International Journal of Public Health (2018) 63:883–893
https://doi.org/10.1007/s00038-018-1100-z

ORIGINAL ARTICLE

Diet and physical activity as possible mediators of the association between educational attainment and body mass index gain among Australian adults

Emma Gearon[1,2] · Anna Peeters[1,2] · Winda Ng[1,2,3] · Allison Hodge[4,5] · Kathryn Backholer[1,2]

Received: 10 November 2017 / Revised: 19 March 2018 / Accepted: 6 April 2018 / Published online: 23 April 2018
© Swiss School of Public Health (SSPH+) 2018

Abstract

Objectives To quantify the mediating role of leisure time physical activity (LTPA) and five dietary behaviours on educational differences in 13-year body mass index (BMI) gain across adulthood.

Methods Participants from the Melbourne Collaborative Cohort Study (4791 women; 3103 men) who maintained or gained BMI over 1990–1994 to 2003–2007 and met our inclusion criteria were selected. Education, potential mediators and confounders (age, alcohol, and smoking) were measured at baseline. We conducted sex-specific multiple mediation analyses using MacKinnon's product of coefficients method.

Results A higher educational attainment was associated with a 0.27 kg m^{-2} (95% CI 0.14, 0.39) lesser 13-year BMI gain among women only. We observed significant indirect effects of educational attainment on 13-year BMI gain through LTPA and nutrient-rich foods (each associated with a higher educational attainment and lesser 13-year BMI gain) and diet soft drink (associated with a lower educational attainment and greater 13-year BMI gain), which mediated 10, 15 and 20% of this relationship, respectively (45% in total).

Conclusions Nutrient-rich foods, LTPA and diet soft drink may represent effective public health targets to reduce inequities in excess weight across adulthood.

Keywords Mediation analysis · Socioeconomic factors · Diet, food, and nutrition · Physical activity · Obesity · Longitudinal studies

Introduction

Obesity and its sequelae are leading contributors to the global burden of disease (Forouzanfar et al. 2016), with direct costs in excess of $3.8 billion per year in Australia (PwC 2015). As such, the prevention and treatment of obesity is a public health priority. Action to prevent and treat excess weight gain is important across the lifespan. In particular, maintaining a healthy weight across adulthood has been shown to reduce the risk of disability (Backholer et al. 2012b), dementia (Tolppanen et al. 2014) and cardio-metabolic outcomes (Xian et al. 2017; Zheng et al. 2017) later in life.

For adults in high income countries, those of a lower socioeconomic position (SEP) are more likely to be obese compared to their counterparts of a higher SEP (Backholer et al. 2012a; Ball and Crawford 2005; Gearon et al. 2015). These socioeconomic inequalities in obesity are likely to contribute to socioeconomic inequalities in morbidity and mortality (Peeters and Backholer 2015).

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00038-018-1100-z) contains supplementary material, which is available to authorized users.

✉ Emma Gearon
Emma.Gearon@deakin.edu.au

[1] Global Obesity Centre, Deakin University, Locked Bag 20000, Geelong, VIC 3220, Australia

[2] School of Public Health and Preventive Medicine, Monash University, Melbourne, Australia

[3] Clinical Diabetes and Epidemiology, Baker Heart and Diabetes Institute, Melbourne, Australia

[4] Cancer Epidemiology and Intelligence Division, Cancer Council Victoria, Melbourne, VIC, Australia

[5] Centre for Epidemiology and Biostatistics, University of Melbourne, Parkville, VIC, Australia

Diet and physical activity are potentially modifiable energy balance behaviours, and are key risk factors for weight gain and obesity (Government Office for Science 2007; Mozaffarian et al. 2011). Furthermore, adults with a lower SEP are more likely to engage in lower levels of leisure time physical activity (LTPA) and consume greater amounts of foods and beverages associated with weight gain, compared to their counterparts with a higher SEP (Beenackers et al. 2012; Darmon and Drewnowski 2008; Turrell et al. 2009).

The extent to which socioeconomic inequalities in weight across adulthood can be attributed to specific health behaviours has been examined in a limited number of cross-sectional studies (Compernolle et al. 2016; Gearon et al. 2013; Kavanagh et al. 2010; Miura and Turrell 2014; Molarius 2003; Wamala et al. 1997; Ward et al. 2007). These studies have demonstrated that the combined influence of (a limited number of) health behaviours on the relationship between SEP and weight outcomes mediates 12% to 51% of this relationship for women (Compernolle et al. 2016; Gearon et al. 2013; Kavanagh et al. 2010; Molarius 2003; Wamala et al. 1997; Ward et al. 2007), and, where a significant relationship between SEP and weight outcomes was observed, 22–27% of this relationship for men (Gearon et al. 2013; Kavanagh et al. 2010; Molarius 2003). However, these studies tend to rely on self-reported measures of weight outcomes, few have decomposed and quantified the different elements of energy balance behaviours, and all studies are limited by the cross-sectional nature and thus the possibility of reverse causation between mediators and weight outcomes. To inform public policies that can effectively and equitably reduce obesity, potential leverage points for intervention that contribute to the observed socioeconomic differences in weight gain must be identified (Peeters and Backholer 2015). To the best of our knowledge, no study to date has examined the extent to which socioeconomic differences in weight gain across adulthood can be attributed to specific energy balance behaviours.

The aim of this study was to identify the extent to which energy balance behaviours mediate the relationship between educational attainment and 13-year gain in body mass index (BMI) among Australian adult women and men who participated in the Melbourne Collaborative Cohort Study (MCCS).

