

DIET AND PHYSICAL ACTIVITY AND WEIGHT GAIN, OVERWEIGHT AND OBESITY IN ADULTS AND CHILDREN<sup>1</sup>: A SUMMARY MATRIX

WCRF/AICR GRADING		DECREASES RISK OF WEIGHT GAIN, OVERWEIGHT AND OBESITY	INCREASES RISK OF WEIGHT GAIN, OVERWEIGHT AND OBESITY
STRONG EVIDENCE	Convincing	Walking	Screen time (children) <sup>2</sup> Sugar sweetened drinks <sup>3</sup>
	Probable	Aerobic physical activity Foods containing dietary fibre 'Mediterranean type' dietary pattern <sup>4</sup> Having been breastfed <sup>5</sup>	Screen time (adults) <sup>2</sup> 'Fast foods' <sup>6</sup> 'Western type' diet <sup>7</sup>
LIMITED EVIDENCE	Limited – suggestive	Wholegrains <sup>8</sup> Fruit and vegetables Lactation (mother)	Sedentary behaviours <sup>9</sup> Refined grains <sup>8</sup>
	Limited – no conclusion	Vegetarian or vegan diets, adherence to dietary guidelines, dietary variety, eating breakfast, family meals, eating in the evening, eating frequency, snacking, pulses (legumes), nuts, fish, dairy, confectionery, water, artificially sweetened drinks, fruit juice, coffee and tea, alcoholic drinks, total carbohydrate, glycaemic load, total protein, caffeine, catechins, strength training, energy density, sleep	
STRONG EVIDENCE	Substantial effect on risk unlikely	None identified	

The factors identified in the matrix as increasing or decreasing risk of weight gain, overweight or obesity do so by promoting excess energy intake (positive energy balance, increased risk) relative to the level of energy expenditure (in particular physical activity), or appropriate energy balance (decreased risk), through a complex interplay of physiological, psychological and social influences.<sup>10</sup>

- The evidence for these conclusions comes mostly from studies of adults, except where specified. However, the CUP Panel judged that the conclusions for adults, unless there is evidence to the contrary, also apply to children aged 5 years and over.
- With the available evidence, the Panel could make separate conclusions for children and adults in relation to screen time. Screen time is a marker of sedentary behaviour and may also be associated with low levels of physical activity, consumption of energy-dense snacks and drinks, and exposure to marketing of such foods and drinks.
- Sugar sweetened drinks are defined here as liquids that are sweetened by adding free sugars, such as sucrose, high fructose corn syrup and sugars naturally present in honey, syrups, fruit juices and fruit juice concentrate. This includes, among others, sodas, sports drinks, energy drinks, sweetened waters, cordials, barley water, and coffee- and tea-based beverages with sugars or syrups added. This does not include versions of these drinks which are 'sugar free' or sweetened only with artificial sweeteners.
- There are recognised scores for quantifying adherence to a 'Mediterranean type' dietary pattern but it is unclear exactly what such a diet comprises. It generally describes a diet rich in fruits and vegetables, with modest amounts of meat and dairy, some fish and wine, and rich in unrefined olive oil. Traditionally it is also associated with high levels of physical activity. Currently most countries around the Mediterranean do not consume such a diet.
- The evidence relates principally to excess weight gain, overweight and obesity in childhood, but overweight and obesity in childhood tends to track into adult life.
- 'Fast foods' are readily available convenience foods that tend to be energy dense and are often consumed frequently and in large portions. Most of the evidence is from studies of foods such as burgers, fried chicken pieces, chips (French fries) and high-calorie drinks (containing sugars, such as cola, or fat, such as shakes), as typically served in international franchise outlets. Many other foods can also be prepared quickly, but the speed of preparation is not the important factor, even though it is characteristic of this group of foods.
- Such diets are characterised by high intakes of free sugars, meat and dietary fat, which are probably the factors responsible for the effects on weight. The overall conclusion includes all these factors.
- Refined grains refers to the grains themselves, or products of such grains, that have been mechanically processed to remove one or more of the bran, germ or endosperm. This is in contrast to wholegrains (or their products), which contain all three constituents.
- Sedentary behaviours comprise both high levels of physical inactivity and low levels of physical activity.
- For discussion of the integration of the exposures into clusters, please see **Section 8**.

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LIMITED EVIDENCE	Limited – suggestive	Wholegrains <sup>8</sup> Fruit and vegetables Lactation (mother)	Sedentary behaviours <sup>9</sup> Refined grains <sup>8</sup>
	Limited – no conclusion	Vegetarian or vegan diets, adherence to dietary guidelines, dietary variety, eating breakfast, family meals, eating in the evening, eating frequency, snacking, pulses (legumes), nuts, fish, dairy, confectionery, water, artificially sweetened drinks, fruit juice, coffee and tea, alcoholic drinks, total carbohydrate, glycaemic load, total protein, caffeine, catechins, strength training, energy density, sleep	
STRONG EVIDENCE	Substantial effect on risk unlikely	None identified	

The factors identified in the matrix as increasing or decreasing risk of weight gain, overweight or obesity do so by promoting excess energy intake (positive energy balance, increased risk) relative to the level of energy expenditure (in particular physical activity), or appropriate energy balance (decreased risk), through a complex interplay of physiological, psychological and social influences.<sup>10</sup>

# Summary of meta-analyses of randomised controlled trials from published reviews investigating intake of wholegrains and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. of studies	Participants
Pol et al. (2013) [105]	Weight change	Interventions to increase wholegrain intake vs control	<b>WMD</b> 0.06 (-0.09, 0.20) kg	0	26	2,060
Ye et al. (2012) [108]		Wholegrain intake, g/day	<b>Beta coefficient</b> -0.0013 (-0.011, 0.009) kg	NR	NR	NR
		Interventions to increase wholegrain intake vs control	<b>WMD</b> -0.18 (-0.54, 0.18) kg	82	9	629
Pol et al. (2013) [105]	Percentage body fat	Interventions to increase wholegrain intake vs control	<b>WMD</b> -0.48 (-0.95, -0.01) %	0	7	1,087

Abbreviations used: g = grams; kg = kilogram; WMD = weighted mean difference; NR = not reported.

## Summary of meta-analyses of randomised controlled trials from published reviews investigating intake of fruit and vegetables combined and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Kaiser et al. (2016) [124]</b>	Weight change	Increased fruit and vegetable intake (varied interventions) vs control	<b>SMD</b> 0.04 (-0.10, 0.17)	5	7	1,149
<b>Mytton et al. (2014) [125]</b>	Weight change	Increased fruit and vegetable intake (50–465 g/day; varied interventions) vs control	<b>MD</b> -0.54 (-1.05, -0.04) kg	73	7	1,026

**Abbreviations used:** g = grams; kg = kilograms; MD = mean difference; SMD = standardised mean difference.

## Summary of meta-analyses of prospective cohort studies from published reviews investigating intake of fruit and vegetables combined and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Schwingshackl et al. (2015) [126]</b>	Odds of weight gain or overweight	Highest vs lowest categories of fruit and vegetable intake	<b>OR</b> 0.91 (0.84, 0.99)	53	5	327,492

**Abbreviations used:** OR = odds ratio.

# Summary of meta-analyses of prospective cohort studies from published reviews investigating intake of fruit and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Bertoia <i>et al.</i> (2015) [122]	Weight change	Per daily serving of fruit over a 4-year period	<b>MD</b> -0.53 (-0.61, -0.44) lb	NR	3	117,918
Schwingshackl <i>et al.</i> (2015) [126]	Weight change	Per additional 100 g/day intake of fruit over 1-year period	<b>Regression coefficient</b> -13.68 (-22.97, -4.40) g	96	5	354,880
	Waist circumference	Increased fruit consumption over 1-year period	<b>Regression coefficient</b> -0.04 (-0.05, -0.02) cm	29	2	48,879
	Odds of weight gain or overweight	Highest vs lowest categories of fruit intake	<b>OR</b> 0.83 (0.71, 0.99)	28	4	93,266

**Abbreviations used:** cm = centimetres; g = grams; lb = pounds; MD = mean difference; NR = not reported; OR = odds ratio.

