British Journal of Nutrition (2022), 127, 1704–1711 doi:10.1017/S0007114521002658

Association between diet quality in adolescence and adulthood and knee symptoms in adulthood: a 25-year cohort study

Tao Meng[1,2], Johanna Wilson[1], Alison Venn[1], Flavia Cicuttini[3], Lyn March[4], Marita Cross[4], Terence Dwyer[1,5], Leigh Blizzard[1], Graeme Jones[1], Laura Laslett[1], Benny Antony[1] and Changhai Ding[1,6]*

[1]*Menzies Institute for Medical Research, University of Tasmania, Hobart, Australia*

[2]*Department of Rheumatology and Immunology, The Second Hospital of Anhui Medical University, Hefei, People's Republic of China*

[3]*Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia*

[4]*Institute of Bone and Joint Research, University of Sydney, Sydney, Australia*

[5]*The George Institute for Global Health, University of Oxford, Oxford, UK*

[6]*Clinical Research Centre, Zhujiang Hospital, Southern Medical University, Guangzhou, People's Republic of China*

(Submitted 31 January 2021 – Final revision received 23 May 2021 – Accepted 9 July 2021 – First published online 14 July 2021)

Abstract

We aimed to describe associations between diet quality in adolescence and adulthood and knee symptoms in adulthood. Two hundred seventy-five participants had adolescent diet measurements, 399 had adult diet measurements and 240 had diet measurements in both time points. Diet quality was assessed by Dietary Guidelines Index (DGI), reflecting adherence to Australian Dietary Guidelines. Knee symptoms were collected using Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC). Data were analysed using zero-inflated negative binomial regressions. The overall adolescent DGI was not associated with adult knee symptoms, although lower intake of discretionary foods (e.g. cream, alcohol, bacon and cake) in adolescence was associated with lower pain (mean ratio (MR) 0·96) and dysfunction (MR 0·94). The overall adult DGI was not associated with knee symptoms; however, limiting saturated fat was associated with lower WOMAC (Pain: MR 0·93; stiffness: MR 0·93; dysfunction: MR 0·91), drinking water was associated with lower stiffness (MR 0·90) and fruit intake was associated with lower dysfunction (MR 0·90). Higher DGI for dairy products in adulthood was associated with higher WOMAC (Pain: MR 1·07; stiffness: MR 1·13; dysfunction: MR 1·11). Additionally, the score increases from adolescence to adulthood were not associated with adult knee symptoms, except for associations between score increase in limiting saturated fat and lower stiffness (MR 0·89) and between score increase in fruit intake and lower dysfunction (MR 0·92). In conclusion, the overall diet quality in adolescence and adulthood was not associated with knee symptoms in adulthood. However, some diet components may affect later knee symptoms.

Key words: Diet quality: Adolescence: Adulthood: Knee symptoms: Osteoarthritis

Knee osteoarthritis (OA) is the most prevalent joint disease worldwide and is associated with pain, stiffness and loss of function. However, no disease-modifying treatments are available[1]. Although knee OA is commonly diagnosed among middle-aged or older population, the disease process can start during earlier life. Studies have found that the prevalence of knee pain can even exceed 30 % among adults aged 30–40 years[2]. The knee symptoms may be one of the early risk factors of knee OA in later life[3]. A systematic review indicated that the evidence from well-conducted case–control studies supported knee pain was a predisposing factor of knee OA, though there was a paucity of well-designed cohort studies[4]. Thus, identifying factors affecting knee symptoms among adults aged about 30–40 years may be important for developing prevention strategies and management of knee OA.

Diet is important in disease prevention (e.g. CVD), as the foods consumed in daily life could have beneficial or detrimental effects on our health[5–8]. Two approaches are generally employed in data analysis of diet: hypothesis-oriented approaches (reflecting the adherence to guidelines or recommendations, e.g. dietary score) and exploratory approaches, which focus on study-specific data, for example, principal component analysis[9,10]. Hypothesis-oriented approaches have advantages over exploratory approaches as they are based on

Abbreviations: ASHFS, Australian Schools Health and Fitness Survey; CDAH, Childhood Determinants of Adult Health; DGI, Dietary Guidelines Index; OA, osteoarthritis; WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

* **Corresponding author:** Changhai Ding, email changhai.ding@utas.edu.au

existing knowledge of optimal diet and provide clear information about healthy levels[11]. Several studies have reported associations between daily diet assessed by dietary score and knee OA in older adults with knee OA or at risk of OA. Cross-sectionally, adherence to a Mediterranean diet was associated with lower knee pain[12] and lower prevalence of knee OA[13]; longitudinally, adherence to a Mediterranean diet was associated with lower risk of knee pain worsening and lower incidence of symptomatic knee OA[14]. However, no studies have assessed the effect of diet on knee symptoms in younger adults, who are an important target group for disease prevention. Moreover, the Mediterranean diet index was defined according to dietary habits[15], but not a national dietary guideline which represents the most recommended diet based on the current evidence.

Therefore, we aimed to describe associations between diet quality (as assessed by adherence to national dietary guidelines) in adolescence and adulthood and knee symptoms in adulthood.