Method

Data source

The MCCS is a prospective cohort study of 41 513 adults aged 27–80 who were recruited from Melbourne between 1990 and 1994 using telephone books and electoral rolls, as well as community announcements and advertisements. While there was no exclusion criteria at baseline, efforts focused on recruiting healthy volunteers aged 40–69 years. An arm of the recruitment drive specifically focused on migrants from Southern Europe in an attempt to explore a wide range of genetic and lifestyle factors, and individuals born in Italy and Greece comprised 24% of the baseline population (Milne 2017). All participants who had not died or withdrawn from the study were encouraged to attend a face-to-face follow-up in 2003–2007, and 26,984 (68% follow-up) (Milne 2017) attended the follow-up clinic. At baseline, all participants were interviewed face-to-face and administered a 121-item food frequency questionnaire (FFQ) which was designed for the study and based on data from a group with similar demographic characteristics to the MCCS (Ireland et al. 1994). At both baseline and follow-up, a practitioner measured weight using Soehnle teleterminal digital electronic scales (to the nearest 0.1 kg) and waist circumference (WC) using a two metre anthropometric tape (to the nearest mm) (Cancer Epidemiology Centre 2008; Lohman et al. 1988). Height was measured at baseline only using a wall-mounted stadiometer (to the nearest mm) (Cancer Epidemiology Centre 2008; Lohman et al. 1988). Subjects removed shoes and outer clothing for all measurements.

Variables

All outcome variables used information from baseline and follow-up, and all exposures, mediating and confounding variables were measured at baseline only. All continuous variables were visually examined for normality, and categorised where appropriate.

Continuous measures of 13-year gain in BMI (kg m^{-2}; outcome variable) and WC (cm; outcome variable in secondary analysis) were calculated by subtracting baseline measurements from follow-up measurements. In line with a number of previous studies (Ball and Crawford 2005; Feng and Wilson 2015), we examined gain in BMI rather than using weight, as any gain in weight will be differently important depending on the height.

Highest educational attainment (exposure variable), dichotomised at completion of secondary school, was used to indicate higher and lower SEP.

Six mediators (five dietary mediators and one physical activity mediator) of interest were examined at baseline and are defined for our analysis in Table 1. All mediators were selected based on variables known to be both socioeconomically patterned (Darmon and Drewnowski 2008) and associated with weight change (Mozaffarian et al. 2011), and grouped in a manner reflecting potential public health intervention targets (Table 1). The dietary

Table 1 Definition of all mediating variables for analyses, Melbourne Collaborative Cohort Study, Australia, 1990–1994

Mediator	Unit	Composition
Leisure time physical activity	Score	Participant's average weekly frequency over the prior 6 months of participating in vigorous and less-vigorous leisure time physical activity and walking for at least 20 min was converted into a continuous score from zero to sixteen, where frequency of vigorous leisure time physical activity was given double the weighting of frequency of less-vigorous leisure time physical activity or walking
		A higher score reflects a higher frequency and intensity of leisure time physical activity
Diet soft drink	0 "< monthly" 1 "≥ monthly"	Participant's monthly frequency of consuming diet soft drink, dichotomised as "never or less than once per month" or "once per month or more"
Soft drink	0 "< weekly" 1 "≥ weekly"	Participant's weekly frequency of consuming soft drink, dichotomised as "never or less than once per week" or "once per week or more"
Nutrient-rich foods	Times per week	Continuous sum of the following foods, measured in weekly frequency:
		Yoghurt
		Nuts
		Wholemeal bread, rolls, toast
		Wheat germ
		Muesli
		Chicken, boiled or steamed
		Fish, boiled or steamed
Fruit and vegetables	Times per day	Daily frequency of consuming of all fresh fruits and vegetables, with the exception of potato
Discretionary foods	Times per week	Continuous sum of the following foods, measured in weekly frequency:
		Cakes and sweet pastries
		Confectionary
		Chocolate
		Sweet biscuits
		White bread
		Pies and savoury pastries
		Dim sims and spring rolls
		Pizza
		Corn and potato chips
		Fried chicken
		Fried potatoes
		Sausages or frankfurters
		Salami or continental sausage
		Manufactured luncheon meats
		Corned beef

mediators included: frequency of intake of diet soft drink, soft drink, nutrient-rich foods, fruits and vegetables, and discretionary foods. LTPA was measured using a score based on participant's average weekly frequency and intensity of participation in LTPA and frequency of walking (MacInnis et al. 2004), where a higher score reflected higher levels of LTPA. All mediators were approximately normally distributed and used as continuous variables, with the exception of soft drink and diet soft drink. Because a large proportion of participants (73%) reported consuming diet soft drinks 'never or less than once per month', we dichotomised this variable at this consumption frequency. The resulting diet soft drink variable was thus categorised as 'never or less than once per month' (< monthly) or 'once per month or more' (≥ monthly). Average soft drink consumption was more

frequent, and we categorised this variable as 'never or less than once per week' (< weekly) or 'once per week or more' (≥ weekly).

Potential confounders of exposure–outcome, exposure–mediator, and mediator–outcome relationships included: smoking status at baseline (categorised as never, former and current), baseline alcohol intake [dichotomised at meeting current Australian guidelines (20 g per day for women and men (National Health and Medical Research Council 2009))] and age (continuous years). Confounders were identified based on existing literature (Darmon and Drewnowski 2008; Mozaffarian et al. 2011), and through use of directed acyclic graphs created using DAGitty software (Textor et al. 2011) (Online Resource 1).

Exclusion criteria

Participants were ineligible for this analysis, if they did not attend the follow-up clinic (n = 14,529), had a southern European country of birth (n = 5171; due to intentional oversampling of participants of southern European ethnicity (Milne 2017) and strong correlation between educational attainment and country of birth (Gearon et al. 2013), if they were aged > 65 years at follow-up (n = 11,280; to account for the inaccuracy of BMI at older age as a measure of body fat (Prentice and Jebb 2001), if they reported having cancer or cardio-metabolic diseases or conditions at baseline (n = 1762; defined as defined as all cancers, hypertension, angina, diabetes, heart attack or stroke, to account for possible moderating role of diseases and conditions on socioeconomic differences in weight), or if they lost weight over time [n = 687; defined as a 5% or greater BMI loss from baseline, to account for the unknown aetiology of weight loss in this population (Mozaffarian et al. 2011)].

Participants were additionally excluded from analyses, if they had extreme energy intakes [n = 98; defined as being in the 1st or 99th percentile of energy intake (Hodge et al. 2011)] or were missing information on variables of interest (n = 92). Our final analytical population comprised 4791 women and 3103 men.

Statistical analyses

All analyses were stratified by sex. Descriptive statistics (means or proportions) were calculated for BMI, and all potential mediators and confounders, stratified by educational attainment.