# Summary of meta-analyses of prospective cohort studies from published reviews investigating intake of vegetables and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Bertoia et al. (2015) [122]	Weight change	Per daily serving of vegetables over a 4-year period	<b>MD</b> -0.25 (-0.35, -0.14) lb	NR	3	117,918
Schwingshackl et al. (2015) [126]	Weight change	Per additional 100 g/day intake of vegetables over 1-year period	<b>Regression coefficient</b> 1.69 (-10.37, 13.74) g	97	4	354,632
	Odds of weight gain or overweight	Highest vs lowest categories of vegetable intake	<b>OR</b> 0.83 (0.70, 0.99)	75	5	172,502

Abbreviations used: g =grams; lb = pounds; MD = mean difference; NR = not reported; OR = odds ratio.

# Summary of meta-analyses of randomised controlled trials from published reviews investigating intake of dietary fibre and weight change in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Wanders <i>et al.</i> (2011) [149]	Weight change	Increased fibre intake (mean dose 11.1 g/day) vs no intervention	<b>WMD</b> -0.7 kg (95% CI NR)	NR	61	2,486
		Per gram increase in fibre intake per day	<b>Regression coefficient</b> -0.014% (95% CI NR) per 4 weeks	NR	61	2,486

**Abbreviations used:** CI = confidence interval; NR = not reported; WMD = weighted mean difference.



Summary of meta-analyses of randomised controlled trials from published reviews investigating consumption of a ‘Mediterranean type’ dietary pattern and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Garcia et al. (2016) [162]	Waist circumference	‘Mediterranean type’ dietary pattern intervention vs control	<b>d+</b> -0.54 (-0.77, -0.31)	96	29	4,133
Kastorini et al. (2011) [161]		‘Mediterranean type’ dietary pattern intervention vs control	<b>MD</b> -0.42 (-0.82, -0.02) cm	~0	11	1,646

Abbreviations used: cm = centimetres; d+ = overall effect size; MD = mean difference.

# Summary of prospective cohort studies from published reviews investigating consumption of refined grains and adiposity in adults

Study [publication]	Outcome	Increment/contrast	Results	No. participants Follow-up
Health Professionals' Follow-up Study (HPFS) [110]	Weight change	Servings per day of refined grain cereal	Positive association, p for trend < 0.001	M: 27,082 8 years
		Categories of refined grain intake	No association	
Nurses' Health Study (NHS) I, NHS II, HPFS (pooled) [177]	Weight change	Increased servings per day of refined grains over a 4-year period	MD 0.39 (0.21, 0.58) lb p < 0.001	M&W: 120,887 20 years
Baltimore Longitudinal Study of Aging [178]	BMI change	'White bread'-defined dietary pattern vs 'healthy' dietary pattern at baseline	Beta coefficient 0.05 (-0.10, 0.23) kg/m <sup>2</sup>	M&W: 459 1 year
	Waist circumference		Beta coefficient 0.90 (0.12, 1.68) cm	
Monitoring of Trends and Determinants in Cardiovascular Disease (MONICA1) [112]	Waist circumference	Per quintile intake of refined bread	<b>M:</b> beta coefficient -0.06 (-0.22, 0.09) cm <b>W:</b> beta coefficient 0.29 (0.07, 0.51) cm	M: 1,127 W: 1,073 6 years
Danish Diet, Cancer and Health study [111]	Waist circumference	Per MJ per day of refined grain products and potatoes	<b>M:</b> beta coefficient 0.06 (-0.12, 0.25) cm <b>W:</b> beta coefficient 0.48 (0.18, 0.78) cm	M: 20,126 W: 22,570 5.3 years
European Prospective Investigation into Cancer (EPIC) (5 centres) [179]	ΔWC <sub>BMI</sub>	100 kcal increments of white bread consumption over 1 year	Beta coefficient 0.01 (0.01, 0.02) cm	M&W: 48,361 5.5 years
NHS I [113]	Odds of weight gain	Highest vs lowest quintile intake of refined grains	OR 1.26 (0.97, 1.64), p for trend = 0.04	W: 74,091 12 years
	Odds of obesity		OR 1.18 (1.08, 1.28), p for trend < 0.0001	
HPFS [114]	Risk of overweight	Intake of > 1 serving of refined grain breakfast cereal per day vs rarely/never eat	RR 0.81 (0.65, 1.01), p for trend = 0.08	M: 17,881 13 years

Abbreviations used: ΔWC<sub>BMI</sub> = waist circumference for a given BMI; cm = centimetre; kcal = kilocalories; lb = pounds; MD = mean difference; MJ = mega joules; OR = odds ratio; RR = relative risk.

## Summary of meta-analyses of randomised controlled trials from published reviews investigating consumption of sugar sweetened drinks and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Malik et al. (2013) [182]</b>	Weight change	Increased SSB intake vs control	<b>WMD</b> 0.85 (0.50, 1.20) kg	0	5	292
<b>Kaiser et al. (2013) [183]</b>	Weight change	Increased SSB intake vs control	<b>SMD</b> 0.28 (0.12, 0.44)	48	7	665

**Abbreviations used:** kg = kilograms; SSB = sugar sweetened beverage; SMD = standardised mean difference; WMD = weighted mean difference.

## Summary of meta-analyses of prospective cohort studies from published reviews investigating consumption of sugar sweetened drinks and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Malik et al. (2013) [182]</b>	Annual weight change	Per 12 oz serving of SSB per day	<b>WMD</b> 0.22 (0.09, 0.34) kg	70	7	170,141
<b>Pan et al. (2013) [185]</b>	Weight change	Per standard serving of SSB per day over 4-year period	<b>MD</b> 0.36 (0.24, 0.48) kg	NR	3	124,988

**Abbreviations used:** kg = kilograms; MD = mean difference; NR = not reported; oz = ounce; SSB = sugar sweetened beverage; WMD = weighted mean difference.

Summary of meta-analyses of randomised controlled trials from published reviews investigating consumption of sugar sweetened drinks and adiposity in children

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Malik et al. (2013) [182]	BMI change	Interventions to reduce SSB intake vs control	WMD -0.17 (-0.39, 0.05) kg/m <sup>2</sup>	75	5	2,772
Kaiser et al. (2013) [183]	'Adiposity' change	Interventions to reduce SSB intake vs control	SMD -0.06 (-0.13, 0.01)	59	8	3,205

Abbreviations used: SSB = sugar sweetened beverage; SMD = standardised mean difference; WMD = weighted mean difference.