Methods

Participants

In 1985, The Australian Schools Health and Fitness Survey (ASHFS) was conducted to provide benchmark data on the health and fitness of Australian schoolchildren/adolescents (7–15 years, *n* 8498) with a nationally representative sample. Two-stage probability sampling was used for the ASHFS to randomly select schools and then children/adolescents within age groups in schools. Students aged 10–15 years completed the 24-h diet record in adolescence and represent the adolescent cohort in the current analysis. The Childhood Determinants of Adult Health (CDAH) Study was a 20-year follow-up of children/adolescents who participated in the ASHFS. During 2004–2006, participants (aged 26–36 years) completed the food frequency questionnaire (FFQ) and food habits questionnaire (FHQ) and attended clinics (*n* 2410) which were located at sites in major cities and regional centres around Australia and represent the adulthood cohort in the current analysis. The CDAH Knee Cartilage Study was a 4–5 year follow-up of the CDAH study. During 2008–2010, participants residing in metropolitan Melbourne and Sydney were invited to attend a computer-assisted telephone interview as part of the CDAH Knee Cartilage Study. The exclusion criteria included: being pregnant, having other forms of arthritis, having contraindications of MRI, and finally 449 participants (aged 31–41 years) participated and completed the knee symptom questionnaire. The detailed reasons for non-participation in the CDAH Knee Cartilage Study included: 1646 did not reside in Melbourne or Sydney, 235 did not respond or refused, three were pregnant during the CDAH Knee Cartilage Study, two had rheumatoid arthritis, thirteen had contraindications of MRI and sixty-two withdrew. The details of enrolment of ASHFS, CDAH and the CDAH Knee Cartilage Study have been published elsewhere[2,16]. In the current analysis, 275 of the 449 with a knee symptom questionnaire had adolescent diet measurements when they were aged 10–15 years in ASHFS, 399 had eligible adult diet measurements (fourteen were pregnant when diet data were collected and thirty-six missed > 10 % FFQ item responses or key FHQ responses), resulting in 240 with eligible diet measurements in both adolescence and adulthood (Fig. 1).

This study was approved by the Southern Tasmania Health and Medical Human Research Ethics Committee, the Monash University Human Research Ethics Committee and the Northern Sydney and Central Coast Area Health Human Research Ethics Committee. All participants provided written informed consent.

Anthropometric measurements

Weight was measured to the nearest 0·5 kg in 1985 and 0·1 kg at follow-up (with shoes and bulky clothing removed) using a scale (Heine). Height was measured to the nearest 0·1 cm (with shoes and socks removed) using a stadiometer (Invicta). BMI was calculated as weight in kg divided by height in metres squared.

Dietary measurements

In ASHFS, participants recorded the time and estimated amount of each food or drink item consumed during a 24-h period. Trained data collectors showed the adolescents how to fill out the food record, and each adolescent was interviewed on collection of the records to check and clarify the entries. The paper questionnaires were manually processed in 1985 to provide the gram weight and the kJ energy content of each food or beverage item. The energy content of each item was determined using a specially compiled database of the nutrient composition of Australian foods[17]. The data for each item were used for this current study to calculate the proportion of a standard serving as defined in the 2013 Australian Dietary Guidelines[18]. For example, if a participant reported consuming 60 g of toast at breakfast in the ASHFS food record, this equates to 1·5 standard 40-g servings of bread. In CDAH, participants completed a 127-item FFQ and an FHQ and the paper questionnaires were scanned using Teleform (version 9.0). Each frequency reported in the FFQ was assumed to be a standard serving defined in the 2013 Australian Dietary Guidelines[18]. For example, if a participant reported eating breakfast cereal once per day, this was assumed to be one standard 30-g serving of grains. Dietary Guidelines Index (DGI) and total energy intake were calculated based on the consumed servings. The DGI comprises nine indicators, and the maximum possible score was 100. A higher score indicated higher diet quality (higher adherence to Australian Dietary Guidelines). Seven indicators, worth 10 points each, related to recommended minimum intakes (dietary variety, vegetables, fruit, grains, lean meats and alternatives, low-fat dairy products and alternatives, water). For example, a participant scored 10 for fruit intake if they consumed at least two servings of fruit as guideline recommended. Participants could receive proportional scores for partially meeting the recommendations. Two indicators were for lower intake of discretionary foods (20 points), including foods high in saturated fat (e.g. cream), alcohol, added salt (e.g. bacon), and added sugars (e.g. cake) and limiting saturated fats (10 points). The details of DGI measures have been published elsewhere[11]. The changes in diet quality from adolescence to adulthood were represented by the score changes of DGI and its components (scores in adulthood minus the corresponding scores in adolescence).