We used the product of coefficients mediation method to assess six mediating pathways of the relationship between educational attainment and 13-year BMI gain. This method utilises a series of regression analyses, all of which are adjusted for potential confounders (age, smoking status and alcohol intake), to yield the total effect of educational attainment on 13-year BMI gain, the 6 indirect effects of educational attainment on 13-year BMI gain through each potential mediator, and the total indirect effect of educational attainment on 13-year BMI gain through all significant mediators. Because the significance of the indirect effect does not depend on the significance of the total effect (MacKinnon 2008), all indirect effects were tested. Stata's binary_mediation command was used to estimate the total effect, indirect effects and total indirect effects, as it accounts for the differing scales of the linear and logit models by standardising all beta-coefficients using the standard deviation of the predictor and outcome (MacKinnon 2008; Winship and Mare 1983). All estimates were bootstrapped with 5000 replications to attain their respective 95% confidence intervals (95% CI's).

Significant mediators were identified as being (1) significantly associated with educational attainment (significant exposure–mediator relationship), calculated using linear regression (continuous mediators) or logit regression (dichotomous mediators, presented as odds ratios in text for ease of interpretation), and (2) significantly associated with 13-year BMI gain, independent of educational attainment and the other potential mediators (significant mediator–outcome relationship), calculated using linear regression. Where a significant total effect was observed, we then calculated the standardised indirect effect for each significant mediator as the product of the standardised beta-coefficients from the exposure–mediator and the mediator–outcome relationships. The standardised total indirect effect was calculated as the sum of all significant indirect effects. The proportion of the standardised total effect which was mediated by each significant mediator (or all significant mediators) was calculated by dividing the standardised indirect effect (or the standardised total indirect effect) by the standardised total effect. All proportions were calculated by bootstrapping standardised values, and are reported to one decimal place. So, that each of the relationships that we examined can be interpreted, we present non-standardised coefficients and odds ratios in tables and text but use standardised coefficients to calculate the proportion mediated by each significant mediator. All significant standardised effects are reported (Online Resource 2).

All assumptions of the product of coefficients mediation method were tested and met: a linear relationship between exposure and outcome, exposure and all continuous mediators, and all continuous mediators and outcome; no exposure–mediator interactions; and no correlation between the predictor variable and residuals for any relationship tested.

Five sensitivity analyses were performed: (1) incident obesity as an outcome, (2) adjusting for changes in

smoking status, (3) restricting analyses to never smokers, (4) restricting analyses to those free from chronic diseases at follow-up, (5) 13-year waist circumference gain as an outcome. The details of these analyses are described in Online Resource 3.

Ethics approval for the current study was obtained through the Monash University Human Research Ethics Committee; CF15/21–2015000018, and through Deakin University Human Research Ethics Committee; 2016-0141.

Results

Summary statistics

For women and men, the mean (SD) BMI at baseline was 24.7 kg m^{-2} (4.0) and 26.1 kg m^{-2} (3.2), respectively. The corresponding mean (SD) gain in BMI over the 13-year of follow-up was 2.0 kg m^{-2} (2.2) and 1.3 kg m^{-2} (1.6).

Compared to their counterparts with a higher educational attainment, women and men with a lower educational attainment had a higher BMI at baseline and follow-up, a greater gain in BMI over 13-year (women only), and at baseline: a higher proportion meeting alcohol consumption guidelines (women only), a lower proportion of never smokers, a lower LTPA score (men only), a lower reported frequency of consuming nutrient-rich foods, fruits and vegetables, and a higher reported frequency of consuming diet soft drink ≥ monthly, soft drink ≥ weekly and discretionary foods (Table 2).

Total effect of educational attainment on 13-year BMI gain

For women, a higher educational attainment was significantly associated with 0.27 kg m^{-2} (95% CI 0.14, 0.39) lesser BMI gain over 13 years compared to a lower educational attainment. For men, there was no significant relationship between educational attainment and 13-year gain in BMI [0.06 kg m^{-2} (95% CI − 0.08, 0.20)].

Exposure–mediator relationship

For both women and men, educational attainment was significantly associated with all potential mediators (Table 3). For women, compared to those with a lower educational attainment, women with a higher educational attainment had a higher frequency and intensity of LTPA, a higher frequency of consuming nutrient-rich foods and fruits and vegetables, and a lower frequency of consuming discretionary foods. Furthermore, women with a higher educational attainment were less likely to consume diet soft drink ≥ monthly or soft drink ≥ weekly. For men, we observed the same direction of association and a similar magnitude of association between educational attainment and each mediator.

Mediator–outcome relationship

For women, a higher LTPA score and higher frequency of consuming nutrient-rich foods was significantly associated with a lesser 13-year BMI gain [− 0.04 kg m^{-2} (95% CI − 0.05, − 0.02) and − 0.01 kg m^{-2} (95% CI − 0.02, − 0.01)], respectively, and consuming diet soft drink ≥ monthly was significantly associated with a greater 13-year BMI gain [0.52 kg m^{-2} (95% CI 0.38, 0.67)] (Table 3). Consuming soft drink ≥ weekly, and frequency of consuming fruits and vegetables and discretionary foods were not significantly associated with 13-year BMI gain for women, independent of educational attainment, all other mediators, and potential confounders.

For men, consuming diet soft drink ≥ monthly was significantly associated with 13-year BMI gain [0.28 kg m^{-2} (95% CI 0.15, 0.43)] (Table 3). LTPA score, consuming soft drink ≥ weekly, and frequency of nutrient-rich foods, fruits and vegetables and discretionary foods were not significantly associated with 13-year BMI gain for men, independent of educational attainment, all other mediators, and potential confounders.

Indirect effect of educational attainment on 13-year BMI gain through significant mediators

For women, we observed a significant indirect effect of educational attainment on 13-year BMI gain through LTPA score, consuming diet soft drink ≥ monthly, and frequency of consuming nutrient-rich foods, which mediated 10.2, 20.1 and 14.7% of this relationship, respectively. Combined, LTPA score, consuming diet soft drink ≥ monthly, and frequency of consuming nutrient-rich foods mediated 45.0% of the relationship between educational attainment and 13-year BMI gain (Table 3).

