

Childhood dietary trajectories and adolescent cardiovascular phenotypes: Australian community-based longitudinal study

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Abstract

Objective: With the intention to inform future public health initiatives, we aimed to determine the extent to which typical childhood dietary trajectories predict adolescent cardiovascular phenotypes.

Design: Longitudinal study. Exposure was determined by a 4 d food diary repeated over eight waves (ages 4–15 years), coded by Australian Dietary Guidelines and summed into a continuous diet score (0–14). Outcomes were adolescent (Wave 8, age 15 years) blood pressure, resting heart rate, pulse wave velocity, carotid intima-media thickness, retinal arteriole-to-venule ratio. Latent class analysis identified 'typical' dietary trajectories from childhood to adolescence. Adjusted linear regression models assessed relationships between trajectories and cardiovascular outcomes, adjusted for *a priori* potential confounders.

Setting: Community sample, Melbourne, Australia.

Subjects: Children (*n* 188) followed from age 4 to 15 years.

Results: Four dietary trajectories were identified: unhealthy (8%); moderately unhealthy (25%); moderately healthy (46%); healthy (21%). There was little evidence that vascular phenotypes associated with the trajectories. However, resting heart rate (beats/min) increased (β ; 95% CI) across the healthy (reference), moderately healthy (4.1; -0.6, 8.9; $P=0.08$), moderately unhealthy (4.5; -0.7, 9.7; $P=0.09$) and unhealthy (10.5; 2.9, 18.0; $P=0.01$) trajectories.

Conclusions: Decade-long dietary trajectories did not appear to influence macro- or microvascular structure or stiffness by mid-adolescence, but were associated with resting heart rate, suggesting an early-life window for prevention. Larger studies are needed to confirm these findings, the threshold of diet quality associated with these physiological changes and whether functional changes in heart rate are followed by phenotypic change.

Keywords
Diet and nutrition
Adolescent nutritional physiology
Child
Cardiovascular system
Heart rate

Atherosclerosis, the infiltration of inflammatory cells and lipid into arterial walls, begins in childhood and causes later CVD⁽¹⁾. The most prevalent risk factors for CVD are modifiable⁽²⁾. Because unhealthy diets cause inflammation to blood vessels and internal organs⁽³⁾, there is a strong link between diet quality and cardiovascular health in adults⁽⁴⁾. It is estimated that following dietary guidelines throughout adulthood could reduce the population-level burden of CVD by at least 20%⁽⁵⁾.

Although dietary intervention in adulthood can mitigate CVD risk⁽⁶⁾, greater benefits might be obtained if healthy diets were initiated and sustained from childhood^(7–9). Unfortunately, many children do not consume the healthy

diets^(10,11) shown to reduce CVD in adults⁽⁶⁾. Therefore, various intervention studies have been established among children. The Special Turku Coronary Risk Factor Intervention Project (STRIP) randomised more than 1000 infants to an unrestricted diet (control) or to repeated dietary counselling facilitating a diet continually low in saturated fats and cholesterol⁽¹²⁾. Throughout late childhood and adolescence, those in the intervention group had healthier body weight, blood pressure, total cholesterol and LDL-cholesterol^(13–16). By adolescence, those in the intervention group had better American Heart Association cardiovascular health scores, which were associated with less adverse cardiovascular intermediate phenotypes, namely

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reduced aortic intima-media thickness and increased aortic elasticity⁽¹³⁾.

While randomised trials are paramount, continued observational research into children's routine diet and cardiovascular phenotypes is still needed⁽¹⁷⁾ to pinpoint the level of childhood dietary quality necessary to trigger cardiovascular change and to better understand the best window during childhood for optimal intervention. Observational studies have shown robust associations of childhood and adolescent consumption of foods and beverages high in sugar/fat or low in protein/fibre with poor CVD risk scores, blood pressure, TAG and total cholesterol levels or retinal microcirculatory health^(18–26). Similarly, increasing consumption of sugary beverages or decreasing consumption of dairy, fruit or vegetables throughout adolescence has been associated with increased cardio-metabolic risk factors in late adolescence^(27–29).

However, because foods are not consumed in isolation, it is more meaningful to study children's whole diet, rather than studying links between individual foods and CVD risk. Questionnaire-derived dietary scores are often based on dietary guidelines or other *a priori* principles and broadly capture 'whole' diet quality. Some dietary quality scores predict CVD events in adults and improve the accuracy of CVD risk prediction models^(30,31). However, this approach has not been widely applied to children⁽³²⁾. Nevertheless, a handful of cross-sectional studies do demonstrate that superior diet scores inversely associate with childhood/adolescent blood pressure, augmentation index or composite CVD risk score^(33–35). Only one group has investigated the association between a score based on the Australian Dietary Guidelines and cardiometabolic risk factors among children or adolescents; worsening diet scores (between 14 and 17 years of age) were associated with increasing heart rate, TAG, insulin and insulin resistance levels⁽³⁶⁾.

The method of trajectory analyses, which identifies groups of participants whose dietary scores closely correspond with one another over time, is emerging in this field^(37,38). This novel approach to capture children's dietary change is uncommon because few studies have the required measures of diet repeated over a sustained period of childhood. However, public health researchers recognise the value in tracking multiple dietary scores/patterns within childhood^(39–43) and using these data to predict later outcomes^(7,44–47). From a public health perspective, longitudinal trajectory analysis of childhood diet would help to improve understanding of population-level health outcomes⁽⁴⁸⁾, including adolescent cardiovascular health.

