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Inflammatory diet and preclinical cardiovascular phenotypes in 11–12 year-olds and mid-life adults: A cross-sectional population-based study

Addison Davis[a,b], Richard Liu[a,b], Jessica A. Kerr[a,b], Melissa Wake[a,b,c], Anneke Grobler[a,b], Markus Juonala[b,e,f], Mengjiao Liu[a,b], Louise Baur[g,h], David Burgner[a,b,d,i,1], Kate Lycett[a,b,j,*,1]

[a] The University of Melbourne, Parkville, Victoria, Australia
[b] Murdoch Children's Research Institute, Parkville, Victoria, Australia
[c] Liggins Institute and Depart of Paediatrics, The University of Auckland, Auckland, New Zealand
[d] The Royal Children's Hospital, Parkville, Victoria, Australia
[e] Department of Medicine, University of Turku, Turku, Finland
[f] Division of Medicine, Turku University Hospital, Turku, Finland
[g] The Children's Hospital, Westmead, NSW, Australia
[h] The University of Sydney, Westmead, NSW, Australia
[i] Department of Pediatrics, Monash University, Clayton, VIC, Australia
[j] Deakin University, Burwood, VIC, Australia

HIGHLIGHTS

- Pro-inflammatory diet may be a modifiable risk factor for cardiovascular disease.
- In adults, a pro-inflammatory diet was associated with cardiovascular phenotypes.
- Associations in adults were seen for vascular function and microvascular structure.
- There was little evidence of associations in children.

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ABSTRACT

Background and aims: Pro-inflammatory diet may be a modifiable risk factor for cardiovascular disease. We examine associations of two inflammatory diet scores with preclinical cardiovascular phenotypes at two life course stages.

Methods: Participants: 1771 children (49% girls) aged 11–12 years and 1793 parents (87% mothers, mean age 43.7 (standard deviation 5.2) years) in the Child Health CheckPoint Study. Measures: 23 items in the Australian National Secondary Students' Diet and Activity (NaSSDA) survey were used to derive two inflammatory diet scores based on: 1) published evidence of associations with C-reactive protein (literature-derived score), and 2) empirical associations with CheckPoint's inflammatory biomarker (glycoprotein acetyls, GlycA-derived score). Cardiovascular phenotypes assessed vascular structure (carotid intima-media thickness, retinal vessel calibre) and function (pulse wave velocity, blood pressure). Analyses: Linear regression models were conducted, adjusted for age, sex, socioeconomic position and child pubertal status, plus a sensitivity analysis also including BMI (z-score for children).

Results: In adults, both inflammatory diet scores showed small associations with adverse cardiovascular function and microvascular structure. Per standard deviation higher GlycA-derived diet score, pulse wave velocity was 0.17 m/s faster (95% CI 0.11 to 0.22), mean arterial pressure was 1.85 mmHg (1.34–2.37) higher, and retinal arteriolar calibre was 1.29 μm narrower (−2.10 to −0.49). Adding BMI to models attenuated associations to-

* Corresponding author. Centre for Community Child Health, Royal Children's Hospital Flemington Road, Parkville VIC, 3052, Australia.
E-mail address: kate.lycett@mcri.edu.au (K. Lycett).
[1] These authors contributed equally to this work as joint senior authors.

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wards null. There was little evidence of associations in children.
Conclusions: Our findings support cumulative adverse effects of a pro-inflammatory diet on preclinical cardiovascular phenotypes across the life course. Associations evident by mid-life were not present in childhood, when preventive measures should be instituted.

1. Introduction

Inflammation is a central pathogenic mechanism and therapeutic target in atherosclerosis [1]. A pro-inflammatory diet is considered a modifiable risk factor for atherosclerotic cardiovascular disease (CVD) [2–4], and western diets have a particularly high pro-inflammatory potential [5]. Specific food groups, such as refined carbohydrates, red/processed meat and sugar-sweetened beverages, are suggested to promote inflammation [6]. Others, such as fish, fruit, vegetables, and low or non-fat dairy sources, have demonstrated anti-inflammatory effects in both animal and human studies [7]. However, investigating only the pro- or anti-inflammatory elements of diets neglects potential synergistic and antagonistic interactions.

The Dietary Inflammatory Index (DII) is a literature derived score that is widely used to capture both pro- and anti-inflammatory dietary patterns [8]. Although DII scores do not always relate to inflammatory biomarkers or inflammatory-driven diseases [9,10], meta-analytic evidence does imply that, overall, higher (i.e. pro-inflammatory) adult DII scores predict CVD events. Two large meta-analyses including prospective and cross-sectional studies of adults aged 38–69 years from the general population reported that those in the highest DII category, compared to the lowest, had a 20–30% increased risk of cardiovascular mortality and disease [2,11]. A pro-inflammatory diet is also associated with increased cardiovascular risk in early to mid-adulthood. For example, two recent NHANES studies demonstrated that a) among 7880 adults aged ≥20 years (mean age ranged 35–51 years), higher DII scores were associated with increased prevalence of obesity, diabetes, hypertension and hypercholesterolemia [12], and b) among 17,689 adults (mean age 47 years), the odds of higher blood pressure and other cardiometabolic risk factors increased across DII quintiles [13]. Smaller European cohorts report similar results for blood pressure [4].