For men, we observed significant exposure–mediator and mediator–outcome relationships for consuming diet soft drink ≥ monthly, however, because the total effect was not significant, the indirect effect and proportion mediated were not calculated.

Sensitivity analyses

Results for all sensitivity analyses are presented in Table 4 and in Online Resource 4.

Table 2 Summary characteristics of the Melbourne Collaborative Cohort Study analytic population by sex and educational attainment, Australia, 1990–1994 to 2003–2007

	Women		Men	
	Lower education Mean (SD), *N* (%) or median (IQR)	Higher education Mean (SD), *N* (%) or median (IQR)	Lower education Mean (SD), *N* (%) or median (IQR)	Higher education Mean (SD), *N* (%) or median (IQR)
Analytic population (*N*)	1864	2927	721	2382
Baseline BMI (kg m^{-2})	25.3 (4.2)	24.4 (3.8)	27 (3.4)	25.8 (3.1)
Follow-up BMI (kg m^{-2})	27.4 (5.0)	26.2 (4.7)	28.4 (4)	27.1 (3.6)
13-year gain in BMI (kg m^{-2})	2.1 (2.2)	1.9 (2.1)	1.4 (1.8)	1.3 (1.6)
Baseline age (years)	47.0 (4.0)	46.1 (4.0)	46.7 (3.9)	46.2 (4.1)
Alcohol (% meeting guidelines)	1671 (90)	2444 (84)	497 (69)	1618 (68)
Never smoker	1138 (61)	1869 (64)	328 (45)	1407 (59)
Former smoker	494 (27)	843 (29)	289 (40)	752 (32)
Current smoker	232 (12)	215 (7)	104 (14)	223 (9)
Leisure time physical activity (score)	4.0 (1.5, 5.5)	4.0 (1.5, 7.0)	4.0 (1.5, 8.0)	4.5 (1.5, 8.0)
Diet soft drink (% $\geq$ monthly)	626 (34)	737 (25)	206 (29)	580 (24)
Soft drink (% $\geq$ weekly)	542 (29)	622 (21)	382 (53)	1000 (42)
Nutrient-rich foods (times per week)	8.5 (4.0, 18.0)	12.5 (7.5, 21.0)	7.0 (2.0, 13.5)	9.5 (4.5, 18.5)
Fruits and vegetables (times per day)	7.4 (5.1, 10.0)	8.3 (6.0, 11.1)	5.4 (3.4, 7.6)	6.5 (4.5, 8.9)
Discretionary foods (times per week)	16.0 (9.5, 25.5)	12.5 (7.5, 21.0)	21.5 (13.0, 33.0)	16.5 (11.0, 26.0)

BMI body mass index (kg m^{-2})

Discussion

Three potentially modifiable energy balance behaviours mediated almost half of the differences in 13-year BMI gain between women with a higher and lower educational attainment who participated in the MCCS. More frequent and intense LTPA (as indicated through a higher LTPA score) and more frequent consumption of nutrient-rich foods were both more common among women with a higher educational attainment and associated with a lesser BMI gain. Consuming diet soft drink once per month or more was both more common among women with a lower educational attainment and associated with a greater BMI gain. These three behaviours accounted for 10, 15 and 20% of the educational differences in 13-year BMI gain, respectively. The strength and direction of this mediation remained relatively consistent in sensitivity analyses. For men, whilst we observed no relationship between educational attainment and BMI gain in the primary analysis, we did observe significant educational differences in incident obesity, which were also mediated by LTPA (5%), intake of nutrient-rich foods (12%) and diet soft drink consumption (7%).

We observed educational differences in BMI at baseline for both men and women; however, differences in BMI gain across the 13-year study period were only observed for women. This is consistent with the literature (Ball and Crawford 2005; Feng and Wilson 2015). For example, an analysis of > 20,000 Australian adults demonstrated socioeconomic differences in BMI widened across adulthood for women but not men (Feng and Wilson 2015). For men, socioeconomic differences in BMI were established prior to adulthood (Feng and Wilson 2015). However, we did observe socioeconomic differences in 13-year BMI gain for men who were never smokers, and socioeconomic differences in incident obesity for men.

The relationships identified in this study demonstrating a higher frequency and intensity of LTPA and higher consumption of nutrient-rich foods with a lesser BMI gain, and higher frequency of these behaviours among those of a higher SEP, are well established in the literature (Beenackers et al. 2012; Darmon and Drewnowski 2008; Government Office for Science 2007; Mozaffarian et al. 2011). Similarly, the positive relationship between SEP and the prevalence of diet soft drink consumption identified in this study is consistent with the most recent national nutrition survey in Australia (Australian Bureau of Statistics 2011–2012). The literature examining the relationship between consuming diet soft drink and a greater BMI gain, however, is somewhat inconsistent. Consumption of non-caloric sweetened beverages has been found to be associated with both weight gain and weight loss across a number of prospective cohort studies (Rogers et al. 2016). The association between diet soft drink consumption and weight gain is frequently attributed to reverse causation (Fowler et al. 2008), as diet soft drink consumption may

Table 3 Relationships between educational attainment (exposure) and all potential mediators, and all potential mediators and 13-year gain in body mass index (outcome), and the proportion of the total effect of educational attainment on 13-year body mass index gain mediated by each significant mediator, Melbourne Collaborative Cohort Study, Australia, 1990–1994 to 2003–2007