The physiological development of cardiovascular damage begins in childhood⁽¹⁾. However, no studies have yet investigated dietary trajectories across multiple time points from pre-school to adolescence and their association with a comprehensive battery of measured cardiovascular phenotypes in this age group. This could provide insights into typical life-course dietary pathways to lifelong

cardiovascular health. For example, it might be that two children follow the same unhealthy trajectory throughout early and middle childhood, but that one becomes much healthier during adolescence. It is of public health interest to understand if this child has better heart health or whether perhaps the healthy change has occurred too late. This could clarify when to target dietary intervention most effectively for better cardiovascular health. In contrast, important and clinically valuable information would be lost by relying on children's average dietary habits over a decade.

In a community-based longitudinal study between 2002 and 2014, we repeatedly measured children's diet at eight time points using the same 4d food diary, and then empirically derived dietary trajectories. In 2014, when participants were adolescent, we measured their intermediate cardiovascular risk phenotypes. Specifically, in the present exploratory study of 188 adolescents we aimed to:

1. empirically derive typical dietary trajectories from age 4 to 15 years; and
2. explore which, if any, trajectories predict better vascular phenotypes at age 15 years.

Methods

Participants and procedures

The Parent Education and Support (PEAS) study is a prospective community-based birth cohort study conducted in Melbourne, Australia (population 3.3 million at time of initial recruitment). Recruitment and follow-up are detailed elsewhere^(49–51). From thirty-one local government areas, three areas were selected for recruitment to provide a broad range of sociodemographic characteristics. Families were recruited from one low, one medium and one highly advantaged local government area (mean annual birth rate per local government area = 1350). Maternal and Child Health Nurses, who provide universal care to all Melbourne children aged 0–5 years, invited parents of all first-born newborns within the three recruitment areas to participate in PEAS. This initial recruitment took place between June 1998 and February 2000. PEAS originally aimed to test the efficacy of brief evidence-based support for common parenting issues during the child's first two years⁽⁵⁰⁾. However, because child health outcomes were similar between control and intervention arms, groups were combined into a single cohort.

The renamed PEAS Kids Growth Study followed these children from age 4 years. It collected data on children's growth and development every 6 months (see Fig. 1, adapted from Hanvey *et al.*⁽⁵¹⁾) from age 4 (current study Wave 1, *n* 402) to 6.5 years (current study Wave 6, *n* 317), then again at age 10 (current study Wave 7, *n* 261) and 15 years (current study Wave 8, *n* 203). All waves included anthropometric measurement and questionnaires, which

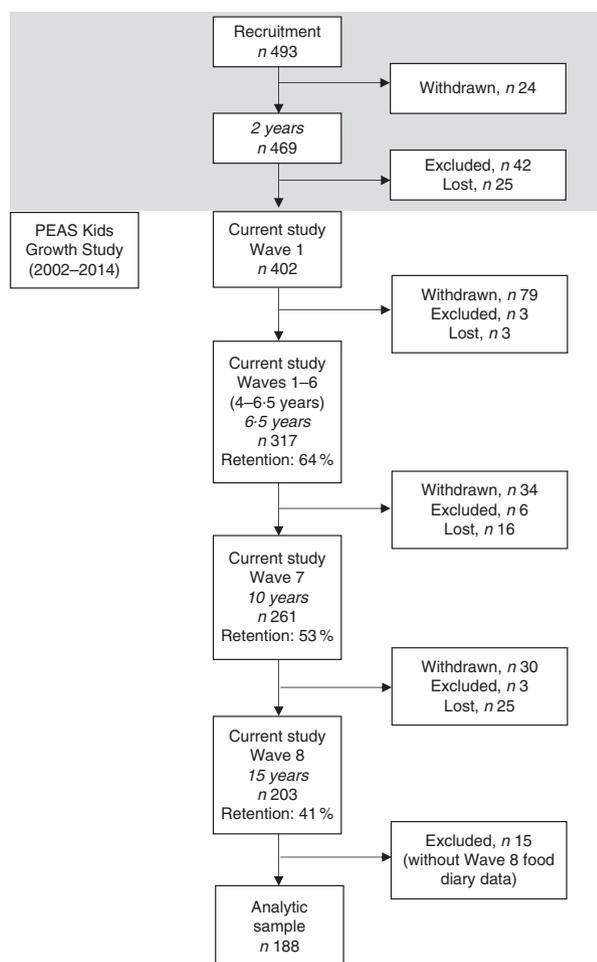


Fig. 1 Participant retention in the Parent Education and Support (PEAS) Kids Growth study. The grey shading refers to the original PEAS Study (in which recruitment took place) before it was renamed the PEAS Kids Growth Study

included the 4 d food diary. At Wave 8, researchers collected height, weight and cardiovascular assessments at Melbourne's Royal Children's Hospital or at home between February and October 2014. Parents and adolescents provided written informed consent. The Royal Children's Hospital Ethics Committee approved all protocols (approval number 28153).