## Summary of meta-analyses of prospective cohort studies from published reviews investigating consumption of sugar sweetened drinks and adiposity in children

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Malik et al. (2013) [182]</b>	Annual BMI change	Per 12 oz serving of SSB per day	<b>WMD</b> 0.07 (0.01, 0.12) kg/m <sup>2</sup>	92	15	25,745
<b>Te Morenga et al. (2013) [184]</b>	Odds of overweight or obesity	More than one serving of SSB per day vs little/no intake	<b>OR</b> 1.55 (1.32, 1.82)	0	5	12,317

**Abbreviations used:** OR = odds ratio; oz = ounce; SSB = sugar sweetened beverage; WMD = weighted mean difference.

Summary of prospective cohort studies from published reviews investigating consumption of ‘fast food’ and adiposity in adults

Study [publication]	Outcome	Increment/contrast	Results	No. participants Follow-up
Pound of Prevention Study [212]	Weight change	Per increase of one ‘fast foods’ meal per week	Beta coefficient 0.72 SE ±0.20 kg p = 0.01	W: 891 3 years
Coronary Artery Risk Development in Young Adults (CARDIA) Study [213–215]	Weight change	Frequency of ‘fast foods’ consumption at baseline	<b>Black participants:</b> Beta coefficient 2.22 SE ±0.72 kg, p = 0.0014 <b>White participants:</b> Beta coefficient 1.56 SE ±0.55 kg, p = 0.0064	Black participants: 1,444 White participants: 1,587 15 years [215]
		Change in frequency of ‘fast foods’ consumption over study duration	<b>Black participants:</b> Beta coefficient 0.74 SE ±0.45 kg, p = 0.1053 <b>White participants:</b> Beta coefficient 1.84 SE ±0.44 kg, p < 0.0001	
	Weight change	Frequency of meals at ‘fast foods’ restaurants per week at baseline	Beta coefficient 0.15 SE ±0.05 kg, p < 0.001	M&W: 3,643 13 years [214]
	Waist circumference		Beta coefficient 0.12 SE ±0.04 cm, p > 0.05	
	BMI change	Increase in frequency of ‘fast foods’ consumption across study period	Beta coefficient 0.20 (0.005, 0.393) kg/m², p = 0.044	M&W: 3,394 3 years [213]
		Increase in frequency of ‘fast foods’ and restaurant food consumption across study period	Beta coefficient 0.29 (0.060, 0.509) kg/m², p = 0.013	
Portland Neighborhood Environment and Health Study [216]	Weight change	More than 1–2 meals at ‘fast foods’ restaurants per week vs no consumption	Beta coefficient 0.65 SE ±0.32 kg, p < 0.05	M&W: 1,145 1 year
	Waist circumference		Beta coefficient 1.06 SE ±0.41 cm, p < 0.05	
Supplemental Nutrition Program for Women, Infants and Children 1998 [217]	BMI change	Frequency per week of eating at ‘fast foods’ restaurants	<b>M:</b> Beta coefficient -0.23 (-0.56, 0.11) kg/m² <b>W (high income):</b> Beta coefficient 0.02 (-0.05, 0.09) kg/m² <b>W (low income):</b> Beta coefficient -0.06 (-0.20, 0.08) kg/m²	M: 198 W (high income): 529 W (low income): 332 1 year
Australian Longitudinal Study on Women’s Health [218]	Odds of weight maintenance	Occasional consumption of ‘fast foods’ relative to never/rarely	OR 0.85 (0.75, 0.96)	W: 8,726 4 years
		Frequent consumption of ‘fast foods’ relative to never/rarely	OR 0.88 (0.76, 1.02)	
The Seguimiento University of Navarra (SUN) Cohort [194]	Odds of weight gain	Highest vs lowest quintile of ‘fast foods’ consumption	OR 1.2 (1.02, 1.41)	M&W: 7,194 28.5 months
Black Women’s Health Study [219]	Risk of obesity	Consumption of specific type of ‘fast foods’ more than once per week vs fewer than five times per year	Hamburgers: HR 1.27 (1.14, 1.41) p for trend < 0.001	W: 19,479 14 years
			Fried chicken: HR 1.08 (0.96, 1.21) p for trend = 0.02	
			Pizza: HR 1.08 (0.92, 1.27), p for trend = 0.04	
			Chinese food: HR 1.20 (1.05, 1.37) p for trend = 0.05	
			Mexican food: HR 0.92 (0.74, 1.14) p for trend = 0.78	
			Fried fish: HR 0.92 (0.75, 1.12), p for trend = 0.78	

cm = centimetres; HR = hazard ratio; kg = kilograms; M = men; OR = odds ratio; SE = standard error; W = women.

# Summary of prospective cohort studies from published reviews investigating consumption of food from restaurants and cafeterias and adiposity in adults

Study [publication]	Outcome	Increment/ contrast	Results	No. participants Follow-up
The SUN Cohort [220]	Weight change	≥ 2 times per week eating out relative to never/ rarely	Beta coefficient 129 (62, 197) g per year, p < 0.001	M&W: 9,182 4.4 years
	BMI change		Beta coefficient 0.07 (0.04, 0.10) kg/m2, p < 0.001	
	Odds of weight gain		OR 1.36 (1.13, 1.63)	
	Risk of overweight or obesity		HR 1.33 (1.13, 1.57)	
The CARDIA Study [213, 214]	Weight change	Increase of one meal at a restaurant per week at baseline	Beta coefficient 0.09 SE ±0.04 kg, p > 0.05	M&W: 3,643 13 years [214]
	Waist circumference		Beta coefficient 0.08 SE ±0.03 cm, p > 0.05	
	BMI change	Increase in frequency of restaurant food consumption across study period	Beta coefficient -0.01 (-0.212, 0.187) kg/m², p = 0.903	M&W: 3,394 3 years [213]
Health and Retirement Study [221]	BMI change	Per \$1 decreased individual spending on eating out	Beta coefficient -0.0003 kg/m², p < 0.05	M&W: 6,012 10 years



## Summary of prospective cohort studies from published reviews investigating consumption of ‘fast food’ and adiposity in children

Study [publication]	Outcome	Increment/contrast	Results	No. participants Follow-up
<b>Growing Up Today Study [222]</b>	BMI change	Increased consumption of fried food away from home from baseline to follow-up	Beta coefficient 0.21 (0.03, 0.39) kg/m <sup>2</sup>	14,355 3 years
		Decreased consumption of fried food away from home from baseline to follow-up	Beta coefficient -0.03 (-0.25, 0.19) kg/m <sup>2</sup>	
<b>Identifying Determinants of Eating and Activity (IDEA) and Etiology of Childhood Obesity (ECHO) cohorts [223]</b>	BMI change	Frequency of ‘fast foods’ purchases over one month	<b>B:</b> No significant association <b>G:</b> No significant association	B: 340 G: 353 2 years
	Percentage body fat		<b>B:</b> No significant association <b>G:</b> No significant association	
<b>National Longitudinal Study of Adolescent Health (NLSAH) cohort [224]</b>	BMI z-score change	Frequency of ‘fast foods’ consumption at baseline	Beta coefficient 0.02 SE ±0.01, p < 0.05	9,919 5 years
<b>Avon Longitudinal Study of Parents and Children (ALSPAC) [225]</b>	BMI z-score change	Frequency of ‘fast foods’ consumption at baseline	Beta coefficient 0.0822 SE ±0.028, p < 0.05	4,022 2 years
	Percentage body fat		Beta coefficient 2.063 SE ±0.3713%, p < 0.05	
	Risk of obesity		OR 1.23 (1.02, 1.49)	
<b>Massachusetts Institute of Technology cohort 1990 [226]</b>	BMI z-score change	Frequency of ‘quick service’ foods at baseline	<b>Never:</b> 0.28 SE ±0.07 <b>Once per week:</b> 0.20 SE ±0.10 <b>≥2 times per week:</b> 0.82 SE ±0.15 F = 6.49, p = 0.0023	101 4–7 years
<b>Health, Eating and Play Study (HEAPS) [227]</b>	BMI z-score change	Frequency of ‘fast foods’ consumption	No significant association	293 3 years
	Percentage body fat		No significant association	
<b>Project Eating Among Teens (EAT) Study [228]</b>	Risk of overweight	Fast food consumption in days per week at baseline	<b>B:</b> OR 1.03 (0.90, 1.17) <b>G:</b> OR 0.88 (0.79, 0.98)	B: 1,119 G: 1,380 5 years

Abbreviations used: B = boys; G = girls; OR = odds ratio; SE = standard error.