Mediator	Sex	Exposure–mediator relationship[a] Beta-coefficient (95% CI)	Mediator–outcome relationship Beta-coefficient (95% CI)	Proportion mediated
Continuous mediating variables				
Leisure time physical activity (score)	W	0.77 (0.54, 0.98)*	− 0.04 (− 0.05, − 0.02)*	10.2 (4.6, 21.6)
	M	0.72 (0.36, 1.05)*	− 0.01 (− 0.02, 0.00)	
Nutrient-rich foods (times per week)	W	3.50 (2.91, 4.09)*	− 0.01 (− 0.02, − 0.01)*	14.7 (6.2, 31.1)
	M	2.92 (2.09, 3.76)*	0.00 (− 0.01, 0.00)	
Fruits and vegetables (times per day)	W	1.05 (0.77, 1.33)*	− 0.01 (− 0.02, 0.01)	
	M	1.22 (0.91, 1.53)*	0.00 (− 0.02, 0.01)	
Discretionary foods (times per week)	W	− 3.49 (− 4.27, − − 2.72)*	0.00 (0.00, 0.01)	
	M	− 5.21 (− 6.56, − 3.83)*	0.00 (0.00, 0.00)	
		Odds ratio (95% CI)	Beta-coefficient (95% CI)	
Dichotomous mediating variables				
Diet soft drink (< monthly/≥ monthly)	W	0.65 (0.57, 0.74)*	0.52 (0.38, 0.67)*	20.1 (11.1, 40.3)
	M	0.80 (0.66, 0.96)*	0.28 (0.15, 0.43)*	
Soft drink (< weekly/≥ weekly)	W	0.64 (0.56, 0.73)*	0.01 (− 0.14, 0.16)	
	M	0.63 (0.53, 0.75)*	− 0.02 (− 0.14, 0.10)	

We present non-standardised beta-coefficients and odds ratios, and the proportion mediated as calculated from the product of standardised coefficients

W women, *M* men

*Denotes significant relationship ($p < 0.05$)

[a]A lower educational attainment is the reference category

represent either dieting behaviour, which has been associated with weight gain (Lowe 2015), or a clustering of unhealthy behaviours. Alternatively, non-caloric sweeteners may directly cause weight gain by driving subsequent food intake and metabolic changes (Fowler 2016; Murray et al. 2016; Shearer and Swithers 2016), increasing preference for other sweet products (Burke and Small 2015), or by causing imbalances in the composition of microbiota within the gut which increase the risk of obesity (Burke and Small 2015; Suez et al. 2014). Our finding that consuming diet soft drink once per month or more mediates the relationship between educational attainment and 13-year BMI gain is robust to all our included sensitivity analyses, and further studies will be required to disentangle the causality of the relationship between diet soft drink consumption and weight gain.

In contrast to a number of other high quality studies (Hu 2013; Mozaffarian 2016; World Cancer Research Fund/American Institute for Cancer Research 2007; World Health Organization 2003), we did not observe an association between consumption of soft drink (once per week or more) or discretionary food intake (continuous weekly frequency) with a greater BMI gain in our analyses. The lack of relationship between these diet variables and 13-year BMI gain may relate to insufficient variation across the population to identify a relationship. Alternatively, as the dietary behaviours in our study were examined at baseline and BMI gain was measured 13 years later, it is possible that participants had changed their dietary behaviours over this time period (Prynne et al. 2005), particularly for consumption of soft drinks which have been found to be decreasing in the Australian population (Australian Bureau of Statistics 2014).

To the best of our knowledge, this is the first longitudinal analysis of the mediating role of potentially modifiable energy balance behaviours on the relationship between SEP and BMI gain across adulthood. Whilst a number of cross-sectional studies have examined the contribution of health behaviours to the relationship between SEP and weight outcomes (Compernolle et al. 2016; Gearon et al. 2013; Kavanagh et al. 2010; Miura and Turrell 2014; Molarius 2003; Wamala et al. 1997; Ward et al. 2007), few have decomposed and quantified the different elements of energy balance behaviours. Nonetheless, these cross-sectional studies have found that combinations of energy balance behaviours, such as takeaway foods (Miura and

Table 4 Key results for sensitivity analyses and when using 13-year waist circumference gain as the outcome variable, Melbourne Collaborative Cohort Study, Australia, 1990–94 to 2003–07

	Sex	Incident obesity	Adjusting for change in smoking status	Never smokers	No known diseases or conditions at follow-up	13-year WC gain used as outcome
Analytic population (*N*)						
	W	4302	4791	3007	3794	5005
	M	2768	3103	1735	2337	3143
Total effect of educational attainment on incident obesity [odds ratio (95% CI)], 13-year BMI gain (kg m^{-2}) or 13-year WC gain (cm) [beta-coefficient (95% CI)]						
	W	0.71 (0.59, 0.86)*	− 0.27 (− 0.4, − 0.15)*	− 0.3 (− 0.46, − 0.14)*	− 0.22 (− 0.37, − 0.08)*	− 0.83 (− 1.26, − 0.41)*
	M	0.68 (0.52, 0.90)*	− 0.07 (− 0.21, 0.07)	− 0.19 (− 0.38, − 0.01)*	− 0.02 (− 0.19, 0.15)	− 0.36 (− 0.92, 0.21)
Proportion of the total effect mediated through each mediator with significant exposure–mediator and mediator–outcome relationships						
Leisure time physical activity (score)	W	6.5 (0.8, 17.6)	10.4 (4.8, 21.4)		8.1 (2.7, 22.3)	8.4 (3.0, 19.4)
	M	5.3 (0.4, 20.7)				
Diet soft drink (< monthly/≥ monthly)	W	24.2 (13.1, 53.2)	19.8 (11.0, 38.8)	20.6 (10.4, 43.1)	26.6 (12.9, 72.0)	20.8 (11.3, 42.4)
	M	6.7 (0.4, 23.6)		10.7 (− 0.6, 59.8)		
Soft drink (< weekly/≥ weekly)	W					
	M					
Nutrient-rich foods (times per week)	W	23.0 (10.8, 53.7)	14.5 (6.2, 30.3)	9.7 (1.6, 25.6)	17.2 (6.4, 48.8)	
	M	11.8 (1.0, 50.6)				
Fruits and vegetables (times per week)	W			4.8 (0.6, 12.7)		
	M					
Discretionary foods (times per week)	W					
	M					
Proportion of the total effect mediated through the sum of each significant mediator (total indirect effect)						
	W	53.7 (32.0, 116.1)	44.7 (27.9, 83.4)	35.1 (19.5, 72.7)	51.9 (28.3, 132.7)	29.1 (16.8, 58.5)
	M	23.8 (9.1, 84.3)		10.7 (− 0.6, 59.8)		

We present the non-standardised total effect, and the proportion mediated as calculated from the product of standardised coefficients

WC waist circumference, *W* women, *M* men

*Denotes significant relationship ($p < 0.05$)

Turrell 2014), fruit and vegetable consumption (Compernolle et al. 2016; Gearon et al. 2013; Ward et al. 2007), macronutrient (Molarius 2003; Wamala et al. 1997) or total energy (Kavanagh et al. 2010; Wamala et al. 1997) intake, LTPA (Compernolle et al. 2016; Gearon et al. 2013; Kavanagh et al. 2010; Molarius 2003; Wamala et al. 1997; Ward et al. 2007), transport-related physical activity (Compernolle et al. 2016) and sedentary time (Compernolle et al. 2016; Proper et al. 2007) mediate between 12 and 51% of this relationship for women, and where a significant relationship between SEP and weight outcomes was observed, 22–27% of this relationship for men (Gearon et al. 2013; Kavanagh et al. 2010; Molarius 2003).