Cardiovascular risk develops across the life course, with childhood a key period for primordial prevention [14]. In 6-24 year-olds, diet quality is clearly associated with dietary anti-inflammatory potential measured by the DII [15], but very little research has directly examined the relationship between children's inflammatory diets and cardiovascular risk factors and preclinical phenotypes. One recent community-based study reported that children's DII scores were associated with their waist-to-height ratio, but not with body composition, blood pressure or heart rate [16]. Therefore, it is still unknown whether the adverse associations seen in adult studies are already evident in childhood, when early atherosclerosis develops and when adverse risk trajectories are established [17]. We therefore aimed to examine associations of inflammatory diet scores with preclinical phenotypes of cardiovascular structure (carotid intima-media thickness, retinal vessel calibre) and function (pulse wave velocity, blood pressure) in population-based cohorts of 11–12 year-olds and mid-life adults.

2. Materials and methods

2.1. Study design and participants

The Child Health CheckPoint study (CheckPoint) was a cross-sectional population-based study nested within the national Longitudinal Study of Australian Children (LSAC). LSAC's initial study design and recruitment are outlined elsewhere [18]. Briefly, LSAC recruited a nationally-representative birth (B) cohort of 5107 infants at age 0–1 years using a two-stage random sampling design, with biennial 'waves' of data collection thereafter [19]. CheckPoint was a detailed cross-sectional biophysical assessment, nested between LSAC's 6th and 7th waves from February 2015 to March 2016. Of the 3764 (74%) families retained at wave 6, 3513 (93%) consented to their contact details being shared with the CheckPoint team. CheckPoint's study design and methods are outlined in detail elsewhere [20], and the procedures and measures germane to this study are outlined below. Informed consent for each child was provided by a parent/guardian. The Royal Children's Hospital (HREC33225) and the Australian Institute of Family Studies (AIFS14-05) Ethics Committees approved the study.

2.2. Procedures

Most participants attended a 3.5 h Main or 2.5 h Mini Assessment Centre in one of Australia's capital cities or large regional towns, where they rotated through a series of 15-min physical assessment and biospecimen collection stations. Participants unable to attend a centre were offered a 90-min home visit. We draw on data collected from all three types of assessments, although one cardiovascular measure was omitted at Mini Assessment centres and two measures at home visits (see below).

2.3. Measures

We draw on measures from CheckPoint and descriptive variables LSAC's wave 1 to 6.

2.3.1. Inflammatory diet scores

Self-reported dietary intake was collected via iPad using REDCap (Research Electronic Data Capture), a secure web-based application. Children and adults separately completed a subset of 26 questions from a standardised food frequency questionnaire, modified from the National Secondary Students' Diet and Activity (NaSSDA) survey [21]. The NaSSDA was designed to monitor Australian secondary students' diet (items included in CheckPoint) and also food marketing exposure and physical activity (not included in CheckPoint, which instead collected accelerometry data). Participants reported their usual daily or weekly intake of a range of foods and drinks, including fruit, vegetables, confectionary, red meat, fish and fruit juice. The level of detail precluded calculating a DII for participants. Therefore we used each relevant NaSSDA item to derive two CheckPoint inflammatory diet scores: 1) a score derived from published literature (the literature-derived score), and 2) a GlycA-derived score based on the statistical correlation between each dietary survey item and levels of the inflammatory biomarker (glycoprotein acetyls, GlycA) [22]. For both scores, we excluded three NaSSDA items related to breakfast skipping, breakfast cereal and diet drink intake, as the nutritional content of breakfast cereal items are highly variable and the inflammatory potential of diet drinks or skipping breakfast is uncertain [6].

The ***literature-derived inflammatory diet score*** was guided by two highly cited reviews [23,24] that determined the 'inflammatory potential' of different food and beverage components from their predicted effects on C-reactive protein (CRP), a known biomarker of inflammation. We categorised each NaSSDA item as either anti-inflammatory (e.g. fish consumption) or pro-inflammatory (e.g. red meat consumption) from their reported associations with CRP. We then assigned each item's response options a value from −2 (anti-inflammatory) to +2 (pro-inflammatory). Finally, we summed all items to generate an overall literature-derived inflammatory diet score for each participant ranging from −6 to +29, where higher scores indicate a more pro-

Table 1
NaSSDA food items[a] and generation of both literature-derived and GlycA-derived scoring systems (exposures)..