Measures

Food diary (Waves 1–8, age 4–15 years)

In Waves 1–6 parents completed the food diaries for their children, at Wave 7 children had the option to complete the food diary themselves, and at Wave 8 children completed the diary by themselves. Because children's dietary habits differ between weekend and weekdays⁽⁵²⁾, the 4 d food diary asked participants to record their consumption (not eaten = 0, eaten once = 1, eaten twice or more = 2) of twelve common food items (e.g. fruit, milk, water, confectionery) on two weekdays and two weekend days. If a

participant missed a whole diary day, data were imputed from the completed weekend or weekday. Based upon a previously published score⁽³⁷⁾, for each individual day we grouped the twelve items into seven common Australian food groups (fruit, vegetables, water, fatty foods, sugary foods, milk/milk products, sweetened drinks) which capture the majority of components relevant to emerging CVD (e.g. saturated fat, sugar, salt, fibre)⁽⁶⁾. We used the 2013 Australian Dietary Guidelines⁽⁵³⁾ to assign each category a score of 0, 1 or 2. A score of 0 was assigned if participants did not meet the particular dietary guideline, 1 was assigned if the guideline was partially met and a score of 2 was given if the guideline was met. Fruit, vegetables, water and milk products or alternatives were positively coded and fatty foods, sugary foods and sweetened drinks were reverse/negatively coded. Participants' resulting 0–2 scores for each of the seven categories were summed to form a 0–14 score, with 14 indicating the highest possible dietary quality. Therefore, participants had four (two weekday, two weekend) 0–14-point diet scores that were then averaged to generate one weekday and one weekend score. To produce an overall diet quality score representative of participants' typical weeks, we multiplied this average weekday habit score by 5 and average weekend habit score by 2, then computed the average score (0–14).

Vascular measures (Wave 8, age 15 years)

We collected data on pulse wave velocity, blood pressure, resting heart rate, carotid intima-media thickness and retinal arteriole-to-venule ratio. Collectively, these measures indicate the function and structure of the cardiovascular system and are associated with emerging cardiovascular health^(54–58).

Vascular function. Resting blood pressure was measured supine after a 2 min rest using the 'pulse wave analysis' setting of the SphygmoCor XCEL (AtCor Medical, NSW, Australia)⁽⁵⁹⁾. A brachial cuff was fitted around the upper right arm. Medians of three measures (with intervals of 1 min between) were calculated for resting heart rate (beats/min, bpm) and brachial systolic and diastolic blood pressure (mmHg).

Carotid-femoral pulse wave velocity, an indicator of arterial stiffness, was measured to the nearest 0.1 m/s using a thigh cuff and tonometer. Trained research assistants measured the distance from participants' carotid pulse to sternal notch, femoral pulse to top of the thigh cuff and sternal notch to top of the thigh cuff. Pulse transit distance in metres was calculated by subtracting the first and second measure from the third measure. Assistants then used a tonometer to record the waveform of participants' carotid pulse, while femoral waveform was recorded by the inflated thigh cuff. Once ten pulse waves were recorded, pulse transit time in seconds was determined. Pulse wave velocity was calculated as

distance (m)/time (s). Higher pulse wave velocity values indicate stiffer arteries (i.e. worse vascular function).

Vascular structure. Vascular structural measures were captured only in participants who could attend the centre-based visit (n 103), as portability limitations meant that equipment was not compatible with home visits. For carotid intima-media thickness, we acquired ultrasound images of the carotid artery (Vivid i ; General Electronics Healthcare, USA) at end diastole with ECG gating, using a linear probe with a frequency of at least 8 MHz. Images of the right carotid artery were optimised to visualise the intima-media complex of the anterior and posterior walls of the right common carotid artery 1 cm proximal to the carotid bulb. Cine loops of at least five cardiac cycles were recorded and saved for later analysis by Heart Research at Murdoch Children's Research Institute using semi-automated edge-detection software (Carotid AnalyzerTM). Increased carotid intima-media thickness

indicates a thicker intima-media, an indicator of pre-clinical atherosclerosis.

Digital retinal photography was conducted with a non-mydratric retinal camera CR-DGi with an EDS 300 SLR camera back using DH client software. We took photos of the right and left eye macula and retina. Saved images of the right eye retina were analysed for vessel calibres. Central retinal arteriole equivalent and central retinal venule equivalent were calculated using the 'big 6' methodology⁽²³⁾. We calculated retinal arteriole-to-venule ratio by dividing the central retinal arteriole equivalent by the central retinal venule equivalent (converted to %). A lower retinal arteriole-to-venule ratio indicates increased venule calibre, decreased arteriole calibre, or both increased venule and decreased arteriole calibres combined. These microvascular parameters have been associated with increased cardiovascular risk in adults⁽⁶⁰⁾.