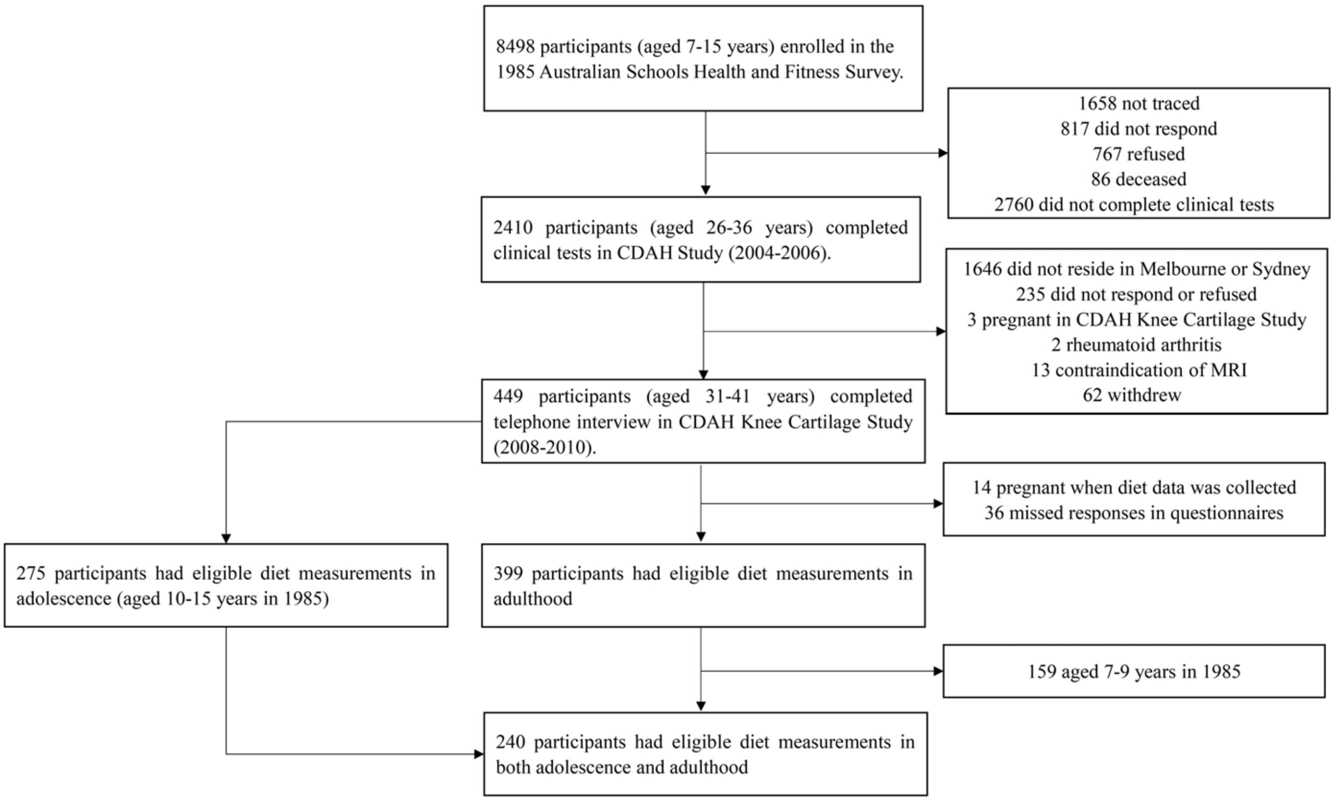

Fig. 1. Flow chart showing selection of the participants for the current study from previous studies. CDAH Study, Childhood Determinants of Adult Health Study.

Physical activity measurements

In ASHFS, participants self-reported past week duration and frequency of discretionary sport or exercise (leisure activity), walking and cycling to and from school (transport activity), school physical education and school sport. For each activity, frequency was multiplied by duration to estimate min/week and activities were summed to estimate total physical activity. In CDAH, physical activity was assessed using the long version of the International Physical Activity Questionnaire. Participants were asked to report the total time and frequency of occupational, commuting, domestic and leisure activity during the past week. Physical activities were calculated by multiplying frequency by duration to estimate min/week of vigorous, moderate and walking activity. Time spent in each domain was summed to estimate total physical activity.

Knee symptom measurements

The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) is widely used for evaluating clinically important symptoms in OA patients[19]. WOMAC has also shown adequate responsiveness, content and construct validity for evaluating knee complaints in adolescents and adults[20]. In the CDAH Knee Cartilage Study, participants completed a questionnaire, including WOMAC. Knee pain, stiffness and dysfunction during the past 30 d were assessed on a scale of 0–9 for each subscale, where 0 indicated no complaints and 9 indicated the maximum intensity of the complaint. WOMAC pain, stiffness and dysfunction were assessed in 5, 2 and 17 subscales, respectively. WOMAC scores were calculated by adding the score of subscales in each domain. The maximum possible scores of pain, stiffness and dysfunction were 45, 18 and 153, respectively. A non-zero score indicates the presence of knee symptom. Knee injury history was also recorded in the questionnaire.

Statistical analyses

Mean (standard deviation), number (percentage) and median (interquartile range) were used to describe the characteristics of the participants. Zero-inflated negative binomial regression analyses were used to assess the associations between diet quality and knee symptoms and estimate the mean ratios, as there are exceeded zeros in the knee symptom data and the sample variances significantly exceeded the sample means (overdispersion)[21,22]. Age, sex, BMI, physical activity, total energy intake and knee injury history were included as potential confounders based on biological plausibility. A *P*-value $<0{\cdot}05$ (2-tailed) was considered as statistical significance. All statistical analyses were performed in Stata (Texas, USA), version 16.0.