Category (Frequency)	Question	Literature-derived score frequency			Adult GlycA-derived scores for each category		
		Assigned frequency	Child Mean (SD)	Adults Mean (SD)	Univariable *p*	Model 1 β; *p*	Model 2 β; *p*
Fish (Weekly)	How often do you eat fish, including canned fish?	**0:** 0 or 0.5 times −**1:** 1.5 or 3.5 times −**2:** 5.5 or 7 times	−0.4 (0.5)	−0.6 (0.5)	< 0.001	−0.0142; < 0.001	−0.0131; < 0.001
Vegetables (Daily)	How many serves of vegetables do you usually eat each day?	**0:** 0, 0.5 or 1 serves −**1:** 2 or 3 serves −**2:** 4, 5 or 6 serves	−0.9 (0.7)	−1.1 (0.6)	< 0.001	−0.0121; < 0.001	−0.0133; < 0.001
Fruit (Daily)	How many serves of fruit do you usually eat each day?	**0:** 0, 0.5 or 1 serves −**1:** 2 or 3 serves −**2:** 4, 5 or 6 serves	−1.0 (0.7)	−0.5 (0.6)	< 0.001	−0.0027; 0.54	n/a
Chicken (Weekly)	How often do you eat chicken?	**0:** 0, 0.5, 1.5, 3.5 times +**1:** 5.5 or 7 times	0.0 (0.2)	0.0 (0.2)	0.06	0.0046; 0.13	0.0045; 0.14
Red meat (Weekly)	How often do you eat red meat?	**0:** 0 or 0.5 times +**1:**1.5 or 3.5 times +**2:**5.5 or 7 times	0.9 (0.5)	1.0 (0.4)	0.11	0.0004; 0.12	0.0045; 0.09
Meat products (Weekly)	How often do you eat meat products?	**0:** 0 or 0.5 times +**1:** 1.5 times +**2:** 3.5, 5.5 or 7 times	1.3 (0.7)	0.7 (0.7)	0.001	0.0018; 0.58	n/a
Bread (Daily)	How many slices of bread do you usually eat each day?	**0:** 0, 0.5, 1 or 2 slices day +**1:** 3 or 4 slices day +**2:** 6 or 8 slices day	0.4 (0.6)	0.1 (0.4)	< 0.001	0.0015; < 0.001	0.0016; < 0.001
Milk (Daily)	How much milk in total do you usually drink each day?	**0:** 0, 0.5, 1 or 2 cups +**1:** 3 or 4 cups +**2:** 5 cups	0.2 (0.5)	0.1 (0.2)	0.18	0.0046; 0.38	n/a
Cheese (Weekly)	How often do you eat cheese?	**0:** 0, 0.5, 1.5, 3.5 or 5.5 times +**1:** 7 times	0.1 (0.3)	0.1 (0.3)	0.003	−0.0053; 0.02	−0.0049; 0.021
Milk products (Weekly)	How often do you eat milk products such as yoghurt, chocolate milk, pudding etc.?	**0:** 0, 0.5, 1.5, 3.5 or 5.5 times +**1:** 7 times	0.1 (0.3)	0.1 (0.3)	< 0.001	−0.0041; 0.05	−0.0037; 0.07
Fruit juice (Weekly)	How much fruit juice do you usually drink?	**0:** 0, 0.5, 2 times +**1:** 5 times +**2:** 10.5, 24.5 or 35 times	0.2 (0.6)	0.1 (0.4)	< 0.001	0.0060; 0.004	0.0059; 0.01
Water (Daily)	How much water do you usually drink each day?	**0:** regardless of consumption	0.0 (0.0)	0.0 (0.0)	0.18	0.0042; 0.18	0.0040; 0.20
Sugar drinks (Weekly)	How much soft drinks, cordials or sports drinks do you usually drink?	**0:** 0 or 0.5 cups +**1:** 2 or 5 cups +**2:** 10.5, 24.5 or 35 cups	0.4 (0.6)	0.2 (0.5)	< 0.001	0.0018; 0.11	0.0020; 0.08
Energy drinks (Weekly)	How much energy drinks do you usually drink?	**0:** 0 or 0.5 cups +**1:** 2 or 5 cups +**2:** 10.5, 24.5 or 35 cups	0.0 (0.2)	0.0 (0.2)	< 0.001	0.0026; 0.67	n/a
Pastas (Weekly)	How often do you eat pasta, rice or noodles?	**0:** 0, 0.5, 2, 5 or 7 times +**1:** 14 times	0.0 (0.1)	0.0 (0.1)	0.46	n/a	n/a
Ice confection (Weekly)	How often do you have ice cream, icy poles or ice blocks?	**0:** 0, 0.5 or 1.5 times +**1:** 3.5 or 5.5 times +**2:** 7 times	0.3 (0.5)	0.1 (0.3)	0.01	0.0047; 0.25	n/a
Fried potato (Weekly)	How often do you eat hot chips, french fries, wedges or fried potatoes?	**0:** 0, 0.5 or 1.5 times +**1:** 3.5 times +**2:** 5.5 or 7 times	0.1 (0.3)	0.0 (0.2)	< 0.001	0.0118; 0.09	0.0140; 0.05
Chips/crisps (Weekly)	How often do you eat potato crisps/chips or other salty snacks?	**0:** 0, 0.5 or 1.5 times +**1:** 3.5 times +**2:** 5.5 or 7 times	0.3 (0.6)	0.1 (0.3)	0.003	−0.0023; 0.60	n/a
Takeaway (Weekly)	How often do you have meals or snacks such as burgers, pizza, chicken or chips?	**0:** 0 or 0.5 times +**1:** 1.5 times +**2:** 3.5, 5.5 or 7 times	0.4 (0.6)	0.2 (0.4)	< 0.001	0.0395; < 0.001	0.0396; < 0.001
Confectionery (Weekly)	How often do you eat confectionery?	**0:** 0, 0.5, 1.5, 3.5 or 5.5 times +**1:** 7 times	0.0 (0.1)	0.0 (0.2)	0.02	−0.0057; 0.02	−0.0056; 0.02
Sweet foods (Weekly)	How often do you eat sweet foods, such as sweet biscuits, cakes or muffins?	**0:** 0, 0.5, 1.5 or 3.5 times +**1:** 5.5 times +**2:** 7 times	0.1 (0.4)	0.1 (0.4)	0.002	−0.0090; < 0.001	−0.0086; 0001

The assigned frequency is listed in bold.
[a] Table excludes 3 food items that examined breakfast, cereals and diet-drinks as their inflammatory value was deemed unclear

inflammatory diet. Table 1 details scoring for each NaSSDA item.