We found that the energy balance behaviours in our analysis accounted for 45% of the difference in 13-year BMI gain among women with lower and higher educational attainment. The remaining 55% may be attributable to other factors known to be both socioeconomically patterned and associated with BMI that were not included in this study. These include individual-level factors such as sedentary time (Compernolle et al. 2016; Proper et al. 2007), sleep duration (Zimberg et al. 2012), and reproductive factors (Wamala et al. 1997), social factors such as psychosocial stress (Moore and Cunningham 2012) and social capital, and environmental level factors such as street connectivity, requirement for a car (Turrell et al. 2013) and green spaces (Sanders et al. 2015). It is also possible that error in our measures of dietary factors and LTPA are limited by measurement bias, whereby survey questions only assessed the frequency of a given behaviour (and not quantity) and were only measured at one point in time (baseline).

The measurement of dietary and LTPA behaviours at baseline only (the frequency of these variables were not measured at follow-up, thus precluding inclusion for this analysis), with 13 years of follow-up, is a major limitation of this study. Smith et al. (2015) demonstrate that while average change over time in dietary and LTPA behaviours is small, the most biologically plausible, robust and consistent results are observed, when dietary change and weight change are examined concurrently. Conversely, this small average change in dietary and LTPA behaviours over time may present a challenge to identify significant exposure–mediator and mediator–outcome relationships.

In addition this study is also limited by the following: first, baseline data for the MCCS, collected in 1990–1994, is now relatively dated and the prevalence of diet and physical activity behaviours in this study may not reflect current population trends; however. the internal relationships are likely to remain valid. Second, the voluntary nature of the MCCS and cohort attrition over time (Milne 2017) increases the possibility of self-selection bias, which may limit the generalisability of the findings. Third, our grouping of dietary variables was informed by the literature, but it is possible that the individual dietary components within each group may be differentially associated with 13-year BMI gain, which would reduce the strength of associations. Fourth, our dietary variables were limited to what could be derived from the FFQ in the MCCS, and there may be other important components of the diet which have not been examined in this analysis. Fifth, although we accounted for key confounders and mediators in our analysis (age, smoking and alcohol) residual confounding by other unknown and unmeasured risk factors may influence our results. One way that the impact of unmeasured confounders is quantified in the literature is through calculating a bias correction factor (Ding and Vanderweele 2016). Unfortunately, we were unable find an appropriate method to calculate a bias correction factor for standardised coefficients, and were therefore unable to evaluate the impact of residual confounding on the mediation results. Six, our use of the binary_mediation Stata command necessitated dichotomising our exposure variable, educational attainment, and we are unable to disentangle drivers of socioeconomic differences in 13-year BMI gain across the socioeconomic spectrum. Finally, we excluded participants with a Southern European country of birth from our analysis, because of the intentional oversampling of these participants in the MCCS cohort and strong correlation between Southern European country of birth and educational attainment. It is possible that the drivers of socioeconomic inequalities in BMI gain may differ across ethnic groups as a result of differences in cultural norms around dietary intake and physical activity.

The primary strength of this study is the prospective nature, which allows an examination of the mediators of socioeconomic inequalities in BMI gain, and reduces the likelihood of reverse causation. Further strengths of the study, which improve the reliability and internal validity of results, include the objective measurement of height and weight by trained interviewers, and the reporting of dietary variables using an extensive and validated FFQ specifically designed for the study. Dietary mediators examined were additionally grouped to reflect potential public health intervention targets, and as such, findings are relevant to current discourse on where to intervene to effectively and equitably reduce obesity. Further, SEP was measured using educational attainment, which is less susceptible to reverse causality compared to other individual-level markers, and is considered a reliable marker of SEP for both women and men (Galobardes et al. 2006).

This study quantified the mediating role of diet and physical activity behaviours on socioeconomic inequalities in 13-year weight gain. Our findings indicate that encouraging LTPA and consumption of nutrient-rich foods may reduce socioeconomic differences in BMI gain across

adulthood. Additionally, our findings suggest that diet soft drink consumption may also be a driver of socioeconomic differences in BMI gain across adulthood. Further evidence incorporating, long-term dietary change, in contemporary cohorts is required to elucidate these relationships further. If our results are confirmed, interventions and policies to address these individual-level behaviours will need to be multifaceted and include a variety of strategies across the individual, social and environmental level (Peeters and Blake 2016).

Acknowledgements This work was supported by an Australian Research Council (ARC) Linkage grant (LP120100418) and in part by the Victorian Government's Operational Infrastructure Support (OIS) Program. EG was supported by an Australian Government Research Training Program (RTP) Scholarship, AP was supported by a National Health and Medical Research Council Career Development Fellowship (1045456) and is a researcher within the NHMRC Centre for Research Excellence in Obesity Policy and Food Systems (APP1041020) and Deakin University, WLN is supported by a Monash Graduate Scholarship, a Monash International Post-graduate Research Scholarship and a Baker Bright Sparks Top-Up Scholarship, and KB was supported by a National Heart Foundation of Australia Post-Doctoral Fellowship (PH 12M6824). The MCCS study was made possible by the contribution of many people, including the original investigators and the diligent team who recruited the participants and completed follow-up. We would like to express our gratitude to the many thousands of Melbourne residents who continue to participate in the study. MCCS Data used in this research were obtained from Cancer Council Victoria. MCCS cohort recruitment was funded by VicHealth and Cancer Council Victoria. The MCCS was further supported by Australian NHMRC grants 209057 and 396414 and by infrastructure provided by Cancer Council Victoria. These funding sources had no input into study design, or collection, analysis and interpretation of the data.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent Informed consent was obtained from all individual participants included in the study.

