20	0.18 ± 0.20	0.16 ± 0.17	0.20
Tea (g/d)	123.93 ± 265.47	231.66 ± 395.11	145.58 ± 238.34	93.94 ± 201.83	21.87 ± 70.40	0.020
n-3 PUFAs (g/d)	1.54 ± 0.71	2.04 ± 0.96	1.65 ± 0.60	1.37 ± 0.41	1.10 ± 0.30	<0.0001
n-6 PUFAs (g/d)	10.25 ± 3.79	11.89 ± 4.99	10.29 ± 3.46	9.84 ± 3.05	8.96 ± 2.64	<0.0001
Ethanol (g/d)	8.15 ± 10.03	6.84 ± 8.75	8.98 ± 10.68	7.90 ± 8.68	8.89 ± 11.62	0.020
Ultra-processed food (serving/day)	4.68 ± 2.47	4.32 ± 2.59	4.44 ± 2.26	4.67 ± 2.21	5.31 ± 2.70	<0.0001
DASH_score	45.19 ± 20.22	49.25 ± 19.90	49.16 ± 20.48	46.48 ± 20.85	35.53 ± 16.06	<0.0001

ADII, adapted dietary inflammatory index; SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; DASH, Dietary approach to stop hypertension.
Continuous variables are expressed as the mean and standard deviation in the case of normal distribution, and median and quartiles in other cases.
The reported intake values were based on a daily energy consumption of 2000 kcal.

documented in our current French study, and also suggest that our study population did not have a sufficiently high ADII to observe an association.

Of note, in the literature, the associations between dietary inflammatory score and PWV were found only in the presence of metabolic disorders, as well as in the case of overweight and obese participants. Since diabetes and obesity are known to be pro-inflammatory or associated with inflammation [46,47], it raises the question of whether such associations may exist only in the context of pre-existing inflammatory conditions. This suggests that an anti-inflammatory diet might be more beneficial for individuals with pre-existing inflammatory conditions. Interestingly, an intervention involving a low cholesterol/low saturated fat diet in hypercholesterolemia patients for 8 weeks resulted in a significant 11% decrease in aortic PWV [48]. This decrease was predicted by the reduction in plasma CRP levels following the diet, rather than by a modest reduction in plasma cholesterol levels. Thus, attenuating systemic inflammation through diet may

Table 3 Cross-sectional association between ADII score quartiles and carotid-femoral pulse-wave velocity weighted on IPW.

Models	Quartile 1 [-16.62, −1.57]	Quartile 2 [-1.57, 0.45]	Quartile 3 [0.45, 2.04]	Quartile 4 [2.04, 8.08]
Log (cfPWV) β (95% CI)	Reference	0.01 (−0.02, 0.04) p = 0.32	0.02 (−0.01, 0.05) p = 0.14	0.02 (−0.01, 0.05) p = 0.13

β, regression coefficient; CI, 95% confidence interval; cfPWV, carotid-femoral pulse-wave velocity. Model was weighted on inverse probability of weighting (IPW). Weights were calculated using propensity scores estimated from logistic regression models including well-known risk factors: age, sex, education level, smoking status, waist circumference, physical activity, SBP, antihypertensive treatments, antidiabetic treatments, and lipid-lowering treatments.

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improve arterial stiffness in the case of a pre-existing inflammatory condition.

However, it is important to note that both our study and the China National Nutrition and Health Survey study by Lin et al. (2021) were cross-sectional in nature. As a result, we can only draw the conclusion that there may be no sizeable association between the level of diet-related inflammation and pulse wave velocity (PWV) at a single time point within the general population. Many factors can induce a shift in dietary habits, such as moving away from the parental home, becoming a parent, or experiencing illness [49]. Further longitudinal studies are needed to explore this relationship in a more comprehensive manner.

5.1. Strengths and limitations

This study features several strengths. First, the analyses were based on a large, general population cohort with complete information regarding food intake, measured by a validated questionnaire. Second, this study was based on quality data owing to the availability of a wide variety of detailed health information data.

Despite its strengths, this study has several limitations that should be considered. First, the results are based on cross-sectional data, and hence the observational nature of the study precludes establishing a causal relationship. Besides this study being cross-sectional does not allow to study temporal sequence. Second, the calculation of ADII score relies on a single-point FFQ which may not fully capture changes in food intake quantity and quality. Longitudinal studies are needed to better understand the relationship between ADII score and PWV. Third, the FFQ questionnaire assessed food intake over the past 3 months. Although seasonal variation may exists, previous works indicates that over time, increased food imports and improved insulation have minimized the seasonal fluctuations in total energy and nutrient intake, resulting in relative stability throughout the year [50]. Besides no interaction was found in our study between ADII score and seasons (all p-value of interaction$>$0.24). Fourth, as frequently observed in other studies using dietary inflammatory scores, intakes of some dietary components could not be calculated from our food frequency questionnaire, i.e. polyphenols, zinc and selenium which could potentially introduce bias in the estimation of the ADII score. Fifth, although the current analysis accounted for confounding variables, there is a possibility that unmeasured confounders might still influence our observations. Lastly, the generalizability of our results may be limited due to the inclusion of participants solely from the Lorraine region. Further studies are necessary to confirm and expand upon these findings.

6. Conclusion

In this cross-sectional analysis carried out in the setting of a family population-based cohort study, no significant association was found between the overall inflammatory potential of the diet and arterial stiffness. Further longitudinal studies in larger cohorts are needed to better comprehend the link between inflammatory diet and arterial stiffness, while also exploring the inflammatory status of the participants.

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Author contributions

PR, NG, and JMB designed the fourth visit of the STANISLAS cohort. LM performed the data management. EB and NG supervised the cardiovascular (arterial stiffness and thickness, and echocardiography, respectively) assessments. SW, JMB, NG, LDA designed the present research. LDA, ZL, KD and SW performed the statistical analysis. LDA, SW and JMB drafted the manuscript.

All authors were involved in the interpretation of the results and the critical review of the manuscript.

Declaration of competing interest

NG received honoraria from AstraZeneca, Bayer, Boehringer, Echosens, Lilly, Roche diagnostics, Novartis. PR: reports consulting for Idorsia, G3P, honoraria from AstraZeneca, Bayer, Boehringer-Ingelheim, Cincor, CVRx, Fresenius, KBP biosciences, Novartis, NovoNordisk, Relypsa, Servier, and Vifor Fresenius Medical Care Renal Pharma; and travel grants from AstraZeneca, Bayer, CVRx, Novartis, and Vifor Fresenius Medical Care Renal Pharma; Cofounder: CardioRenal. The other co-authors have no conflicts of interest to disclose related to the submitted work.

Appendix A. Supplementary data

The list of acknowledgment and Supplementary data to this article can be found online at https://doi.org/10.1016/j.numecd.2024.03.022.

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