The ***GlycA-derived inflammatory diet score*** was based on an inflammatory biomarker, GlycA, measured in CheckPoint. As there are only adult data to date thus far support on GlycA as a marker of chronic inflammation [22,25], we created the GlycA-derived score for the adults in our cohort and then applied this score to the children data. GlycA was measured from semi-fasting plasma (only collected at Main Assessment centres) by nuclear magnetic resonance (Nightingale, Helsinki, Finland), as previously described [26]. GlycA values were highly positively skewed, so these values were natural-log-transformed to meet assumptions of normality for linear regression analyses. Adult responses to each of the NaSSDA items were individually regressed against their GlycA values. Twenty of 23 univariable associations reached a statistical significance level of $p < 0.20$, [27,28] Fourteen of these 20 NaSSDA items remained associated with GlycA ($p < 0.20$) in a combined multivariable model, and were entered into a final multivariable model (Table 1). Coefficients from this final model were then used to generate an inflammatory diet score for each adult with the following formula:

> "sum (model β for item x * participants' NaSSDA item x response value) + "model constant".

This same formula was used to create the GlycA-derived score for children with NaSSDA data. Higher scores on the GlycA-derived score indicate a more pro-inflammatory diet.

2.3.2. Preclinical cardiovascular phenotypes

Detailed methods for each cardiovascular preclinical phenotypes are described elsewhere [26,29–31], and are briefly described here.

Vascular function: *Pulse wave velocity (PWV)* and *blood pressure* were assessed using the SphygmoCor XCEL device (AtCor medical, Sydney, NSW, Australia). Data were collected after several minutes of rest in the supine position three times and the mean calculated from at least two valid measurements. PWV was measured between the right carotid and femoral artery using tonometry, providing an estimate of large arterial function, with quicker scores representing greater arterial stiffness. *Systolic* and *diastolic blood pressure* and *mean arterial pressure* parameters were measured at the right brachial artery using an appropriately sized cuff.

Vascular structure: *Carotid intima-media thickness* (carotid IMT) was assessed at Main and Mini Assessment Centres via B-mode ultrasound (Vivid-I BT06 with 10 MHz L-RS Vascular probe, GE Healthcare, Chicago, IL, USA) of the right common carotid artery. Subjects were in the supine position and head rotated left 45°. A modified 3-lead ECG mapped the cardiac cycle, and sonographic images of a minimum of three longitudinal loops of 5–10 cardiac cycles were taken 10 mm proximal to the carotid bulb. Image analysis using semi-automated edge-detection software (Carotid Analyser, Medical Imaging Applications, Coraville, IA, USA) calculated carotid IMT far wall mean, with higher scores representing poorer cardiovascular structure. The within-observer and between-observer coefficients of variation were 6.5% and 9.5% for mean carotid IMT values, respectively [32]. Reliability was comparable to other published results [33]. *Retinal vessel calibre* was assessed at Main Assessment centre only via digital photographs of the optic disc in the right eye using CR-DGi fitted with an EOS 60D SLR digital camera (Canon Inc., Tokyo, Japan) by trained technicians. Computer-assisted retinal analysis software IVAN (University of Wisconsin, Madison, WI, USA) measured vessel calibre. The central retinal arteriolar and venular calibre were estimated using the Big-6 method [34], where smaller arterioles and larger venules are deemed less favourable. Inter- and intra-grader intraclass correlation coefficients between graders ranged from 0.76 to 0.99, indicating high reproducibility [31].