# Summary of meta-analyses of randomised controlled trials from published reviews investigating components of the ‘Western type’ diet and weight change in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Sugars						
<i>Interventions to reduce sugars intake</i>						
<b>Te Morenga et al. (2013) [184]</b>	Weight change	Ad libitum diet with reduced free sugars intake vs habitual diet	<b>WMD</b> -0.80 (-1.21, -0.39) kg	17	5	1,286
<i>Interventions to exchange sugars with other macronutrients</i>						
<b>Te Morenga et al. (2013) [184]</b>	Weight change	Isoenergetic exchange of free sugars vs complex CHO	<b>WMD</b> 0.04 (-0.04, 0.13) kg	32	11	144
<b>Sievenpiper et al. (2012) [236]</b>	Weight change	Isoenergetic exchange of fructose vs other dietary CHO	<b>MD</b> -0.13 (-0.37, 0.10) kg	8	13	417
<i>Interventions to increase sugars intake</i>						
<b>Te Morenga et al. (2013) [184]</b>	Weight change	Hyperenergetic addition of free sugars vs habitual diet	<b>WMD</b> 0.75 (0.30, 1.19) kg	82	10	382
<b>Sievenpiper et al. (2012) [236]</b>	Weight change	Hyperenergetic addition of fructose vs habitual diet	<b>MD</b> 0.37 (0.15, 0.58) kg	0	8	176
Dietary fat						
<b>Hooper et al. (2015) [240]</b>	Weight change	Reduced proportion of energy as dietary fat vs habitual diet	<b>MD</b> -1.54 (-1.97, -1.12) kg	77	24	53,647

Abbreviations used: CHO = carbohydrates; kg = kilograms; MD = mean difference; WMD = weighted mean difference.

## Summary of meta-analyses of prospective cohort studies from published reviews investigating components of the 'Western type' diet and weight change in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Sugars						
<b>Te Morenga et al. (2013)</b> <b>[184]</b>	Weight change	Additional daily serving of sweets (candy) increase from baseline	<b>Regression coefficient</b> 0.02 (-0.02, 0.07) <sub>units NR</sub>	91	2	50,670
Dietary fat						
<b>Summerbell et al. (2009)</b> <b>[106]</b>	Weight change	Dietary fat as percentage of total energy intake	<b>Regression coefficient</b> 0.07 (-0.03, 0.16) <sub>units NR</sub>	NR	4	9,753

**Abbreviations used:** NR = not reported.

# Overview of prospective cohort studies (not included in meta-analyses) from published reviews investigating components of the ‘Western type’ diet and adiposity in adults

Exposure (increased intake)		Publications	Association with adiposity
‘Western type’ diet	Free sugars	[111, 112, 138, 241]	<b>14 results from 4 publications (3 cohorts):</b> 10 results reported positive (adverse) associations, of which 3 were significant 4 results reported inverse associations, of which 1 was significant
	Dietary fat	[111, 138, 150, 151, 242–244]	<b>23 results from 7 publications:</b> 17 results reported positive (adverse) associations, of which 7 were significant 6 results reported inverse associations, of which 3 were significant
	Meat	Total meat (Also see <b>Table 19</b> )	<b>27 results from 8 publications:</b> 25 results reported positive (adverse) associations, of which 17 were significant 1 result reported an inverse association (not significant) 1 result reported no association
		Red meat	<b>11 results from 7 publications:</b> 9 results reported positive (adverse) associations, of which 4 were significant 2 results reported inverse associations, of which 2 were significant
		Processed meat	<b>17 results from 6 publications:</b> 15 results reported positive (adverse) associations, of which 9 were significant 2 results reported inverse associations, of which 1 was significant
		Poultry	<b>4 results from 3 publications:</b> 3 results reported positive (adverse) associations, of which 2 were significant 1 result reported an inverse association (not significant)

For dietary fat, only studies with more than 1,000 participants are reported; please see the [Energy balance and body fatness literature review 2017](#).

Study [publication]	Outcome	Increment/contrast	Results	No. participants Follow-up
<b>EPIC-PANACEA [245]</b>	Weight change	Per 100 kcal increase in total meat intake	Beta coefficient 65 (39, 90) g/year, p < 0.00001	M&W: 373,803 5 years
<b>The SUN Cohort [136]</b>	Weight change	Tertiles of meat intake	<b>Low:</b> 0.41 (0.26, 0.56) kg <b>Mid:</b> 0.62 (0.40, 0.84) kg <b>High:</b> 0.79 (0.56, 1.02) kg p for trend = 0.001	M&W: 6,319 28 months
<b>EPIC-Oxford [246]</b>	Weight change	‘Meat eater’ dietary pattern vs ‘fish eater’ dietary pattern over one year	<b>M:</b> No significant difference <b>W:</b> Significantly greater weight gain in ‘meat eater’ dietary pattern, p < 0.05	M: 5,373 W: 16,593 5.3 years
		‘Meat eater’ dietary pattern vs ‘vegetarian’ dietary pattern over one year	<b>M:</b> No significant difference <b>W:</b> No significant difference	
		‘Meat eater’ dietary pattern vs ‘vegan’ dietary pattern over one year	<b>M:</b> Significantly greater weight gain in ‘meat eater’ dietary pattern, p < 0.05 <b>W:</b> Significantly greater weight gain in ‘meat eater’ dietary pattern, p < 0.05	
<b>Cancer Prevention Study II [139]</b>	BMI change	Highest vs lowest quintile of meat intake	<b>M:</b> MD 0.34 kg/m <sup>2</sup> SE ±0.05, p < 0.001 <b>W:</b> MD 0.19 kg/m <sup>2</sup> SE ±0.05, p < 0.001	M: 35,156 W: 44,080 10 years
	Odds of ‘gaining weight at the waist’		<b>M:</b> OR 1.46 (1.25, 1.71) <b>W:</b> OR 1.50 (1.20, 1.87)	
<b>Medical Research Council National Survey of Health and Development (MRC NSHD) 1964 birth cohort [247]</b>	BMI change	Per 10 g increase in total meat intake at baseline	<b>M:</b> Beta coefficient 0.013 SE±0.003 kg/m <sup>2</sup> , p < 0.001 <b>W:</b> Beta coefficient 0.013 SE±0.005 kg/m <sup>2</sup> , p = 0.008	M: 517 W: 635 10 years
	Waist circumference		<b>M:</b> Beta coefficient 0.034 SE±0.009 cm, p < 0.001 <b>W:</b> Beta coefficient 0.035 SE±0.012 cm, p = 0.003	
<b>MONICA1 [112]</b>	Waist circumference	Per quintile increase of meat product intake	<b>M:</b> Beta coefficient 0.11 (-0.06, 0.28) cm <b>W:</b> Beta coefficient 0.20 (-0.05, 0.44) cm	M: 1,166 W: 1,120 6 years
<b>EPIC-Diet, Obesity and Genes (DiOGenes) [179]</b>	ΔWC <sub>BMI</sub>	100 kcal increments of meat product intake over one year	Beta coefficient 0.02 (0.00, 0.03) cm, p = 0.036	M&W: 48,631 5.5 years
<b>EPIC-Potsdam [138]</b>	Odds of weight gain (>2kg)		<b>M:</b> OR 1.06 (0.85, 1.32) <b>W:</b> OR 1.36 (1.04, 1.79)	M: 6,364 W: 11,005 2.2 years
	Odds of weight gain (<2kg)		<b>M:</b> OR 1.00 (0.83, 1.20) <b>W:</b> OR 1.21 (0.98, 1.50)	
	Odds of weight loss (<2kg)	Per 100 g of meat intake	<b>M:</b> OR 1.01 (0.85, 1.21) <b>W:</b> OR 0.79 (0.64, 0.97)	
	Odds of weight loss (>2kg)		<b>M:</b> OR 0.79 (0.63, 1.00), p <0.05 <b>W:</b> OR 0.81 (0.64, 1.03)	