Results

Among the participants included in data analysis, the average adolescent and adult age with dietary data was 12·6 (SD 1·8)

British Journal of Nutrition

and 30·9 (SD 2·8) years, and the average adolescent and adult DGI was 46·5 (SD 12·6) and 55·4 (SD 11·3), respectively. The prevalence of knee pain, stiffness and dysfunction (at mean age 35·4 years) was 35·1 % (30·4 %, 40·0 %), 31·6 % (27·0 %, 36·4 %) and 39·9 % (35·1 %, 44·8 %), respectively (Table 1).

Overall adolescent DGI was not associated with knee pain, stiffness or dysfunction in adulthood (Table 2). Similarly, most DGI components (vegetable, food variety, fruit, grain, lean protein, dairy products, water, limiting saturated fat) were not associated with adult knee symptoms, although higher score for lower intake of discretionary foods in adolescence was significantly associated with lower knee pain and dysfunction in adulthood (Table 2).

Overall adult DGI and some DGI components (vegetable, food variety, grain, lean protein, lower intake of discretionary foods) were not associated with knee pain, stiffness or dysfunction in adulthood (Table 3). However, higher score for limiting saturated fat in adulthood was significantly associated with lower WOMAC pain, stiffness and dysfunction (Table 3). In addition, higher score for drinking water was significantly associated with lower stiffness and higher score for fruit intake was significantly associated with lower dysfunction (Table 3). In contrast, higher DGI score for dairy intake was significantly associated with higher WOMAC pain, stiffness and dysfunction (Table 3).

The changes of overall DGI and most of the component scores (vegetable, food variety, grain, lean protein, dairy products, water, lower intake of discretionary foods) from adolescence to adulthood were not associated with knee pain, stiffness or dysfunction in adulthood (Table 4). However, the change of component score for limiting saturated fat from adolescence to adulthood was associated with lower stiffness, and the change score for fruit intake was associated with lower dysfunction in adulthood (Table 4).

Table 1. Characteristics of participants
(Median values and interquartile range (IQR); numbers and percentages; mean values and standard deviations)

Characteristics	Mean	SD
Adolescence	*n* 275	
Age (years)	12·6	1·8
Female		
n	131	
%	47·6	
BMI (kg/m²)	19·0	2·4
Physical activity (h/week)		
Median	7·6	
IQR	3·1, 9·8	
Total energy intake (kJ/d)	8753	3019
Dietary Guideline Index (DGI)		
Vegetable (0–10)	3·7	3·3
Food variety (0–10)	7·9	1·7
Fruit (0–10)	5·5	3·6
Grain (0–10)	5·1	2·5
Lean protein (0–10)	4·6	3·3
Dairy products (0–10)	3·1	2·1
Water (0–10)	6·4	1·5
Lower intake of discretionary foods (0–20)	3·5	6·8
Limiting saturated fat (0–10)	6·7	0·9
Overall adolescent DGI (0–100)	46·5	12·6
Adulthood	*n* 399	
Age (years)	30·9	2·8
Female		
n	193	
%	48·4	
BMI (kg/m²)	25·0	4·0
Physical activity (h/week)		
Median	9·3	
IQR	5·6, 15·9	
Knee injury history		
n	72	
%	18·1	
Total energy intake (kJ/d)	8640	2646
Dietary Guidelines Index		
Vegetable (0–10)	4·1	2·2
Food variety (0–10)	5·6	1·5
Fruit (0–10)	7·1	2·8
Grain (0–10)	5·7	2·4
Lean protein (0–10)	8·3	1·2
Dairy products (0–10)	7·3	2·9
Water (0–10)	7·7	1·9
Lower intake of discretionary foods	2·3	5·3
Limiting saturated fat (0–10)	7·3	2·7
Overall adult DGI (0–100)	55·4	11·3
WOMAC (>0)	*n*	%
Pain	140	35·1
Stiffness	126	31·6
Dysfunction	159	39·9

WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

Discussion

This is the first study describing the associations between diet quality in adolescence and adulthood and knee symptoms in adulthood. We found overall DGI and a large number of DGI components in adolescence and adulthood and their changes from adolescence to adulthood were not associated with knee symptoms in adulthood. However, several DGI components (lower intake of discretionary foods in adolescence, limiting saturated fat, fruit intake and water intake in adulthood) were associated with lower knee symptoms, whereas higher DGI score for dairy products in adulthood was associated with higher knee symptoms. Moreover, the change of DGI score from adolescence to adulthood for limiting saturated fat was associated with lower stiffness, and the change of DGI score for fruit intake was associated with lower dysfunction.

We did not find significant associations between the overall DGI in adolescence and adulthood and knee symptoms in adulthood. The negative results may be because the effects from the different DGI components were different. Although some components (e.g. limiting saturated fat in adulthood) were associated with lower knee symptoms, the effects may be neutralised by another component (e.g. dairy products) which has the opposite effects. Our results were consistent with a previous review, which suggested that different nutrients could have antagonistic effects on a chronic disease[10].