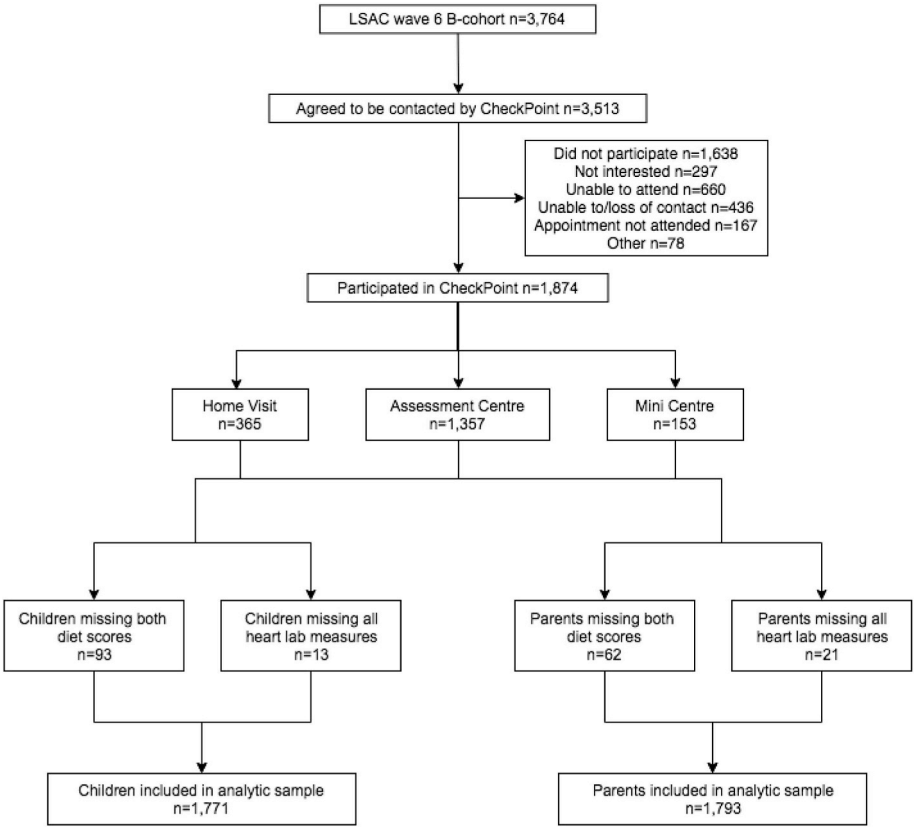

Fig. 1. Recruitment and retention of participants in the Child Health CheckPoint.

2.3.3. Potential confounders

Potential confounders included age, sex, socioeconomic position and (in children only) pubertal status, as well as children's arterial diameter in carotid IMT analyses. Socioeconomic position was calculated from the most recently available parent-reported education, income and occupation data at LSAC's wave 6. Scores were internally standardised to have a mean of 0 and standard deviation (SD) of 1; higher scores represent higher socioeconomic position [35], which has been strongly linked with higher cardiovascular risk [32] and poor diet [36]. Pubertal status was calculated from self-reported ratings of secondary sexual characteristics and growth using the 5-item Pubertal Development Scale [37]. Early puberty is associated with poorer long-term health outcomes [38]. For carotid IMT analyses, we also adjusted for children's minimal vessel diameter to account for differences in body and vessel size during growth.

2.3.4. Sensitivity analysis variables

Body mass index (BMI; weight/height [2]) for adults and children (z-score, standardised for age and sex, calculated from US Centres for Disease Control (CDC) reference values [39]) were included in a sensitivity analysis. BMI is strongly linked to both poor dietary patterns and cardiovascular risk [40], so could plausibly lie on the causal pathway between inflammatory diets and preclinical cardiovascular phenotypes. Further sensitivity analyses 1) excluded adults who self-reported current blood pressure and/or cholesterol lowering medication, and those with a prior heart condition and 2) additionally adjusted for smoking or passive smoke exposure over the past decade. We created a binary variable of “ever exposed to passive smoke” for children or “smoked in the previous decade” for adults from the LSAC questionnaire data. This variable was positive if in any of the six LSAC waves prior to CheckPoint (ages 0–1 to 10–11 years) the answer was > 0.

2.4. Statistical analysis

All data were analysed using Stata version 14.2. For this cross-sectional study, we conceptualised the two inflammatory diet scores as “risk factors” and preclinical cardiovascular phenotypes as “outcomes”. Participants were included if they completed the NaSSDA and had at least one cardiovascular measure.

We tested linear regression assumptions when fitting models. Unadjusted and adjusted linear regression models estimated associations of inflammatory diet scores with preclinical cardiovascular phenotypes. Both inflammatory diet scores were internally standardised to have a mean of zero and SD of one. Adjusted models included age, sex, socioeconomic position and pubertal status (children only). Child carotid IMT analyses were additionally adjusted for minimal vessel diameter. We also performed three sensitivity analyses: 1) including BMI/BMI z-score, 2) excluding adults taking blood pressure or cholesterol lowering medication and/or with a prior heart condition, and 3) including smoking/passive smoking exposure.

Finally, interaction tests were performed to assess for differential sex associations, which may be of particular relevance in adults given sex-specific differences in cardiovascular risk [41].

3. Results

3.1. Sample characteristics

Of the 3764 eligible families, 1875 (47% of LSAC wave 6) child-parent pairs participated in CheckPoint. Dietary and preclinical cardiovascular phenotypes were available for 1771 children and 1793 adults. Fig. 1 shows the flow through the study, including the numbers with each of the vascular function and structure measures.

On average, children and adults were aged 11.5 (SD: 0.5) and 43.7 (SD: 5.2) years, respectively, and adults were predominantly female (87.7%, children 49.5% female; Supp Table 1)). The socioeconomic position of the analytic sample was 0.2 SD units higher than the national Australian average, indicating a slightly more advantaged population. Most children were in early to mid-puberty (77%), and children and adults had similar levels of overweight (15% and 33%) and obesity (9% and 29%) to current Australian norms [42].

A total of 8% of adults self-reported having a heart condition, high blood pressure and/or taking high cholesterol or blood pressure medication. 15% of adults had smoked and 16% of children had been exposed to passive smoke in the household over the preceding decade. Children and adult's average literature-derived diet scores were 2.50 (SD 3.04; range −5 to 14) and 0.77 (SD 2.46; −5 to 13), and their GlycA-derived scores were 0.065 (SD 0.063; −0.14 to 0.42) and 0.025 (SD 0.057; −0.15 to 0.35), respectively.

Table 2
Child regression coefficients estimating variation in preclinical CV phenotypes for each SD unit higher in inflammatory diet scores.