Table 1 Description of measures used in the PEAS Kids Growth Study, Melbourne, Australia, 2002–2014

Construct	Measure	Wave	Additional information
Maternal education	Maternal self-reported	1	Mothers completed a questionnaire which asked about their highest level of completed education: did not complete Year 12, completed Year 12, degree educated. In Australia, Year 12 is the final year of high school
Neighbourhood-level SEP	SEIFA index	1	Residential postcode was converted into SEIFA-IRSD score (national mean 1000 (SD 100)) from the previous Australian Bureau of Statistics 5-yearly census ⁽⁶¹⁾ . A lower score indicates more disadvantage
Adolescent pubertal status	Self-reported	8	As part of the questionnaire, participants completed the Pubertal Development Scale ⁽⁶²⁾ . Questions were dependent on participants' sex and included items such as 'Would you say that your growth in height (growth spurt) has: not started, barely started, definitely started, seems complete?' Responses were categorised into: pre-, early, mid-, late or postpubertal
Adolescent physical activity	Accelerometry	8	Accelerometers (model GT3X; ActiGraph LLC, Pensacola, FL, USA) were used to objectively measure physical activity. The participant was fitted with a waist-worn, lightweight accelerometer during his/her appointment (Day 0) and asked to wear it continuously for seven days. Non-wear times were recorded on an accompanying activity log card. Movement was recorded in 60 s epochs. Once accelerometers were returned via post, data were downloaded using the accompanying ActiLife program (version 6). Data from Day 0 and any day after the seven-day wear period were discarded. The minimum data required for inclusion were ≥ 4 'valid' days of ≥ 10 h of non-missing data between 06.00 and 23.00 hours. Missing data were defined as any periods of ≥ 60 min of consecutive '0' counts. Average percentage time spent in moderate-to-vigorous physical activity (> 2296 counts/min) was defined using Evenson's thresholds ⁽⁶³⁾
Adolescent BMI Z-score	Researcher measured participant's height and weight	8	Anthropometry-trained research assistants measured height (portable rigid stadiometer model IP0955; Invicta, Leicester, UK) as the mean of two measurements, or the median of three measurements if the first two differed by > 0.5 cm. They measured weight with a calibrated digital scale (TI-THD646; Tanita, Tokyo, Japan) with the participant wearing light clothing, without shoes or socks. BMI was calculated as $[\text{weight (kg)}] / [\text{height (m)}]^2$. We used CDC reference values to calculate adolescents' BMI Z-score ⁽⁶⁴⁾ , using the Stata 'zanthro' function ⁽⁶⁵⁾

PEAS, Parent Education and Support; SEP, socio-economic position; SEIFA, Socio-Economic Indexes for Areas; IRSD, Index of Relative Socio-economic Disadvantage; CDC, US Centers for Disease Control and Prevention.

Other measures

At Waves 1 and 8, we also measured maternal education and socio-economic position, and children's age, sex, pubertal development, time spent in moderate-to-vigorous physical activity, height and weight (Table 1)^(61–65).

Analysis

We required that participants had Wave 8 (age 15 years) food diary data to be included in trajectory analyses (*n* 188). On average, these participants had complete diary data for seven of the eight waves. For study aim 1, we conducted latent class analyses using Mplus 5.1⁽⁶⁶⁾. This analysis identifies and allocates participants to their most likely dietary trajectory based on the similarity between participants' food diary measurements over time. We ascertained trajectory categories using up to eight of the participants' diet scores from age 4 to 15 years (i.e. Waves 1–8). We fitted models with two, three, four and five trajectories to the data, then selected the best model as judged from class size and model fit statistics. That is, *a priori* we intended to select the model that had an entropy

value greater than 0.8 and at least 5% of the sample in each class/trajectory, balanced with the lowest possible Bayesian information criterion (BIC), Akaike information criterion (AIC) and adjusted BIC values^(67,68). In addition, model fit was evaluated with *P* values obtained from Vuong–Lo–Mendell–Rubin likelihood ratio tests of whether adding an additional trajectory category improved model fit⁽⁶⁶⁾.

For analyses pertaining to study aim 2, we used these dietary trajectories as a categorical predictor in adjusted linear regression analyses (using Stata/IC 14.1). To be included in this analytic sample, we required participants to have completed a cardiovascular assessment at Wave 8 (*n* 188). Because of their potential gradient with both the exposure and outcomes^(8,69–71), *a priori* identified confounders were Wave 1 maternal education and socio-economic position, and Wave 8 child age, sex, pubertal development and percentage of time spent in moderate-to-vigorous physical activity. In our final model, we further adjusted for Wave 8 BMI *Z*-score, to determine the strength of association between the dietary trajectories and adolescent cardiovascular phenotypes regardless of adolescent body mass.

Table 2 Sample characteristics of the children from the PEAS Kids Growth Study, Melbourne, Australia, 2002–2014*