References

Australian Bureau of Statistics (2011–2012) Australian Health Survey, Nutrition and Physical Activity, 2011–12, Table Builder. Findings based on use of ABS TableBuilder data

Australian Bureau of Statistics (2014) Australian health survey: nutrition first results - foods and nutrients, 2011–12. Australian Bureau of Statistics, Canberra

Backholer K et al (2012a) Projected socioeconomic disparities in the prevalence of obesity among Australian adults. Aust N Z J Public Health 36:557–563. https://doi.org/10.1111/j.1753-6405.2012.00885.x

Backholer K, Pasupathi K, Wong E, Hodge A, Stevenson C, Peeters A (2012b) The relationship between body mass index prior to old age and disability in old age. Int J Obes (Lond) 36:1180–1186. https://doi.org/10.1038/ijo.2012.99

Ball K, Crawford D (2005) Socioeconomic status and weight change in adults: a review. Soc Sci Med 60:1987–2010

Beenackers MA, Kamphuis CB, Giskes K, Brug J, Kunst AE, Burdorf A, van Lenthe FJ (2012) Socioeconomic inequalities in occupational, leisure-time, and transport related physical activity among European adults: a systematic review. Int J Behav Nutr Phys Act 9:116. https://doi.org/10.1186/1479-5868-9-116

Burke MV, Small DM (2015) Physiological mechanisms by which non-nutritive sweeteners may impact body weight and metabolism. Physiol Behav 152:381–388. https://doi.org/10.1016/j.physbeh.2015.05.036

Cancer Epidemiology Centre (2008) Melbourne collaborative cohort study databook. The Cancer Council Victoria, Melbourne

Compernolle S et al (2016) Mediating role of energy-balance related behaviors in the association of neighborhood socio-economic status and residential area density with BMI: the SPOTLIGHT study. Prev Med 86:84–91. https://doi.org/10.1016/j.ypmed.2016.01.005

Darmon N, Drewnowski A (2008) Does social class predict diet quality? Am J Clin Nutr 87:1107–1117. https://doi.org/10.1093/ajcn/87.5.1107

Ding P, Vanderweele TJ (2016) Sharp sensitivity bounds for mediation under unmeasured mediator-outcome confounding. Biometrika 103:483–490. https://doi.org/10.1093/biomet/asw012

Feng X, Wilson A (2015) Getting Bigger, Quicker? Gendered socioeconomic trajectories in Body Mass Index across the adult lifecourse: a longitudinal study of 21,403 Australians. PLoS One 10:e0141499. https://doi.org/10.1371/journal.pone.0141499

Forouzanfar MH et al (2016) Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet 388:1659–1724. https://doi.org/10.1016/S0140-6736(16)31679-8

Fowler SP (2016) Low-calorie sweetener use and energy balance: results from experimental studies in animals, and large-scale prospective studies in humans. Physiol Behav 164:517–523. https://doi.org/10.1016/j.physbeh.2016.04.047

Fowler SP, Williams K, Resendez RG, Hunt KJ, Hazuda HP, Stern MP (2008) Fueling the obesity epidemic? Artificially sweetened beverage use and long-term weight gain Obesity (Silver Spring) 16:1894–1900. https://doi.org/10.1038/oby.2008.284

Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G (2006) Indicators of socioeconomic position (part 1). J Epidemiol Community Health 60:7–12. https://doi.org/10.1136/jech.2004.023531

Gearon E, Backholer K, Hodge A, Peeters A (2013) The mediating role of dietary factors and leisure time physical activity on socioeconomic inequalities in body mass index among Australian adults. BMC Public Health 13:1214. https://doi.org/10.1186/1471-2458-13-1214

Gearon E et al (2015) Trends in body mass index according to educational attainment for urban Australian adults between 1980 and 2007. Int J Obes (Lond) 39:1019–1026. https://doi.org/10.1038/ijo.2015.27

Government Office for Science (2007) Foresight. http://www.bis.gov.uk/assets/foresight/docs/obesity/11.pdf

Hodge AM, Jenkins AJ, English DR, O'Dea K, Giles GG (2011) NMR-determined lipoprotein subclass profile is associated with dietary composition and body size. Nutr Metab Cardiovasc Dis 21:603–609. https://doi.org/10.1016/j.numecd.2009.12.003

Hu FB (2013) Resolved: there is sufficient scientific evidence that decreasing sugar-sweetened beverage consumption will reduce the prevalence of obesity and obesity-related diseases. Obes Rev 14:606–619. https://doi.org/10.1111/obr.12040

Ireland P et al (1994) Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving an ethnically diverse cohort. Asia Pac J Clin Nutr 3:19–31

Kavanagh A, Bentley RJ, Turrell G, Shaw J, Dunstan D, Subramanian SV (2010) Socioeconomic position, gender, health behaviours and biomarkers of cardiovascular disease and diabetes. Soc Sci Med 71:1150–1160. https://doi.org/10.1016/j.socscimed.2010.05.038

Lohman TG, Roche AF, Martorell R (1988) Anthropometric standardization reference manual. Human Kinetics Books, Champaign

Lowe MR (2015) Dieting: proxy or cause of future weight gain? Obes Rev 16(Suppl 1):19–24. https://doi.org/10.1111/obr.12252

MacInnis RJ, English DR, Hopper JL, Haydon AM, Gertig DM, Giles GG (2004) Body size and composition and colon cancer risk in men. Cancer Epidemiol Biomark Prev 13:553–559

MacKinnon DP (2008) Introduction to statistical mediation analysis. Multivariate applications. Psychology Press, New York

Milne RL et al (2017) Cohort profile: the Melbourne Collaborative Cohort Study (Health 2020). Int J Epidemiol https://doi.org/10.1093/ije/dyx085