11-12 year-olds CV intermediate phenotypes	Mean (SD)	Adjusted for age, sex, SEP and puberty		Additionally adjusted for BMI z-score	
		β (95% CI)	*p*	β (95% CI)	*p*
Literature-derived inflammatory diet score (SD units)					
Carotid intima-media thickness max (μm)	581 (46)	[a]-0.4 (−2.9 to 2.1)	0.76	[a]-0.3 (−2.8 to 2.3)	0.84
Retinal arteriolar calibre (μm)	159.0 (11.9)	0.34 (−0.35 to 1.03)	0.33	0.26 (−0.42 to 0.94)	0.45
Retinal venular calibre (μm)	230.5 (16.5)	0.21 (−0.75 to 1.17)	0.67	0.22 (−0.74 to 1.18)	0.65
Pulse wave velocity (m/s)	4.46 (0.57)	−0.01 (−0.04 to 0.02)	0.35	−0.01 (−0.03 to 0.02)	0.64
Systolic blood pressure (mmHg)	108.2 (7.9)	−0.4 (−0.8 to 0.0)	0.07	−0.1 (−0.5 to 0.3)	0.51
Diastolic blood pressure (mmHg)	62.4 (5.7)	−0.1 (−0.3 to 0.3)	0.76	0.02 (−0.3 to 0.3)	0.90
Mean arterial pressure (mmHg)	76.1 (6.3)	−0.2 (−0.5 to 0.2)	0.38	−0.02 (−0.3 to 0.3)	0.90
GlycA-derived inflammatory diet score (SD units)					
Carotid intima-media thickness max (μm)	As above	[a]0.9 (−1.6 to 3.4)	0.48	[a]0.8 (−1.7 to 3.3)	0.53
Retinal arteriolar calibre (μm)		0.42 (−0.27 to 1.11)	0.23	0.44 (−0.24 to 1.12)	0.21
Retinal venular calibre (μm)		−0.29 (−0.99 to 0.93)	0.95	−0.03 (−0.99 to 0.93)	0.95
Pulse wave velocity (m/s)		−0.00 (−0.03 to 0.03)	0.95	−0.00 (−0.03 to 0.02)	0.82
Systolic blood pressure (mmHg)		−0.1 (−0.5 to 0.4)	0.80	−0.1 (−0.4 to 0.3)	0.78
Diastolic blood pressure (mmHg)		0.0 (−0.3 to 0.3)	0.94	0.0 (−0.3 to 0.3)	0.94
Mean arterial pressure (mmHg)		0.0 (−0.3 to 0.4)	0.80	0.0 (−0.3 to 0.4)	0.79

[a]Further adjusted for minimal vessel diameter.

CV: cardiovascular; β: estimated regression coefficient; SEP: socioeconomic position; BMI: body mass index; CI: confidence interval; SD: standard deviation; μm: micrometres; m/s: metres per second; mmHg: millimetres of mercury.

3.2. Associations of inflammatory diet scores with preclinical cardiovascular phenotypes

Given their similarity to the unadjusted models for both children and middle-aged adults, we present adjusted results only.

In children, there was little evidence to suggest that either inflammatory diet score was associated with cardiovascular structural or functional phenotypes (Table 2).

In adults, there was modest statistical evidence that both inflammatory diet scores were associated with worse cardiovascular function (Table 3). For example, per SD higher GlycA-derived diet score, pulse wave velocity was 0.17 m/s faster (95% confidence interval (CI) 0.11 to 0.22), systolic and diastolic blood pressure was 1.65 (95% CI 1.02 to 2.28) and 1.36 (95% CI 0.92 to 1.80) mmHg higher respectively, mean arterial pressure was 1.85 mmHg (95% 1.34 to 2.37) higher, and retinal arteriolar calibre was 1.29 μm narrower (−2.10 to −0.49). These associations were small, representing only 0.1–0.2 SD effect sizes for the cardiovascular function measures. Results were similar, although, smaller for literature-derived inflammatory diet scores. When BMI was added to the models, these associations were attenuated partially for the GlycA-derived and fully for the literature-derived inflammatory diet scores.

There was little evidence to suggest that inflammatory diet scores were associated with carotid IMT or retinal venular calibre in adults. However, higher inflammation on both diet scores was consistently associated with narrower retinal arteriolar calibre per SD higher GlycA-derived and literature-derived inflammatory diet score, arteriolar calibre narrowed by 1.29 μm (95% CI -2.10 to −0.49) and 1.20 μm (95% CI -2.01 to −0.39) respectively. Again, these effect sizes were small (less than 0.1 SD). When BMI was added these associations attenuated, and a new association between the GlycA-derived inflammatory score and carotid IMT became evident.

Excluding the 8% of adults taking blood pressure or cholesterol lowering medication, and/or with a prior heart condition, and including smoking exposure made little difference to the results. Similarly, interaction tests did not provide statistical evidence to suggest that associations differed by sex in children or adults (data not shown).

4. Discussion

4.1. Principal findings

In children aged 11–12 years, there was little evidence that an inflammatory diet was associated with preclinical phenotypes of either vascular function or structure. By mid-life, a pro-inflammatory diet was consistently associated with adverse variations in vascular function (all measures) and microvascular structure (i.e. retinal arteriolar calibre). These associations were small, but are likely to worsen with cumulative exposure to an inflammatory diet. All associations were stronger for the GlycA-derived than the literature-derived inflammatory diet score, and attenuated towards the null when BMI was added to the models.