**Abbreviations used:** ΔWC<sub>BMI</sub> = waist circumference for a given BMI; cm = centimetres; g = grams; kcal = kilocalories; M = men; MD = mean difference; OR = odds ratio; PANACEA = Physical Activity, Nutrition, Alcohol, Cessation of Smoking, and Eating out of Home in Relation to Anthropometry; SE = standard error; W = women.

# Summary of meta-analyses of randomised controlled trials from published reviews investigating aerobic physical activity and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Hespanhol et al. (2016) [266]</b>	Weight change	Running programme vs no intervention	<b>WMD</b> -2.74 (-3.43, -2.06) kg	0	21	979
<b>Kelley and Kelley (2006) [267]</b>		Varied aerobic exercise vs control	<b>MD</b> -3.4 (-5.3, -1.5) kg	NR*	3	NR
<b>van 't Riet et al. (2014) [268]</b>	BMI change	Active video gaming vs no intervention	<b>SMD</b> 0.68 (0.13, 1.24)	68	6	142
<b>Hespanhol et al. (2016) [266]</b>		Running programme vs no intervention	<b>WMD</b> -0.23 (-0.61, 0.15) kg/m <sup>2</sup>	0	10	256
<b>Hespanhol et al. (2016) [266]</b>	Percentage body fat change	Running programme vs no intervention	<b>WMD</b> -1.63 (-2.15, -1.12) %	0	11	657
<b>Kelley and Kelley (2006) [267]</b>		Varied aerobic exercise vs control	<b>MD</b> -1.4 (-2.3, -0.6) %	NR*	3	NR
<b>Oja et al. (2015) [269]</b>	Fat mass change	Interventions to participate in football (soccer) vs no intervention	<b>MD</b> -2.64 (-6.06, 0.78) kg	16	5	NR
<b>Ismail et al. (2012) [270]</b>	VAT change	Varied aerobic exercise interventions vs control	<b>SMD</b> -0.23 (-0.35, -0.12)	71	27	1,409

\*I<sup>2</sup> statistic not reported; Q statistic for weight change meta-analysis, Q = 2.8, p = 0.25; Q statistic for percentage body fat change meta-analysis, Q = 1.7, p = 0.43.

**Abbreviations used:** kg = kilograms; MD = mean difference; NR = not reported; SMD = standardised mean difference; VAT = visceral adipose tissue; WMD = weighted mean difference.

## Summary of meta-analyses of randomised controlled trials from published reviews investigating aerobic physical activity and adiposity in children

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Bochner et al. (2015) [272]</b>	Weight change	Active video gaming vs no intervention	<b>SMD</b> -0.08 (-0.25, 0.08) kg	NR*	7	588
<b>van 't Riet et al. (2014) [268]</b>	BMI change	Active video gaming vs no intervention	<b>SMD</b> 0.20 (-0.08, 0.48)	46	5	561
<b>Costigan et al. (2015) [273]</b>	BMI change	High-intensity interval training programme vs control	<b>MD</b> -0.6 (-0.9, -0.4) kg/m <sup>2</sup>	0	8	870
	Percentage body fat change	High-intensity interval training programme vs control	<b>MD</b> -1.6 (-2.9, -0.5) %	63	7	786
	Waist circumference	High-intensity interval training programme vs control	<b>MD</b> -1.5 (-4.1, -1.1) cm	68	6	NR

\*I<sup>2</sup> value not reported; test for heterogeneity  $\chi^2 = 0.69$ , degrees of freedom = 6, P = 1.0.

**Abbreviations used:** cm = centimetres; kg = kilograms; MD = mean difference; NR = not reported; SMD = standardised mean difference.

# Summary of meta-analyses of randomised controlled trials from published reviews investigating walking and adiposity in adults

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Murphy et al. (2007) [275]</b>	Weight*	Walking intervention vs habitual lifestyle	<b>WMD</b> -0.95 SD $\pm$ 0.61 kg, p < 0.001	NR	18	738
<b>Gao et al. (2016) [276]</b>	Weight change	Walking intervention vs habitual lifestyle	<b>WMD</b> -1.14 (-1.86, -0.42) kg	20	8	853 women only
<b>Murtagh et al. (2015) [277]</b>		Walking intervention vs habitual lifestyle	<b>WMD</b> -1.37 (-1.75, -1.00) kg	66	25	1,275
<b>Murphy et al. (2007) [275]</b>	BMI*	Walking intervention vs habitual lifestyle	<b>WMD</b> -0.28 SD $\pm$ 0.20 kg/m <sup>2</sup> p = 0.015	NR	16	836
<b>Gao et al. (2016) [276]</b>	BMI change	Walking intervention vs habitual lifestyle	<b>WMD</b> -0.33 (-0.62, -0.04) kg/m <sup>2</sup>	11	6	701 women only
<b>Hanson and Jones (2015) [278]</b>		Walking intervention vs habitual lifestyle	<b>MD</b> -0.71 (-1.19, -0.23) kg/m <sup>2</sup>	0	12	451
<b>Murtagh et al. (2015) [277]</b>		Walking intervention vs habitual lifestyle	<b>MD</b> -0.53 (-0.72, -0.35) kg/m <sup>2</sup>	70	23	1,201
<b>Murphy et al. (2007) [275]</b>	Percentage body fat*	Walking intervention vs habitual lifestyle	<b>WMD</b> -0.63 SD $\pm$ 0.66%, p = 0.035	NR	12	604
<b>Gao et al. (2016) [276]</b>	Percentage body fat change	Walking intervention vs habitual lifestyle	<b>WMD</b> -2.36 (-3.21, -1.52) %	0	3	444 women only
<b>Hanson and Jones (2015) [278]</b>		Walking intervention vs habitual lifestyle	<b>MD</b> -1.31 (-2.10, -0.52) %	0	7	328
<b>Murtagh et al. (2015) [277]</b>		Walking intervention vs habitual lifestyle	<b>WMD</b> -1.22 (-1.70, -0.73) %	68	14	719
<b>Hanson and Jones (2015) [278]</b>	Waist circumference	Walking intervention vs habitual lifestyle	<b>MD</b> -3.55 (-8.08, 0.98) cm	0	2	35
		Walking intervention vs habitual lifestyle	<b>WMD</b> -1.51 (-2.34, -0.68) cm	38	11	574
<b>Murtagh et al. (2015) [277]</b>	Waist-hip ratio	Walking intervention vs habitual lifestyle	<b>WMD</b> -0.01 (-0.02, 0.00)	60	14	706

\*Unclear if result is difference in change between groups or difference in attained measure between groups.