	<i>n</i>	Total (<i>n</i> 188)			Range
		Mean	%	SD	
Sociodemographic characteristics at outcome					
Age (years)	188	15.1		0.5	14.2–16.2
Male (%)	188		48		–
BMI <i>Z</i> -score	187	0.4		0.9	–2.3–2.5
Puberty stage (%)	181				
Prepubertal	0	–		–	–
Early pubertal	8		4		–
Mid-pubertal	29		16		–
Late pubertal	119		66		–
Postpubertal	25		14		–
Neighbourhood-level SEP	187	1051		41	882–1151
Maternal education (%)	184				
Did not complete high school	37		20		–
Completed high school	61		33		–
Completed tertiary or postgraduate study	86		47		–
Exposure					
Diet quality score					
Wave 1 (age 4 years)	163	10.2		1.8	4.9–14.0
Wave 2 (age 4–5 years)	152	10.4		1.7	4.9–13.4
Wave 3 (age 5 years)	137	10.4		1.7	4.4–14.0
Wave 4 (age 5–5 years)	160	10.3		1.6	3.4–13.0
Wave 5 (age 6 years)	167	10.3		1.6	5.0–13.4
Wave 6 (age 6–5 years)	174	10.2		1.6	5.3–13.3
Wave 7 (age 10 years)	149	10.1		1.6	5.0–13.6
Wave 8 (age 15 years)	188	9.9		1.7	3.7–13.2
Outcomes					
Resting heart rate (bpm)	186	66.9		9.9	42.0–94.0
Systolic blood pressure (mmHg)	186	119.3		9.7	91.0–145.0
Diastolic blood pressure (mmHg)	186	65.9		6.2	51.0–85.0
Pulse wave velocity (m/s)	177	4.6		0.7	2.7–9.3
Carotid intima-media thickness (µm)	99	475.3		57.3	340.0–570.0
Retinal arteriole-to-venule ratio (%)	103	69.1		4.8	57.0–82.0

PEAS, Parent Education and Support; SEP, socio-economic position; bpm, beats/min.

*Data presented are mean and SD except for variables male, puberty stage and maternal education, which are %.

Table 3 Correlations between study variables, PEAS Kids Growth Study, Melbourne, Australia, 2002–2014

Exposure	1	2	3	4	5	6	7	8	9	10	11	12	13
Diet quality score													
1. Wave 1 (age 4 years)	0.62												
2. Wave 2 (age 4.5 years)	0.66	0.65											
3. Wave 3 (age 5 years)	0.68	0.61	0.71										
4. Wave 4 (age 5.5 years)	0.59	0.59	0.70	0.71									
5. Wave 5 (age 6 years)	0.53	0.57	0.60	0.67	0.66								
6. Wave 6 (age 6.5 years)	0.46	0.34	0.38	0.57	0.56	0.53							
7. Wave 7 (age 10 years)	0.28	0.28	0.41	0.40	0.41	0.37	0.41						
8. Wave 8 (age 15 years)													
Wave 8 outcomes													
9. Resting heart rate (bpm)	-0.08	-0.09	-0.06	-0.03	-0.02	-0.06	-0.18	-0.03					
10. Systolic blood pressure (mmHg)	0.05	-0.02	-0.02	-0.01	-0.09	-0.13	-0.11	-0.15	-0.00				
11. Diastolic blood pressure (mmHg)	0.00	0.04	-0.15	-0.13	-0.18	-0.11	-0.11	-0.13	0.25	0.51			
12. Pulse wave velocity (m/s)	0.08	0.08	0.03	-0.01	0.02	0.01	0.03	-0.12	0.11	0.30	0.32		
13. Carotid intima-media thickness (µm)	0.00	-0.09	0.00	0.01	0.01	0.10	0.11	0.06	0.11	-0.02	-0.20	-0.01	
14. Retinal arteriole-to-venule ratio (%)	-0.05	-0.04	0.05	0.02	0.06	-0.12	0.11	-0.01	-0.02	-0.03	-0.18	-0.01	-0.04

PEAS, Parent Education and Support; bpm, beats/min. Significant correlations ($P < 0.05$) are indicated in bold.

Results

Sample characteristics are shown in Table 2. On average, children were aged 4.2 (SD 0.2) years at Wave 1 and 15.1 (SD 0.5) years at Wave 8. The sample contained a similar distribution of male and female participants, and the majority were classed as late pubertal at Wave 8. Baseline values of socio-economic position (mean 1050 (SD 41)) were higher than the national mean of 1000 (SD 100)⁽⁶¹⁾, which suggests less disadvantage and heterogeneity in our sample. Participants' mothers were also slightly more educated than population norms⁽⁷²⁾. Compared with reference values, adolescent mean BMI Z-score was slightly elevated⁽⁶⁴⁾, blood pressure was normal⁽⁷³⁾, and both pulse wave velocity and resting heart rate averages were close to the 25th percentile^(74,75). We are not aware of reference values or cut-off points for children's carotid intima-media thickness and retinal arteriole-to-venule ratio values that have been linked to cardiovascular risk.

Table 3 shows the correlation matrix for study variables. Food diary scores correlated strongly with each other across time but were largely uncorrelated with the measured outcomes. Adolescent blood pressure correlated positively with pulse wave velocity and with resting heart rate (diastolic only).

Dietary trajectories

Model fit statistics for the two-, three-, four- and five-trajectory models can be found in Table 4. Models with two, three and four trajectories had an entropy above 0.8, with the model fit statistics (AIC, BIC, adjusted BIC) decreasing with the addition of extra trajectory classes, indicating improved model fit. The five-trajectory solution led to the identification of two small latent classes ($\leq 7\%$ of the sample) but a lower entropy and a larger likelihood ratio P value, so was not appropriate. Therefore, in balancing the *a priori* model criteria stated above (e.g. entropy above 0.8), we selected the four-trajectory model (entropy = 0.81, likelihood ratio test $P = 0.09$) as it optimised class size, interpretability of trajectories and the model fit statistics. The average posterior probabilities for most likely latent class membership (i.e. the probability that participants were accurately classified into their respective trajectory) were all close to 1.0: trajectory 1 = 0.98; trajectory 2 = 0.89; trajectory 3 = 0.88; trajectory 4 = 0.89.