4.2. Interpretation in relation to previous research

To our knowledge, no studies have thoroughly examined the relationship between inflammatory diet scores and multiple preclinical cardiovascular phenotypes in a mid-life population cohort. Our observed associations in mid-life adults are in the same direction as commonly reported associations between the DII and single cardiovascular risk factors (e.g. blood pressure) and disease in mid-late adulthood [2,4,11–13], but our population was younger and the magnitude of the associations were notably smaller. In keeping with our results, most [4,12,13] but not all [9] previous studies of community/population cohorts have shown that in comparison to mid-life adults in the lowest DII category, those in the highest category have slightly higher blood pressure. Meta-analytic results (n = 534,906) from both clinical trials and epidemiological studies demonstrate that adherence to the anti-inflammatory Mediterranean dietary pattern has beneficial effects on blood pressure and other components of the metabolic syndrome (waist circumference and glucose levels) [43]. In population-based cohorts, anti-inflammatory dietary patterns have also demonstrated associations with common carotid IMT and adiposity, but not with blood pressure [44].

Most studies that examine associations of dietary inflammation with PWV or carotid IMT in adults tend to focus on high risk populations, rather than community and/or population samples, making direct comparison with our results challenging. For example, adherence to an anti-inflammatory diet (Mediterranean or Dietary Approaches to Stop Hypertension) in both randomised and non-randomised controlled trials has been shown to improve carotid IMT, PWV and BP among clinical samples of adults with a high risk cardiovascular disease [45–47], but whether these associations hold in population-based samples is unknown.

There are no comparable population-based data for children. One small community-based study demonstrated that although children's higher DII scores were associated with higher waist-to-height-ratio, there were no associations with blood pressure or heart rate [16].

Table 3
Adult regression coefficients estimating variation in preclinical CV phenotypes for each SD unit higher in inflammatory diet scores.

Adults (mean age: 44 yrs) CV intermediate phenotypes	Mean (SD)	Adjusted for age, sex and SEP		Additionally adjusted for BMI	
		β (95% CI)	*p*	β (95% CI)	*p*
Literature-derived inflammatory diet score (SD units)					
Carotid intima-media thickness max (μm)	663.0 (97.2)	−0.3 (−5.2 to 4.6)	0.91	−3.5 (−8.4 to 1.4)	0.16
Retinal arteriolar calibre (μm)	151.24 (13.86)	−1.20 (−2.01 to −0.39)	0.004	−0.80 (−1.62 to 0.02)	0.06
Retinal venular calibre (μm)	219.03 (18.52)	−0.01 (−1.10 to 1.07)	0.98	−0.03 (−1.1 to 1.1)	0.96
Pulse wave velocity (m/s)	6.86 (1.13)	0.09 (0.04 to 0.15)	0.001	0.03 (−0.02 to 0.09)	0.21
Systolic blood pressure (mmHg)	119.4 (12.6)	0.6 (−0.1 to 1.2)	0.08	−0.3 (−0.8 to 0.3)	0.34
Diastolic blood pressure (mmHg)	73.0 (8.6)	0.5 (0.1 to 1.0)	0.02	0.2 (−0.3 to 0.6)	0.48
Mean arterial pressure (mmHg)	86.5 (10.2)	0.7 (0.2 to 1.2)	0.01	0.0 (−0.4 to 0.6)	0.71
GlycA-derived inflammatory diet score (SD units)					
Carotid intima-media thickness max (μm)	As above	0.1 (−4.8 to 5.0)	0.97	−5.1 (−10.0 to −0.1)	0.04
Retinal arteriolar calibre (μm)		−1.29 (−2.10 to −0.49)	0.002	−0.70 (−1.53 to 0.14)	0.10
Retinal venular calibre (μm)		0.62 (−0.46 to 1.70)	0.26	0.64 (−0.49 to 1.77)	0.27
Pulse wave velocity (m/s)		0.17 (0.11 to 0.22)	< 0.001	0.07 (0.02 to 0.12)	0.01
Systolic blood pressure (mmHg)		1.7 (1.0 to 2.3)	< 0.001	0.3 (−0.3 to 0.8)	0.39
Diastolic blood pressure (mmHg)		1.4 (0.9 to 1.8)	< 0.001	0.7 (0.3 to 1.2)	0.001
Mean arterial pressure (mmHg)		1.9 (1.3 to 2.4)	< 0.001	0.9 (0.4 to 1.4)	< 0.001

Previous studies in childhood have focused on adherence to anti-inflammatory diets – neglecting possible contributions from pro-inflammatory foods – in participants with increased cardiovascular risk. In a small uncontrolled 12-month trial (n = 36) of pre-pubescent children with hypercholesterolaemia, carotid IMT fell with a Mediterranean diet intervention [48]. However, in 237 children with a recent diagnosis of type I diabetes, a dietary pattern characterised by pro-inflammatory foods was not associated with higher PWV [49].

We attempted to extend previous studies that use the DII by creating both a literature-derived and GlycA-derived inflammatory diet score. For example, the DII is essentially a literature-derived score that is a proxy for a 'healthy' diet and the biological basis for it as a measure of 'diet-related inflammation' is mixed [9,50]. We overcame this limitation by specifically examining the association of each component of our food frequency questionnaire with GlycA (i.e. GlycA-derived score).