We found that eating only lower amounts of discretionary foods in adolescence, independent of BMI, was associated with lower knee pain and dysfunction in adulthood, which has not been reported in previous studies. Consuming greater amounts of discretionary foods has been associated with metabolic diseases (e.g. type 2 diabetes) in young adults[23], and the associations may even persist after adjustment for BMI[24,25]. Knee OA

https://doi.org/10.1017/S0007114521002658 Published online by Cambridge University Press

Table 2. Association between diet quality in adolescence and knee symptoms in adulthood*
(Mean values and 95 % confidence intervals, *n* 275)

	Pain		Stiffness		Dysfunction	
	Mean ratio	95 % CI	Mean ratio	95 % CI	Mean ratio	95 % CI
Vegetable (0–10)	0·98	0·93, 1·04	1·01	0·95, 1·07	1·04	0·95, 1·15
Food variety (0–10)	0·97	0·86, 1·10	0·92	0·80, 1·05	1·08	0·88, 1·32
Fruit (0–10)	1·02	0·96, 1·08	0·95	0·87, 1·05	1·04	0·95, 1·13
Grain (0–10)	0·99	0·90, 1·08	0·95	0·88, 1·03	0·98	0·89, 1·08
Lean protein (0–10)	1·03	0·96, 1·11	0·93	0·87, 1·01	0·99	0·90, 1·08
Dairy products (0–10)	1·02	0·91, 1·14	1·00	0·89, 1·13	1·09	0·94, 1·27
Water (0–10)	0·88	0·76, 1·03	0·95	0·82, 1·11	0·96	0·81, 1·15
Lower intake of discretionary foods (0–20)	**0·96**	**0·92, 1·00**	0·97	0·93, 1·02	**0·94**	**0·90, 0·99**
Limiting saturated fat (0–10)	0·87	0·61, 1·24	0·91	0·64, 1·29	0·69	0·46, 1·04
Overall DGI (0–100)	0·99	0·97, 1·01	0·98	0·97, 1·00	0·99	0·97, 1·02

DGI, Dietary Guidelines Index.
Bold denotes statistical significance, $P < 0·05$.
*Adjusted for age, sex, BMI, physical activity, total energy intake in adolescence and knee injury history in adulthood.

Table 3. Association between diet quality in adulthood and knee symptoms in adulthood*
(Mean values and 95 % confidence intervals, *n* 399)

	Pain		Stiffness		Dysfunction	
	Mean ratio	95 % CI	Mean ratio	95 % CI	Mean ratio	95 % CI
Vegetable (0–10)	0·98	0·91, 1·07	0·96	0·86, 1·07	0·92	0·80, 1·05
Food variety (0–10)	1·03	0·92, 1·16	1·05	0·93, 1·19	0·98	0·85, 1·14
Fruit (0–10)	0·99	0·91, 1·07	1·00	0·92, 1·08	**0·90**	**0·81, 0·99**
Grain (0–10)	0·93	0·87, 1·00	1·05	0·98, 1·13	0·97	0·88, 1·06
Lean protein (0–10)	0·94	0·80, 1·09	0·94	0·81, 1·10	0·94	0·77, 1·15
Dairy products (0–10)	**1·07**	**1·00, 1·13**	**1·13**	**1·05, 1·21**	**1·11**	**1·02, 1·21**
Water (0–10)	0·98	0·90, 1·07	**0·90**	**0·83, 0·99**	0·94	0·85, 1·05
Lower intake of discretionary foods (0–20)	1·02	0·99, 1·05	1·03	0·99, 1·07	1·03	0·99, 1·09
Limiting saturated fat (0–10)	**0·93**	**0·87, 0·99**	**0·93**	**0·87, 0·99**	**0·91**	**0·83, 0·99**
Overall DGI (0–100)	1·00	0·98, 1·01	1·01	0·99, 1·03	1·00	0·98, 1·02

DGI, Dietary Guidelines Index.
Bold denotes statistical significance, $P < 0·05$.
*Adjusted for age, sex, BMI, physical activity, total energy intake and knee injury history in adulthood.

Table 4. Association between the change in diet quality and knee symptoms*
(Mean values and 95 % confidence intervals, *n* 240)

	Pain		Stiffness		Dysfunction	
Per 1 score increase	Mean ratio	95 % CI	Mean ratio	95 % CI	Mean ratio	95 % CI
Vegetable	0·99	0·94, 1·05	0·96	0·91, 1·02	0·92	0·85, 1·01
Food variety	0·95	0·84, 1·07	1·04	0·92, 1·18	0·89	0·75, 1·05
Fruit	0·96	0·91, 1·02	1·02	0·96, 1·09	**0·92**	**0·86, 0·99**
Grain	0·93	0·85, 1·01	1·03	0·95, 1·12	1·04	0·93, 1·17
Lean protein	0·93	0·86, 1·01	1·10	0·98, 1·23	1·03	0·91, 1·16
Dairy products	1·05	0·98, 1·13	1·07	0·99, 1·14	0·99	0·90, 1·09
Water	1·00	0·87, 1·14	0·86	0·74, 1·00	0·90	0·76, 1·05
Lower intake of discretionary foods	1·02	0·99, 1·05	1·04	0·99, 1·08	1·03	0·99, 1·08
Limiting saturated fat	0·94	0·87, 1·01	**0·89**	**0·80, 0·98**	0·93	0·81, 1·07
Overall DGI	0·99	0·98, 1·01	1·01	0·99, 1·03	0·99	0·96, 1·02