Miura K, Turrell G (2014) Reported consumption of takeaway food and its contribution to socioeconomic inequalities in body mass index. Appetite 74:116–124. https://doi.org/10.1016/j.appet.2013.12.007

Molarius A (2003) The contribution of lifestyle factors to socioeconomic differences in obesity in men and women—a population-based study in Sweden. Eur J Epidemiol 18:227–234

Moore CJ, Cunningham SA (2012) Social position, psychological stress, and obesity: a systematic review. J Acad Nutr Diet 112:518–526 https://doi.org/10.1016/j.jand.2011.12.001

Mozaffarian D (2016) Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. Circulation 133:187–225. https://doi.org/10.1161/CIRCULATIONAHA.115.018585

Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB (2011) Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med 364:2392–2404. https://doi.org/10.1056/NEJMoa1014296

Murray S, Tulloch A, Criscitelli K, Avena NM (2016) Recent studies of the effects of sugars on brain systems involved in energy balance and reward: relevance to low calorie sweeteners. Physiol Behav 164:504–508. https://doi.org/10.1016/j.physbeh.2016.04.004

National Health and Medical Research Council (2009) Australian guidelines to reduce health risks from drinking alcohol. Canberra, Australia

Peeters A, Backholer K (2015) Reducing socioeconomic inequalities in obesity: the role of population prevention. Lancet Diabetes Endocrinol 3:838–840. https://doi.org/10.1016/S2213-8587(15)00373-3

Peeters A, Blake MRC (2016) Socioeconomic Inequalities in Diet Quality: from Identifying the problem to implementing solutions. Curr Nutri Rep 5:150–159. https://doi.org/10.1007/s13668-016-0167-5

Prentice AM, Jebb SA (2001) Beyond body mass index. Obes Rev 2:141–147

Proper KI, Cerin E, Brown WJ, Owen N (2007) Sitting time and socio-economic differences in overweight and obesity. Int J Obes (Lond) 31:169–176. https://doi.org/10.1038/sj.ijo.0803357

Prynne CJ, Paul AA, Mishra GD, Greenberg DC, Wadsworth ME (2005) Changes in intake of key nutrients over 17 years during adult life of a British birth cohort. Br J Nutr 94:368–376

PwC (2015) Weighing the costs of obesity: a case for action. https://www.pwc.com.au/pdf/weighing-the-cost-of-obesity-final.pdf

Rogers PJ et al (2016) Does low-energy sweetener consumption affect energy intake and body weight? A systematic review, including meta-analyses, of the evidence from human and animal studies. Int J Obes (Lond) 40:381–394. https://doi.org/10.1038/ijo.2015.177

Sanders T, Feng X, Fahey PP, Lonsdale C, Astell-Burt T (2015) Greener neighbourhoods, slimmer children? Evidence from 4423 participants aged 6 to 13 years in the Longitudinal Study of Australian children. Int J Obes (Lond) 39:1224–1229. https://doi.org/10.1038/ijo.2015.69

Shearer J, Swithers SE (2016) Artificial sweeteners and metabolic dysregulation: lessons learned from agriculture and the laboratory. Rev Endocr Metab Disord 17:179–186. https://doi.org/10.1007/s11154-016-9372-1

Smith JD, Hou T, Hu FB, Rimm EB, Spiegelman D, Willett WC, Mozaffarian D (2015) A comparison of different methods for evaluating diet, physical activity, and long-term weight gain in 3 prospective cohort studies. J Nutr 145:2527–2534. https://doi.org/10.3945/jn.115.214171

Suez J et al (2014) Artificial sweeteners induce glucose intolerance by altering the gut microbiota. Nature 514:181–186. https://doi.org/10.1038/nature13793

Textor J, Hardt J, Knuppel S (2011) DAGitty: a graphical tool for analyzing causal diagrams. Epidemiology 22:745. https://doi.org/10.1097/EDE.0b013e318225c2be

Tolppanen AM, Ngandu T, Kareholt I, Laatikainen T, Rusanen M, Soininen H, Kivipelto M (2014) Midlife and late-life body mass index and late-life dementia: results from a prospective population-based cohort. J Alzheimers Dis 38:201–209. https://doi.org/10.3233/JAD-130698

Turrell G, Bentley R, Thomas LR, Jolley D, Subramanian S, Kavanagh AM (2009) A multilevel study of area socio-economic status and food purchasing behaviour. Public Health Nutr 12:2074–2083. https://doi.org/10.1017/S1368980009004911

Turrell G, Haynes M, Wilson LA, Giles-Corti B (2013) Can the built environment reduce health inequalities? A study of neighbourhood socioeconomic disadvantage and walking for transport. Health Place 19:89–98. https://doi.org/10.1016/j.healthplace.2012.10.008

Wamala SP, Wolk A, Orth-Gomer K (1997) Determinants of obesity in relation to socioeconomic status among middle-aged Swedish women. Prev Med 26:734–744. https://doi.org/10.1006/pmed.1997.0199

Ward H, Tarasuk V, Mendelson R, McKeown-Eyssen G (2007) An exploration of socioeconomic variation in lifestyle factors and adiposity in the Ontario Food Survey through structural equation modeling. Int J Behav Nutr Phys Act 4:8. https://doi.org/10.1186/1479-5868-4-8

Winship C, Mare RD (1983) Structural equations and path analysis for discrete data. Am J Sociol 89:54–110

World Cancer Research Fund/American Institute for Cancer Research (2007) Food, nutrition, physical activity, and the prevention of cancer: a global perspective. American Institute for Cancer Research, Washington DC

World Health Organization (2003) Joint WHO/FAO expert report on diet, nutrition and the prevention of chronic disease. World Health Organization, Geneva

Xian H et al (2017) Steeper change in body mass across four decades predicts poorer cardiometabolic outcomes at midlife. Obesity (Silver Spring) 25:773–780. https://doi.org/10.1002/oby.21791

Zheng Y et al (2017) Associations of weight gain from early to middle adulthood with major health outcomes later in life. JAMA 318:255–269. https://doi.org/10.1001/jama.2017.7092

Zimberg IZ et al (2012) Short sleep duration and obesity: mechanisms and future perspectives. Cell Biochem Funct 30:524–529. https://doi.org/10.1002/cbf.2832