**Abbreviations used:** cm = centimetres; kg = kilogram; MD = mean difference; NR = not reported; WMD = weighted mean difference.



Summary of prospective cohort studies with more than 1,000 participants from published reviews investigating sedentary behaviours and adiposity in adults

Study [publication]	Outcome	Increment/contrast	Results	No. participants Follow-up
Australian Longitudinal Study on Women's Health [218, 303, 304]	Weight change	Hours per weekday spent sitting down at baseline	Beta coefficient 0.030 (-0.051, 0.112) Units of weight unclear	W: 5,562 6 years [303]
	Percentage weight change	Hours per day spent sitting down over 3 years (2001–2004)	Beta coefficient 0.64 (-0.20, 1.48) %	W: 8,233 6 years [304]
		Hours per day spent sitting down over 3 years (2004–2007)	Beta coefficient -0.51 (-1.35, 0.33) %	
	Odds of weight gain	> 52 hours per week sitting time vs < 33 hours	OR 0.80 (0.70, 0.91)	W: 8,726 4 years [218]
Copenhagen City Heart Study [305]	BMI (attained)	Quartiles of leisure time physical activity at baseline relative to Q1 ('sedentary')	<b>Q2</b> 25.9 SD ±3.8, p > 0.05 <b>Q3</b> 26.0 SD ±3.9, p > 0.05 <b>Q4</b> 25.8 SD ±3.6, p > 0.05	M: 6,506 15 years
			<b>Q2</b> 24.9 SD ±4.6, p > 0.05 <b>Q3</b> 24.9 SD ±4.5, p > 0.05 <b>Q4</b> 24.6 SD ±4.1, p > 0.05	W: 7,708 15 years
		Transition between quartiles of leisure time physical activity (Q1 = 'sedentary') across study period relative to no change	<b>Becoming more sedentary:</b> 27.0 kg/m² SD ±4.4, p > 0.05 <b>Becoming less sedentary:</b> 26.5 kg/m² SD ±3.7, p > 0.05	M: 2,946 15 years
			<b>Becoming more sedentary:</b> 26.0 SD ±5.0, p > 0.05 <b>Becoming less sedentary:</b> 25.5 SD ±4.4, p > 0.05	W: 4,124 15 years
University of North Carolina Alumni Heart Study [306]	BMI change	Categorised as sedentary at baseline and follow-up vs non-sedentary at baseline and follow-up	Beta coefficient 0.09 (0.05, 0.13) kg/m²	M&W: 2,070 8 years
		Categorised as becoming non-sedentary across study period vs non-sedentary at baseline and follow-up	Beta coefficient -0.04 (-0.08, 0.00) kg/m² per year	
		Categorised as becoming sedentary across study period vs non-sedentary at baseline and follow-up	Beta coefficient 0.06 (0.03, 0.09) kg/m² per year	
1958 British Birth Cohort [307]	BMI change	Per hour per day increase in sitting at work	MD -0.01 (-0.04, 0.02) kg/m²	M&W: 6,562 5 years
Cancer Prevention Study II [289]	Odds of weight gain	> 6 hours per day of non-occupational sedentary behaviour vs < 3 hours	OR 1.06 (0.87, 1.30)	W: 18,583 7 years
NHS [308]	Risk of obesity	Number of hours per week sitting at work or away from home vs 0–1 hours	<b>2–5 hours:</b> RR 1.02 (0.89, 1.18) <b>&gt;40 hours:</b> RR 1.25 (1.02, 1.54)	W: 50,277 6 years
		Number of hours per week sitting at home vs 0–1 hours	<b>2–5 hours:</b> RR 0.99 (0.83, 1.18) <b>&gt;40 hours:</b> RR 1.11 (0.85, 1.45)	
The SUN Cohort [309]	Risk of obesity	Annual distance travelled in motor vehicles > 20,000 km vs < 10,000 km	HR 1.00 (0.85, 1.17)	M&W: 6,808 6.4 years
Whitehall II Cohort [310]	Risk of obesity	> 40 hours sedentary time at work per week vs 0–6 hours	OR 1.10 (0.59, 1.96)	M&W: 10,308 6 years
		> 17 hours non-TV leisure time per week vs 0–6 hours	OR 0.88 (0.40, 1.95)	

HR = hazard ratio; M = men; OR = odds ratio; Q = quartile; RR = relative risk; SD = standard deviation; W = women.

## Summary of meta-analyses of randomised controlled trials from published reviews investigating sedentary behaviours and adiposity in children

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Azevedo et al. (2016) [300]</b>	BMI or BMI z-score change	Interventions to reduce sedentary behaviours vs no intervention	SMD -0.060 (-0.098, -0.022)	50	71	29,650
	BMI change		MD -0.158 (-0.238, -0.077) kg/m <sup>2</sup>	88	51	18,012

**Abbreviations used:** MD = mean difference; SMD = standardised mean difference.

# Summary of prospective cohort studies with more than 1,000 participants from published reviews investigating screen time and adiposity in adults

Study [publication]	Outcome	Increment/contrast	Results	No. participants Follow-up
NHS I, NHS II, HPFS (pooled) [177]	Weight change	Per hour per day increase in TV viewing	Beta coefficient 0.31 (0.20, 0.42) lb	M&W: 120,877 20 years
NHS [308]	Risk of obesity	Number of hours per week watching TV vs 0–1 hours	<b>2–5 hours per week:</b> RR 1.22 (1.06, 1.42) <b>&gt;40 hours per week:</b> RR 1.94 (1.51, 2.49)	W: 50,277 6 years
National Weight Control Register [323]	Weight change	Frequency of TV viewing at baseline	Beta coefficient 0.081 kg t = 2.532, p = 0.011	M&W: 1,422 1 year
		Increase in frequency of TV viewing from baseline	Beta coefficient 0.123 kg t = 3.885, p = 0.000	
1958 British Birth Cohort [307, 324]	BMI change	Per hour per day increase in TV viewing	MD 0.06 (0.01, 0.12) kg/ m²	M&W: 6,562 5 years [307]
	Waist circumference	Watching TV 3–4 times per week vs < 2 times at baseline	Beta coefficient 0.351 (-0.659, 1.361) cm	M&W: 5,972 21 years [324]
		Watching TV > 5 times per week vs < 2 times at baseline	Beta coefficient 1.166 (0.325, 2.008) cm	
AusDiab [325]	Waist circumference	Per 10 hours per week of TV viewing at baseline	<b>M:</b> Beta coefficient -0.25 (-0.56, 0.06) cm <b>W:</b> Beta coefficient 0.04 (-0.31, 0.39) cm	M: 1,703 W: 2,143 5 years
		Increase in TV viewing (hours per week)	<b>M:</b> Beta coefficient 0.43 (0.08, 0.78) cm <b>W:</b> Beta coefficient 0.68 (0.30, 1.05) cm	
Atherosclerosis Risk in Communities (ARIC) [326]	Odds of overweight or obesity	Level of TV exposure at baseline	<b>High:</b> OR 0.93 (0.83, 1.04) <b>Medium:</b> OR 1.03 (0.92, 1.15)	M&W: 12,678 6 years
Whitehall II Cohort [310]	Risk of obesity	> 19 hours TV viewing per week vs 0–6 hours	OR 0.97 (0.41, 2.29)	M&W: 1,071 6 years

Abbreviations used: cm = centimetres; kg = kilograms; lb = pounds; M = men; MD = mean difference; OR = odds ratio; RR = relative risk; TV = television; W = women.