DGI, Dietary Guidelines Index.
Bold denotes statistical significance, $P < 0·05$.
*Adjusted for sex, age, BMI, physical activity, total energy intake in adolescence and adulthood and knee injury history in adulthood.

shares many causal pathways with these metabolic diseases[26]. Thus, eating lower amounts of discretionary foods may have beneficial effects on knee joint by reducing activation of causal pathways for metabolic diseases. These beneficial effects were only associated with adolescent diets not adult diets, even though the sample size was larger in adulthood than that in adolescence. The reason is unclear but may suggest that eating lower discretionary foods is particularly important in adolescence and could have long-term effects.

We found limiting saturated fat in adulthood was associated with lower knee pain, stiffness and dysfunction in adulthood. The underlying mechanism may be the decreased detrimental effects from saturated fat and/or increased beneficial effects from unsaturated fat on knee joint. Although there are no similar studies based on younger adult populations, some studies have reported that intake of saturated fat was associated with knee structural signs of OA in middle-aged healthy adults[27] and knee OA structural progression in patients with radiographic knee OA[28]. In addition, intake of unsaturated fat could have protective effects on knee structures[29] and symptoms (pain and function)[30,31] among patients with knee OA.

We found that intake of fruit in adulthood was associated with lower knee dysfunction in adulthood, which is partly consistent with previous trials. The trials reported that freeze-dried strawberry powder[32] and freeze-dried blueberry powder[33] could reduce pain and dysfunction in adults with knee OA. However, we did not find evident effects on pain or stiffness; the reason may be that not all fruits have the same analgesic effects as berries[32]. We also found that water intake in adulthood was associated with low stiffness in adulthood. Drinking plenty of water has been associated with lower risk of chronic diseases, such as type 2 diabetes[34], suggesting its potential benefits on metabolic fitness. The underlying mechanism of our results warrants further studies but may have some shared metabolic pathways between diabetes and knee OA[35].

Our finding that dairy score was associated with higher knee pain, stiffness and dysfunction should be interpreted cautiously. Previous studies reported that milk consumption (any kind) was associated lower prevalence of symptomatic knee OA cross-sectionally[36] and less radiographic OA progression (decrease of joint space width) longitudinally[37]. However, the effects from specific kinds of milk (full-fat and skimmed milk) were not identified. A third study reported that the consumption of full-fat dairy, but not skimmed dairy, was associated with lower prevalence of knee OA[38]. In our DGI calculation, the dairy score was made up of 2 parts: 5 points were allocated for amounts of dairy products (any kind), and 5 for whether it was reduced fat[11]. Therefore, participants consuming full-fat dairy would get lower dairy scores than those consuming skimmed dairy; however, they may get beneficial effects on knee symptoms from consuming full-fat dairy.

We also found the change of score for limiting saturated fat was associated with lower stiffness and the change of score for fruit intake was associated with lower dysfunction. As diet was measured with a 24-h food record in adolescence and an FFQ in adulthood, it is not clear if the change scores represent the improvements of diet quality or are the results of different dietary measurement methods. Moreover, the sample size decreased in these analyses due to fewer participants having eligible diet measurements in both adolescence and adulthood. This means that we have less power to find statistically significant results, although some effect sizes in our results were relatively large (e.g. association between the score change for limiting saturated fat and knee pain). Therefore, further studies are needed to verify and interpret our results.

We observed that the significant associations between diet components and knee symptoms were largely evident in adulthood. This is in line with our previous study, where the effect of adult adiposity measures on knee structures in adults were much more evident than that of adolescent adiposity measures[39]. Adolescent diet could be changeable during growth, so it may only have a few residual effects. Moreover, the single 24-h food record may not fully reflect the daily adolescent diet.

Our study has some strengths. First, this study used the 25-year prospective data from adolescence to adulthood and this is the first study focusing on knee symptoms in adults who are important in knee OA prevention. Second, the use of DGI represented the adherence to the Australian dietary guidelines, and the core recommendations of these national guidelines are consistent with most dietary guidelines worldwide[40]. Third, our results have been adjusted for important potential confounders, including total energy intake and BMI. Some limitations of our study should be considered. First, the current sample size was determined by the available data from original cohort (ASHFS). A formal power calculation for sample size was not performed because this was a secondary analysis of the data collected in the main study. We only had a modest sample size due to the low proportion of participants who could be included in analyses. In particular, the adolescent dietary measures were only collected among adolescents aged 10–15 years, but not the whole cohort (aged 7–15 years). Participants in the current study were 1·8-year older and 0·8 kg/m^2 higher in BMI than those in the remainder of the ASHFS, whereas female proportions were comparable. Thus, the generalisability of our results may be limited, and further confirmatory studies are needed. Second, we only collected knee symptom data once, so we were unable to describe longitudinal changes in knee symptoms. Third, the dietary data were collected using different measurements (the 24-h food record for adolescence and the 127-item FFQ for adulthood), which necessitated different methods of scoring the DGI components. The different scoring methods may introduce bias in calculating the longitudinal changes of DGI. In adolescence, only a single 24-h food record was taken, whereas multiday food records may be more accurate, as they may be more reflective of daily diet. Reassuringly, this method has been suggested to be extremely valuable in collecting children's diet data despite its flaws[41]. In adulthood, the FFQ only collected data on frequency of food consumption and each frequency was assumed to be a standard serving, this may lead the inaccuracy in quantifying the food intake. However, the assignment of a constant portion size has been validated in epidemiological studies, though it may result in a reduction of interindividual variance[42]. Fourth, we did not collect the data regarding family history of knee OA and bleeding disorders history, which may have impacts in the development of knee OA, so we were unable to assess their potential effects on our findings.