4.3. Implications

Diet is one of the cornerstone lifestyle behaviours that can modulate the inflammatory process. The lack of associations of either inflammatory score with children's vascular phenotypes suggest that the negative effects associated with an inflammatory diet are cumulative and manifest only later in the life course. In keeping with this cumulative life course hypothesis, we showed associations of two inflammatory diet scores with vascular function and retinal arteriolar calibre in mid-life adults (the children's parents). However, the magnitude of associations was small and attenuated following adjustment for BMI, suggesting that the association of pro-inflammatory diets with cardiovascular health in mid-life adults may be mediated in part via BMI – itself an inflammatory condition.

Nevertheless, although the magnitude of these results is unlikely to have immediate-clinical relevance, these small gradients within the normal range may be of importance to population health. Furthermore, if an inflammatory diet has small detrimental effects on vascular phenotypes in a healthy population-based mid-life cohort, more marked adverse changes will likely become evident with age due to ongoing cumulative exposure and additional risk factors.

Children had slightly higher inflammatory diets, compared to adults, which could reflect adult's greater social desirability to be seen to be 'healthy' eaters. However, it may also reflect real differences given children's abundant access to inflammatory food (e.g. unhealthy snacks and children's restaurant menus, which often contain sweet biscuits and chips respectively). Our data may inform preventive work in both clinical and public health settings. Results imply that the damage inflicted by an inflammatory diet presents post-childhood. Therefore, modifying dietary habits holds the potential to protect against adulthood cardiovascular damage. However, further longitudinal and intervention research is needed to pinpoint the best window for preventive work and its efficacy.

4.4. Limitations

We recognise some limitations of this study. Our participants were, on average, slightly more advantaged socioeconomically and homogeneous than the general Australian population, which may limit the generalisability to very disadvantaged people. Secondly, only 12% of the adults were male. Thirdly, in comparison to the comprehensive and widely used DII or a nutrient intake survey, the NaSSDA's brevity means that our dietary scores do not capture all dietary or nutrient components (e.g. salt intake, alcohol and coffee) that may influence inflammation. The beneficial trade-offs for such brevity are the large national cross-generational samples and depth of cardiovascular measures. In addition, the self-reported nature of diet data may be subject to inaccuracies and recall biases [51,52]. Nonetheless, previous studies have shown the fruit and vegetable items of the NaSSDA demonstrate good validity when compared to adult reported 24-h recall [21]. Fourthly, although the GlycA-derived score used a new novel biomarker of inflammation, the child GlycA-derived score was calculated using the coefficients from the adult GlycA analysis, as there are virtually no data on GlycA in children. Diet and other factors may affect GlycA differently through additional as yet unknown pathways at younger ages. Fifthly, given that we used an inflammatory biomarker to derive one of the inflammatory diet scores, it is possible that we also captured any direct association between inflammation (i.e. GlycA) and preclinical cardiovascular phenotypes. However, the highly consistent results for the literature-derived score suggests we have identified associations related to inflammatory diet and provides confidence in our results and interpretation. Lastly, due to the cross-sectional nature of the study, temporal or causal relationships cannot be addressed.

4.5. Strengths

Strengths include the large population-derived samples spanning a large geographical area, the inclusion of individuals at two important life stages (late childhood and mid-life), and the breadth and depth of objective measurements of preclinical cardiovascular phenotypes – both structural and functional. All measurements were obtained using trained technicians and qualified graders. The NaSSDA questionnaire, although brief, better reflects habitual dietary habits than food diaries or diet recalls [53]. Finally, the direction of our obtained effects is in the expected direction and is reinforced by replication between two related, but unique, inflammatory diet scores.

5. Conclusions

A pro-inflammatory diet was consistently associated with small adverse variations in preclinical cardiovascular phenotypes of function and microvascular structure in mid-life adults, but not in 11–12 year-olds. These findings support a life course paradigm, where negative impacts of a pro-inflammatory diet on preclinical cardiovascular phenotypes may accumulate so they are detectable by mid-life, and presumably continue to accrue thereafter.

Conflict of interest

The authors declared they do not have anything to disclose regarding conflict of interest with respect to this manuscript.

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findings and views reported in this paper are those of the author and should not be attributed to DSS, AIFS or the ABS.

Author contributions

Dr Davis initiated this work as part of his student project, made substantial contributions to the conception and design of the study, conducted the analyses, interpreted the data, drafted the article, and revised it critically based on co-author feedback.

Dr Liu contributed to the study design, analysis of the data, interpretation of data, and revised the article critically for important intellectual content.

Dr Kerr co-supervised Dr Davis, made substantial contributions to the conception and design of the study, analysis of the data, interpretation of data, and revised the article critically for important intellectual content.

Professor Wake is the Chief Investigator of The Child Health CheckPoint study, she contributed to the study design, interpretation of data, and revised the article critically for important intellectual content.

Dr Grobler contributed to the study design, provided statistical advice, interpretation of data, and revised the article critically for important intellectual content.

Professors Juonala and Baur are Investigators on the Child Health CheckPoint study, they made contributions to the study design, interpretation of data, and revised the article critically for important intellectual content.

Ms Liu contributed to the study design, interpretation of data, and revised the article critically for important intellectual content.

Professor Burgner primary supervised Dr Davis and is an Investigator of The Child Health CheckPoint study. He made substantial contributions to the conception and design of the study, interpretation of data, and revised the article critically for important intellectual content.

Dr Lycett co-supervised Dr Davis, made substantial contributions to the conception and design of the study, analysis of the data, interpretation of data, revised the article critically for important intellectual content.

All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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Appendix A. Supplementary data

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