## Summary of meta-analyses of randomised controlled trials from published reviews investigating screen time and BMI in children

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Tremblay et al. (2011) [311]</b>	BMI change	Intervention to decrease screen time vs no intervention	<b>MD</b> -0.89 (-1.67, -0.11) kg/m <sup>2</sup>	46	4	326
<b>Wahi et al. (2011) [312]</b>	BMI change	Intervention to decrease screen time vs no intervention	<b>MD</b> -0.10 (-0.28, 0.09) kg/m <sup>2</sup>	38	6	708

**Abbreviations used:** MD = mean difference.

## Summary of meta-analyses of prospective cohort studies from published reviews investigating screen time and combined measures of adiposity in children

Published review	Outcome	Increment/ contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Marshall <i>et al.</i> (2004) [313]</b>	Combined measures of body fatness	Increased time spent watching TV	r <sub>c</sub> 0.053 (0.030, 0.052) Units NR	NR	6	15,797

**Abbreviations used:** NR = not reported; r<sub>c</sub> = fully corrected sample-weighted mean effect size.

## Summary of meta-analyses of randomised controlled trials from published reviews investigating having been breastfed and adiposity

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
<b>Giugliani et al. (2015) [347]</b>	Weight z-score	Increased BF duration (varied interventions) vs usual care/no intervention	<b>SMD</b> 0.03 (-0.06, 0.12)	78	16	14,736
	BMI or weight-for-height z-score	Increased BF duration (varied interventions) vs usual care/no intervention	<b>SMD</b> -0.06 (-0.12, 0.00)	61	12	29,063

**Abbreviations used:** BF = breastfeeding; SMD = standardised mean difference.

## Summary of meta-analyses of prospective cohort studies from published reviews investigating having been breastfed and adiposity

Published review	Outcome	Increment/ contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Owen et al. (2005a) [348]	BMI	BF vs formula fed (varied definitions)	MD -0.04 (-0.05, -0.02) kg/m <sup>2</sup>	NR	36	355,301
Horta et al. (2015) [349]	Odds of overweight or obesity	BF vs not-BF (varied definitions)	OR 0.79 (0.73, 0.85)	12	54	NR
Yan et al. (2014) [350]		BF vs not-BF (varied definitions)	OR 0.78 (0.73, 0.82)	NR	15	141,247
Weng et al. (2012) [351]		Ever BF vs never BF (varied definitions)	OR 0.85 (0.74, 0.99)	73	10	NR
Arenz et al. (2004) [352]		BF vs not-BF (varied definitions)	OR 0.73 (0.64, 0.85)	NR	2	4,389
Owen et al. (2005b) [353]		BF vs formula fed	OR 0.87 (0.85, 0.89)	NR*	29	298,900
Harder et al. (2005) [354]		Total duration of BF (up to 12 months)	Regression coefficient 0.94 (0.89, 0.98)	NR	17	121,072
	Per month of BF	OR 0.96 (0.94, 0.98)	NR	11	74,102	

\*I<sup>2</sup> value not reported; test for heterogeneity  $\chi^2_{28} = 111$ ,  $p < 0.001$ .

**Abbreviations used:** BF = breastfed or breastfeeding; MD = mean difference; NR = not reported; OR = odds ratio.

# Summary of meta-analyses from published reviews investigating lactation and adiposity in the mother

Published review	Outcome	Increment/contrast	Result (95% CI)	I <sup>2</sup> (%)	No. studies	Participants
Meta-analyses of randomised controlled trials						
He et al. (2015) [367]	Postpartum weight retention	Exclusive breastfeeding or mixed feeding vs formula feeding	<b>SMD</b> 0.57 (0.19, 0.94) kg	NR	3	NR
Meta-analyses of prospective cohort studies						
He et al. (2015) [367]	Postpartum weight retention	Exclusive breastfeeding or mixed feeding vs formula feeding	<b>SMD</b> 1.18 (0.74, 1.62) kg	NR	8	NR
Meta-analyses of combined randomised controlled trials and prospective cohort studies						
He et al. (2015) [367]	Postpartum weight retention	Breastfeeding duration 1 to ≤3 months	<b>SMD</b> -0.09 (-0.76, 0.58) kg	NR	4	NR
		Breastfeeding duration 3–6 months	<b>SMD</b> 0.87 (0.57, 1.17) kg	NR	11	NR
		Breastfeeding duration 6 to ≤ 9 months	<b>SMD</b> 0.21 (-0.42, 0.83) kg	NR	3	NR
		Breastfeeding duration 9 to ≤ 12 months	<b>SMD</b> 0.37 (0.14, 0.61) kg	NR	3	NR

Abbreviations used: kg = kilograms; NR = not reported; SMD = standardised mean difference.



# Summary of common and complementary mechanisms of how exposures promote energy balance (weight maintenance) or positive energy balance (weight gain)

Promotes energy balance (weight maintenance)		
<b><i>Aerobic physical activity (including walking)</i></b> <ul style="list-style-type: none"> <li>Increases total energy expenditure</li> <li>Improves appetite sensitivity</li> <li>Favourable effects on lipid metabolism and insulin sensitivity</li> </ul>	<b><i>Wholegrains; Foods containing dietary fibre; Fruit and vegetables</i></b> <ul style="list-style-type: none"> <li>Low energy density</li> <li>Promotes satiety and satiation</li> <li>Modifies digestion, absorption and metabolism favouring energy balance</li> <li>Low glycaemic index</li> <li>Micronutrients influence energy homeostasis</li> </ul>	<b><i>‘Mediterranean type’ dietary pattern</i></b> <ul style="list-style-type: none"> <li>Source of dietary fibre</li> <li>Favourable dietary fat composition</li> <li>Low glycaemic index</li> <li>Lower bioavailability of energy</li> <li>Dietary polyphenol content influencing energy homeostasis</li> <li>Associated with higher levels of physical activity</li> </ul>
Promotes positive energy balance (weight gain)		
<b><i>Sedentary behaviours; Screen time</i></b> <ul style="list-style-type: none"> <li>Decreases total energy expenditure</li> <li>Dysregulates appetite sensitivity</li> <li>Increases exposure to marketing and promotions</li> <li>Part of overall pattern of behaviours related to positive energy balance</li> <li>Displace more active pursuits</li> </ul>	<b><i>Sugar sweetened drinks; Refined grains; ‘Fast foods’</i></b> <ul style="list-style-type: none"> <li>High energy density</li> <li>Lack of compensation for high energy intake</li> <li>May modify fat deposition and fat tissue synthesis</li> <li>Alters hedonics associated with food and drink</li> <li>High glycaemic index</li> <li>Higher intake may displace other foods associated with energy balance</li> <li>Higher degree of processing</li> <li>Embodies a cluster of characteristics promoting positive energy balance</li> <li>Lack of control of preparation and service</li> </ul>	<b><i>‘Western type’ diet</i></b> <ul style="list-style-type: none"> <li>High energy density</li> <li>Unfavourable influences on appetite, for example desensitisation to appetite signals</li> </ul>

This table is a summary only; please see **Appendix 2: Mechanisms** for further details.