We labelled the four dietary trajectories between age 4 and 15 years as follows.

1. Healthy ($n = 40$, 21%, mean diet score = 11.7; reference category).
2. Moderately healthy ($n = 87$, 46%, mean diet score = 10.5).
3. Moderately unhealthy ($n = 46$, 25%, mean diet score = 9.3).
4. Unhealthy ($n = 15$, 8%, mean diet score = 7.2).

The majority (67%) of participants followed the two healthiest trajectories. Regardless of dietary trajectory, the

Table 4 Model fit statistics, PEAS Kids Growth Study, Melbourne, Australia, 2002–2014

Trajectories	Class sizes*	AIC	BIC	Adjusted BIC†	Entropy	P value‡
2	27:73	4742	4823	4745	0.87	0.03
3	8:45:47	4565	4676	4568	0.84	0.07
4	8:24:46:22	4507	4647	4511	0.81	0.09
5	5:32:35:7:22	4494	4663	4499	0.79	0.30

PEAS, Parent Education and Support; AIC, Akaike's information criterion; BIC, Bayesian information criterion.

Model fit statistics considered included the following: (i) AIC, BIC and adjusted BIC, all of which we aimed to minimise; (ii) model entropy, which we aimed to maximise; and (iii) the *P* value from the Vuong–Lo–Mendell–Rubin (VLMR) likelihood ratio test of whether adding an additional trajectory category improved model fit. The model chosen is indicated in bold font.

*Percentage of the sample in each class/trajectory.

†Adjusted for sample size.

‡VLMR likelihood ratio test *P* value.

obtained patterns suggest that changes in children's dietary quality occurred during the interval between 6.5 and 10 years of age. Within this period, children in the least healthy trajectories tended to begin an upward trend in diet quality, while those in the healthiest trajectories started to become slightly less healthy. Consequently, all four trajectories converged with age resulting in trajectories that were less distinct by the adolescent years (Fig. 2).

Regression analyses

No associations were evident between dietary trajectories and blood pressure, pulse wave velocity, carotid intima-media thickness and retinal arteriole-to-venule ratio. If anything, carotid intima-media thickness was thinner and pulse wave velocity better in those with the least healthy dietary trajectories (Table 5).

In contrast, higher (worse) resting heart rate was associated with worsening dietary trajectories. In the fully adjusted regression model, the effect for the unhealthy trajectory, compared with the reference healthy trajectory, was large and statistically significant (10.5 bpm, 95% CI

2.9, 18.0, $d=1.1$). Against available population-based norms (US) for adolescent resting heart rate, this means that children following the least healthy trajectory have a resting heart rate on the 50th percentile⁽⁷⁴⁾, which is more than one standard deviation higher (worse) than those children following the healthiest trajectory, whose resting heart rate is close to the 10th percentile⁽⁷⁴⁾. In addition, the effects for the moderately unhealthy (4.5 bpm, 95% CI -0.7, 9.7, $d=0.5$) and moderately healthy (4.1 bpm, 95% CI -0.6, 8.9, $d=0.4$) trajectories were in the same direction (Table 5), albeit these effect sizes were small (about one-third of a standard deviation) with higher *P* values ($P<0.10$). All associations were similar with (shown) and without (not shown) adjustment for concurrent BMI *Z*-score.

Discussion

Statement of principal findings

Of the four dietary trajectories, most participants followed the healthiest trajectories, with only 8% following the least healthy trajectory. Changes in children's diet quality occurred between 6.5 and 10 years of age, with marked early-life differences in diet quality becoming slightly less marked by adolescence. Finally, independent of potential confounders, children who consumed the least healthy diets recorded the highest resting heart rates, but no differences were evident in other cardiovascular measures.

Comparison with prior literature

Many Australian children and adolescents fail to meet dietary recommendations^(10,11,76), although only 8% of our sample continually followed the least healthy trajectory. This result is very similar to previous research within population-representative cohorts of Australian children⁽³⁷⁾, although it is possible that children in our relatively affluent sample follow healthier trajectories than the general population. We also showed that all four typical trajectories inflected to move closer together somewhere between 6.5 and 10 years of age, possibly reflecting the age period at which children gain more influence over their parents' purchasing behaviour and/or when they

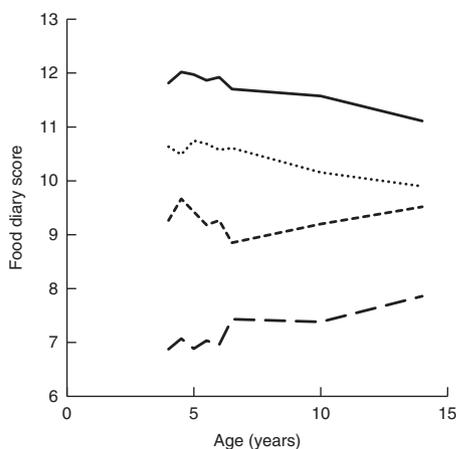


Fig. 2 Empirically derived dietary score trajectories (—, trajectory 1: healthy (21%); ·····, trajectory 2: moderately healthy (46%); - - - -, trajectory 3: moderately unhealthy (25%); — · —, trajectory 4: unhealthy (8%)) from age 4 to 15 years among 188 children from the PEAS Kids Growth Study, Melbourne, Australia, 2002–2014 (PEAS, Parent Education and Support)