In conclusion, the overall diet quality in adolescence and adulthood was not associated with knee symptoms in adulthood. However, some diet components such as limiting saturated fat in adulthood may affect later knee symptoms.

Acknowledgements

Special thanks to the participants who made this study possible. The roles of Liz O'Loughlin, Judy Hankin in collecting the data and Marita Dalton in managing the database are gratefully acknowledged.

This work was supported by the National Health and Medical Research Council of Australia (Grant number: 490006). The funders had no role in the design, analysis or writing of this article.

All authors participated in the study conception and design, acquisition of data, analysis and interpretation of data, preparation of manuscript, approved the manuscript for submission and publication and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

There are no conflicts of interest.

References

1. Kolasinski SL, Neogi T, Hochberg MC, *et al.* (2020) 2019 American College of Rheumatology/Arthritis foundation guideline for the management of osteoarthritis of the hand, hip, and knee. *Arthritis Care Res* **72**, 149–162.
2. Antony B, Jones G, Venn A, *et al.* (2015) Association between childhood overweight measures and adulthood knee pain, stiffness and dysfunction: a 25-year cohort study. *Ann Rheum Dis* **74**, 711–717.
3. Antony B, Jones G, Jin X, *et al.* (2016) Do early life factors affect the development of knee osteoarthritis in later life: a narrative review. *Arthritis Res Ther* **18**, 202.
4. Thomas MJ, Wood L, Selfe J, *et al.* (2010) Anterior knee pain in younger adults as a precursor to subsequent patellofemoral osteoarthritis: a systematic review. *BMC Musculoskelet Disord* **11**, 201.
5. Ros E, Martinez-Gonzalez MA, Estruch R, *et al.* (2014) Mediterranean diet and cardiovascular health: teachings of the PREDIMED study. *Adv Nutr* **5**, 330S–336S.
6. Daneshzad E, Dorosty-Motlagh A, Bellissimo N, *et al.* (2021) Food insecurity, dietary acid load, dietary energy density and anthropometric indices among Iranian children. *Eat Weight Disord* **26**, 839–846.
7. Daneshzad E, Moradi M, Maracy MR, *et al.* (2020) The association of maternal plant-based diets and the growth of breastfed infants. *Health Promot Perspect* **10**, 152–161.
8. Askari M, Daneshzad E, Bellissimo N, *et al.* (2021) Food quality score and anthropometric status among 6-year-old children: a cross-sectional study. *Int J Clin Pract* **75**, e14102.
9. Schulze MB & Hoffmann K (2006) Methodological approaches to study dietary patterns in relation to risk of coronary heart disease and stroke. *Br J Nutr* **95**, 860–869.
10. Gerber M (2001) The comprehensive approach to diet: a critical review. *J Nutr* **131**, 3051S–3055S.
11. Wilson JE, Blizzard L, Gall SL, *et al.* (2019) An age- and sex-specific dietary guidelines index is a valid measure of diet quality in an Australian cohort during youth and adulthood. *Nutr Res* **65**, 43–53.
12. Veronese N, Stubbs B, Noale M, *et al.* (2016) Adherence to the Mediterranean diet is associated with better quality of life: data from the Osteoarthritis Initiative. *Am J Clin Nutr* **104**, 1403–1409.
13. Veronese N, Stubbs B, Noale M, *et al.* (2017) Adherence to a Mediterranean diet is associated with lower prevalence of osteoarthritis: data from the osteoarthritis initiative. *Clin Nutr* **36**, 1609–1614.
14. Veronese N, Koyanagi A, Stubbs B, *et al.* (2018) Mediterranean diet and knee osteoarthritis outcomes: a longitudinal cohort study. *Clin Nutr* **38**, 2735–2739.
15. Willett WC, Sacks F, Trichopoulou A, *et al.* (1995) Mediterranean diet pyramid: a cultural model for healthy eating. *Am J Clin Nutr* **61**, 1402S–1406S.
16. Gall S, Jose K, Smith K, *et al.* (2009) The childhood determinants of adult health study: a profle of a cohort study to examine the childhood influences on adult cardiovascular health. *Australas Epidemiologist* **16**, 35–39.
17. English R, Cashel K, Lewis J, *et al.* (1988) *National Dietary Survey of School Children (aged 10–15 years): 1985. Report no. 1 – Foods Consumed*. Canberra: Australian Government Publishing Services.
18. National Health and Medical Research Council of Australia (2013) Eat for Health: Australian Dietary Guidelines. https://www.eatforhealth.gov.au/guidelines (accessed January 2021).
19. McConnell S, Kolopack P & Davis AM (2001) The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC): a review of its utility and measurement properties. *Arthritis Rheum* **45**, 453–461.
20. Heintjes EM, Bierma-Zeinstra SM, Berger MY, *et al.* (2008) Lysholm scale and WOMAC index were responsive in prospective cohort of young general practice patients. *J Clin Epidemiol* **61**, 481–488.
21. Hu MC, Pavlicova M & Nunes EV (2011) Zero-inflated and hurdle models of count data with extra zeros: examples from an HIV-risk reduction intervention trial. *Am J Drug Alcohol Abuse* **37**, 367–375.
22. Rose CE, Martin SW, Wannemuehler KA, *et al.* (2006) On the use of zero-inflated and hurdle models for modeling vaccine adverse event count data. *J Biopharm Stat* **16**, 463–481.
23. Jaworowska A, Blackham T, Davies IG, *et al.* (2013) Nutritional challenges and health implications of takeaway and fast food. *Nutr Rev* **71**, 310–318.
24. Krishnan S, Coogan PF, Boggs DA, *et al.* (2010) Consumption of restaurant foods and incidence of type 2 diabetes in African American women. *Am J Clin Nutr* **91**, 465–471.
25. Duffey KJ, Gordon-Larsen P, Steffen LM, *et al.* (2009) Regular consumption from fast food establishments relative to other restaurants is differentially associated with metabolic outcomes in young adults. *J Nutr* **139**, 2113–2118.
26. Fernandes GS & Valdes AM (2015) Cardiovascular disease and osteoarthritis: common pathways and patient outcomes. *Eur J Clin Invest* **45**, 405–414.
27. Wang Y, Davies-Tuck ML, Wluka AE, *et al.* (2009) Dietary fatty acid intake affects the risk of developing bone marrow lesions in healthy middle-aged adults without clinical knee osteoarthritis: a prospective cohort study. *Arthritis Res Ther* **11**, R63.
28. Lu B, Driban JB, Xu C, *et al.* (2017) Dietary fat intake and radiographic progression of knee osteoarthritis: data from the osteoarthritis initiative. *Arthritis Care Res* **69**, 368–375.
29. Loef M, Schoones JW, Kloppenburg M, *et al.* (2019) Fatty acids and osteoarthritis: different types, different effects. *Joint Bone Spine* **86**, 451–458.
30. Hill CL, March LM, Aitken D, *et al.* (2016) Fish oil in knee osteoarthritis: a randomised clinical trial of low dose versus high dose. *Ann Rheum Dis* **75**, 23–29.
31. Gruenwald J, Petzold E, Busch R, *et al.* (2009) Effect of glucosamine sulfate with or without *n*-3 fatty acids in patients with osteoarthritis. *Adv Ther* **26**, 858–871.
32. Schell J, Scofield RH, Barrett JR, *et al.* (2017) Strawberries improve pain and inflammation in obese adults with radiographic evidence of knee osteoarthritis. *Nutrients* **9**, 949.

33. Du C, Smith A, Avalos M, *et al.* (2019) Blueberries improve pain, gait performance, and inflammation in individuals with symptomatic knee osteoarthritis. *Nutrients* **11**, 290.
34. Carroll HA, Davis MG & Papadaki A (2015) Higher plain water intake is associated with lower type 2 diabetes risk: a cross-sectional study in humans. *Nutr Res* **35**, 865–872.
35. Courties A, Sellam J & Berenbaum F (2017) Metabolic syndrome-associated osteoarthritis. *Curr Opin Rheumatol* **29**, 214–222.
36. Kacar C, Gilgil E, Tuncer T, *et al.* (2004) The association of milk consumption with the occurrence of symptomatic knee osteoarthritis. *Clin Exp Rheumatol* **22**, 473–476.
37. Lu B, Driban JB, Duryea J, *et al.* (2014) Milk consumption and progression of medial tibiofemoral knee osteoarthritis: data from the Osteoarthritis Initiative. *Arthritis Care Res* **66**, 802–809.
38. Denissen KFM, Boonen A, Nielen JTH, *et al.* (2018) Consumption of dairy products in relation to the presence of clinical knee osteoarthritis: the Maastricht Study. *Eur J Nutr* **58**, 2693–2704.
39. Meng T, Venn A, Eckstein F, *et al.* (2019) Association of adiposity measures in childhood and adulthood with knee cartilage thickness, volume and bone area in young adults. *Int J Obes* **43**, 1411–1421.
40. Herforth A, Arimond M, Alvarez-Sanchez C, *et al.* (2019) A Global review of food-based dietary guidelines. *Adv Nutr* **10**, 590–605.
41. Foster E & Bradley J (2018) Methodological considerations and future insights for 24-hour dietary recall assessment in children. *Nutr Res* **51**, 1–11.
42. Noethlings U, Hoffmann K, Bergmann MM, *et al.* (2003) Portion size adds limited information on variance in food intake of participants in the EPIC-Potsdam study. *J Nutr* **133**, 510–515.