Table 5 Regression results of the association between childhood dietary trajectories and adolescent cardiovascular phenotypes among 188 children from the PEAS Kids Growth Study, Melbourne, Australia, 2002–2014

Cardiovascular outcome	Descriptive statistics								Fully adjusted* mean difference from the healthy reference trajectory								
	Healthy trajectory (reference)		Moderately healthy trajectory		Moderately unhealthy trajectory		Unhealthy trajectory		Moderately healthy trajectory			Moderately unhealthy trajectory			Unhealthy trajectory		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	β	95% CI	<i>P</i>	β	95% CI	<i>P</i>	β	95% CI	<i>P</i>
Resting heart rate (bpm)									4.1	-0.6, 8.9	0.08	4.5	-0.7, 9.7	0.09	10.5	2.9, 18.0	0.01
Unadjusted	65.1	7.8	67.0	10.1	67.3	9.7	69.7	13.5									
Adjusted†	63.7	12.1	67.9	12.4	68.2	11.5	74.2	12.1									
Systolic blood pressure (mmHg)									-0.3	-4.6, 4.0	0.88	1.9	-2.9, 6.6	0.43	-1.1	-8.0, 5.7	0.74
Unadjusted	117.9	9.5	119.2	10.2	120.7	9.5	119.9	7.6									
Adjusted†	118.8	11.1	118.4	11.3	120.6	10.5	117.6	11.0									
Diastolic blood pressure (mmHg)									0.3	-2.7, 3.2	0.86	-0.0	-3.2, 3.2	0.99	0.4	-4.2, 5.1	0.85
Unadjusted	65.1	4.9	66.0	7.1	65.8	5.5	67.4	5.6									
Adjusted†	65.5	7.5	65.8	7.7	65.5	7.1	66.0	7.5									
Pulse wave velocity (m/s)									-0.0	-0.4, 0.4	0.87	-0.0	-0.4, 0.3	0.81	-0.2	-0.8, 0.3	0.38
Unadjusted	4.6	0.6	4.7	0.6	4.6	1.0	4.6	0.8									
Adjusted†	4.7	0.9	4.6	0.9	4.6	0.8	4.4	0.9									
Carotid intima-media thickness (μ m)									-9.4	-45.2, 26.4	0.60	-10.5	-49.7, 28.7	0.60	-14.7	-80.5, 51.1	0.66
Unadjusted	481.3	54.8	472.1	62.4	476.1	47.6	470.0	81.2									
Adjusted†	483.2	67.9	473.8	70.7	472.7	69.2	468.5	71.7									
Retinal arteriole-to-venule ratio (%)									-0.0	-2.6, 2.6	0.99	1.4	-1.5, 4.3	0.33	2.0	-2.9, 6.9	0.42
Unadjusted	69.5	4.0	68.8	3.9	69.6	6.6	66.7	4.3									
Adjusted†	68.5	5.1	68.5	5.3	69.9	5.2	70.5	5.4									

PEAS, Parent Education and Support; SEIFA, Socio-Economic Indexes for Areas; IRSD, Index of Relative Socio-economic Disadvantage.

Model statistics remain similar between unadjusted and partially adjusted regression models (i.e. without BMI Z-score). Analytic sample included participants with one or more cardiovascular measure at Wave 8 AND a complete Wave 8 food diary. More stringent food diary inclusion criteria (Wave 1 AND Wave 8 diaries) did not change the trajectories or results obtained.

**A priori* confounders in adjusted models: Wave 1 maternal education, SEIFA-IRSD score; Wave 8 child age, sex, accelerometer-measured physical activity, adolescent-reported puberty, BMI Z-score.

†Adjusted marginal mean (i.e. the mean adjusted for all *a priori* confounders in the model).

begin to have more independent food choice. While we could not pinpoint the timing of these changes more precisely, previous research using the same measure biennially in two large Australian cohorts confirms these inflections and places their timing at about 7–8 years⁽³⁷⁾.

The childhood dietary trajectories predicted differences in adolescent resting heart rate. This result aligns with data from the Western Australia Pregnancy (Raine) Cohort⁽³⁶⁾, where heart rate, but not blood pressure, decreased with better adherence to Australian Dietary Guidelines; thus, a 30-point change in participants' diet score was associated with a 3-year fall of 2.4 bpm between age 14 and 17 years. Although the differences between our study and the Raine study (e.g. age range, dietary measure) preclude direct comparisons, the direction of effect (i.e. healthier diet = lower heart rate) is the same and the magnitude of heart rate change is comparable. We reinforce and extend this finding by demonstrating that following a consistently unhealthy childhood diet is associated with having a resting heart rate that is on average 11 bpm (about one standard deviation) faster in adolescence than the heart rate of children who followed a consistently healthy diet. Moreover, although effect sizes were small and only marginally significant due to our small sample size, the patterns suggest that data follow a dose–response relationship. Thus, children following even moderately healthy diets throughout childhood also recorded a resting heart rate on average 4–5 bpm faster (about one-third of a standard deviation) than did children with consistently healthy diets.

The majority of our results do not support previous research linking childhood or adolescent diet to indicators of cardiovascular health, such as blood pressure or retinal microvascular parameters^(18,20,21,24–26,28,29,33). These results were consistently weak, such that this lack of statistical association did not seem to primarily reflect our small sample size. As our study is unique in the frequency and number of dietary measurements, these discrepancies may reflect differences depending on whether diet is measured cumulatively throughout childhood, or whether it is measured infrequently in early life or adolescence. Diet is notoriously difficult to measure via self- and/or parent-report⁽⁷⁷⁾ but identifying latent variables across multiple time points should reduce the effect of measurement errors concomitant with the single measures and more reliably identify patterns over time⁽⁷⁸⁾.

Strengths and weaknesses

Because dietary data were questionnaire-based, the trajectories may be subject to reporting bias and these self-reported data did not allow us to account for portion size or the energy density of food items; new online measures with enhanced accuracy should address this issue for future research. Furthermore, we did not have resources to collect data on circulating biomarkers or on cardio-respiratory fitness and our results may therefore be

affected by unmeasured residual confounding. In addition, with successive waves, dietary measurement decreased in frequency and graduated from parent- to self-reported. As noted, the diets of children in the healthiest trajectories became less healthy over time, while those in the least healthy trajectories became healthier. While the patterns may resemble regression to the mean, this should not be an issue as latent class analysis considers patterns of diet over the fully observed period without any selectivity of individuals according to extremity of their baseline score. We acknowledge the possibility that these patterns may reflect the change in measurement frequency after Wave 6 and/or child self-reporting diet at Waves 7 and 8, rather than actual dietary change.

The main limitation is the small sample size (especially for the vascular structural measures) and relatively advantaged cohorts available for these exploratory analyses. Clearly, larger studies are needed to confirm and extend these findings, and such data sets are now becoming available. Nevertheless, the current study is strengthened by its longitudinal design, community sample, repeated dietary measures throughout the early life course, and the unusual rigour and breadth of cardiovascular measures that have been out of reach of most previous studies. Our adjustment for multiple confounding variables may partly explain differences in effect sizes with previous research.

Implications

The differences between 'typical' dietary pathways followed by Australian children in our small study were striking. Very marked differentiation of long-term diet quality trajectories was entrenched by the pre-school years, suggesting that interventions to maximise dietary healthfulness may need to occur in the early years. Given the continuity of autonomic risk across the full range of dietary quality, such interventions may need to be universal. Conversely, interventions in the early primary school years may best be specifically designed to ward off declines in dietary quality – needing different strategies. Nevertheless, because effects did not emerge for the remaining cardiovascular measures, we recommend replication of our data before drawing this conclusion.

Heart rate (a reflection of sympathetic nervous system activity) and vascular function are correlated; for example, a reduction in vascular stiffness following weight loss in obese 20–45-year-old adults paralleled concomitant slowing of resting heart rate⁽⁷⁹⁾. There is good evidence that risk factors present early in the life course increase the risk of CVD in adulthood⁽⁸⁾. Supported by previous research during later adolescence⁽³⁶⁾, our study raises the novel possibility that dietary trajectories in healthy children may effect changes in heart rate by age 15 years, with diet-related changes in adult arterial stiffness potentially following⁽⁸⁰⁾. What little evidence exists for adolescents is inconsistent as to the importance of heart rate differences

within the normal range^(55,81). However, small increments in adult resting heart rate (often within the normal range) predict both cardiovascular and all-cause mortality over a relatively short period^(54,82). In the Atherosclerosis Risk in Communities (ARIC) study of middle-aged adults, every 5-bpm increase in resting heart rate from the preceding visit (≈ 3 years previously) incurred a $\approx 12\%$ increased risk of mortality over 28 years of follow-up⁽⁸²⁾. Additional analyses demonstrated that this risk was associated with a time-adjusted resting heart rate of >66 bpm, compared with 60 bpm. This suggests that the size of our apparent dose–response differences in adolescent resting heart rate by worsening childhood diet quality trajectories is important, with our adjusted means higher among each worsening diet trajectory (63.7 to 67.9 to 68.2 to 74.2 bpm; Table 5). The ARIC findings⁽⁸²⁾ suggest that these effects sizes could be very important to later-life mortality if relationships between diet quality and resting heart rate remain stable or become more pronounced with age. Future research should examine whether improving diet before or during the adolescent years lowers heart rate and mitigates adult cardiovascular risk.

Conclusion

Our study extends currently limited research investigating childhood diet and early-life cardiovascular phenotypes. Decade-long dietary trajectories in healthy children did not appear to influence macro- or microvascular structure or stiffness by mid-adolescence, but were associated with resting heart rate, suggesting an early-life window for prevention. Although participants' heart rate values were within the normal range, the obtained gradient in adolescence may have cumulative long-term impacts on life-course outcomes. Larger studies are needed to confirm these findings, the threshold of diet quality associated with these physiological changes and whether functional changes in heart rate are followed by structural changes in large and small vessel phenotypes.

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