Common and complementary mechanisms of dietary and physical activity exposures promoting energy balance (weight maintenance) or positive energy balance (weight gain)

Promotes energy balance (weight maintenance)
<b>Aerobic physical activity (including walking)</b>
<b>Increased total energy expenditure:</b> <ul style="list-style-type: none"><li>Physical activity is a major contributor to total energy expenditure; as total energy expenditure increases, this can lead to energy balance (assuming energy expenditure is equalled by energy intake through foods and drinks), or to negative energy balance (assuming insufficient compensation by energy intake).</li></ul> <b>Appetite sensitivity:</b> <ul style="list-style-type: none"><li>Higher levels of physical activity sensitise individuals to appetite signals, directly potentiating satiety signals via the gastrointestinal tract (reviewed in Blundell <i>et al.</i> (2012) [65] and MacLean <i>et al.</i> (2017) [66]). This promotes energy balance at a higher level of total energy intake (and expenditure). In addition, habitually active people appear to be able to better compensate for higher energy density diets [296].</li><li>Increased physical activity is also associated with shifts in body composition, favouring lean mass over fat mass [297]; increased lean mass relative to fat mass alters resting metabolic rate, energy demand and drive to eat [66]; also see <b>Section 3</b> on fundamental concepts.</li></ul> <b>Lipid metabolism and insulin sensitivity:</b> Endurance aerobic activity, such as long-distance running, promotes fat oxidation, which may explain the favourable effects of such activities on changes to body fat (for a summary, see Hespanhol <i>et al.</i> (2015) [266]). In addition, increased physical activity has beneficial effects for <i>insulin</i> sensitivity [298].
<b>Wholegrains; Foods containing dietary fibre; Fruit and vegetables</b>
<b>Low energy density foods:</b> Eating foods with lower energy density reduces the likelihood of passive overconsumption. In general, people tend to consume roughly the same amount of food from day to day, measured by bulk and weight, indicating that appetite is more influenced by mass of food (weight and volume) than the intrinsic amount of energy, at least in the short to medium term [67, 145]. <b>Satiety and satiation:</b> <ul style="list-style-type: none"><li>Increased satiation – the termination of a current meal owing to a feeling of fullness – when eating wholegrains may be due to the additional chewing required, related to their fibre content, particle size and structural integrity. This may be modified by the degree of processing. (For a summary, see Karl and Saltzman (2012) [115].)</li><li>Fibre may increase satiation by increasing chewing, slowing gastric emptying and elevating stomach distension, and stimulating cholecystokinin release [155–158].</li></ul> <b>Modified digestion, absorption and metabolism:</b> <ul style="list-style-type: none"><li>Eating a meal of barley kernels (relative to white bread) led to increased release of GLP-1, as well as depressing energy intake and hunger over two subsequent meals [116]. However, these results may not be applicable to all wholegrains in general.</li><li>Some limited evidence in human trials has shown that consumption of wholegrains can favourably modulate glycaemic response to both the current and the subsequent meal. For example, a favourable (depressed) glycaemic response was observed following a standardised breakfast when barley kernels were consumed the previous evening compared with an equivalent amount of refined-grain wheat bread [117, 118] (for a summary, see Karl and Saltzman (2012) [115]). However, these results may be specific to barley kernels and not wholegrains in general.</li><li>It is hypothesised that fermentation of wholegrains in the bowel influences appetite. Gut microbiota can ferment certain wholegrain fibres to produce <i>short chain fatty acids</i>. These can influence glucose and lipid metabolism and stimulate the secretion of gut <i>hormones</i> implicated in appetite regulation, gastrointestinal transit and glucose metabolism, such as PYY and GLP-1 [119].</li><li>The increased viscosity of soluble fibre can reduce the overall rate and extent of digestion, which may also result in a blunted post-prandial glycaemic and insulinaemic response to carbohydrates [158].</li><li>Fibre-induced delayed absorption and the resultant presence of <i>macronutrients</i> in the distal small intestine, known as the ileal brake, mediate the release of several gut hormones such as PYY and GLP-1 [159].</li></ul> <b>Low glycaemic index:</b> Most non-starchy vegetables tend to have a low glycaemic index; foods with lower glycaemic indices tend to promote favourable insulin responses and post-prandial blood glucose profiles, enhancing appropriate appetite regulation [146]. <b>Micronutrients:</b> Fruits and vegetables contain high concentrations of a range of micronutrients and other phytochemicals, including antioxidants and phytoestrogens, that may also have a beneficial influence on energy homeostatic pathways [147, 148]. <ul style="list-style-type: none"><li>Several flavonoid subclasses have been shown to decrease energy intake, increase glucose uptake in muscle in vivo and decrease glucose uptake in adipose tissue in vivo (animal models and short-term human studies) (for a summary, see Bertoia <i>et al.</i> (2016) [123]).</li></ul>
<b>‘Mediterranean type’ dietary pattern</b>
<b>Source of dietary fibre:</b> The ‘Mediterranean diet’ is a dietary pattern rich in plant foods, which provide a high amount and wide variety of both soluble and insoluble dietary fibres (see <b>Foods containing dietary fibre</b> above). <b>Dietary fat composition:</b> Typically, the ‘Mediterranean type’ dietary pattern is high in unsaturated fatty acids relative to saturated fatty acids. Experimental studies in humans have demonstrated that dietary fatty acid composition can influence fat oxidation and daily energy expenditure; in particular oleic acid, a mono-unsaturated fatty acid, may increase oxidation and energy expenditure [169, 170]. This is consistent with results from the PREDIMED trial, which showed no adverse effect on body weight from long-term adherence to a ‘Mediterranean type’ dietary pattern, supplemented with either olive oil or nuts, compared with the control group [171]. <b>Low glycaemic index:</b> The ‘Mediterranean type’ dietary pattern has a low glycaemic load [172]; foods with lower glycaemic indices tend to promote favourable insulin responses and post-prandial blood glucose profiles, enhancing appropriate appetite regulation [146]. <b>Available energy:</b> Some foods common in the ‘Mediterranean type’ dietary pattern, for example nuts and seeds, resist digestion and absorption, leading to lower bioavailability of energy [173-175]. <b>Dietary polyphenol content:</b> A cross-sectional study within the PREDIMED trial reported a significant inverse association between urinary polyphenol concentrations and body weight [176]. It is suggested that the diversity in structure and function of polyphenols mean they could influence a variety of metabolic pathways, such as inhibition of lipogenesis, stimulation of catabolic pathways, reduction of chronic inflammation and upregulation of uncoupling proteins. However, further studies are required to confirm the roles and interactions of the polyphenol group; for a review of existing studies, see Guo <i>et al.</i> (2017) [176]. <b>Associated with higher levels of physical activity:</b> Traditional lifestyles in the Mediterranean region, similar to other traditional lifestyles around the world, are associated with higher levels of habitual physical activity. Increased physical activity leads to favourable shifts in body composition, appetite regulation and insulin sensitivity (see <b>Aerobic physical activity</b> above and Section 3 on fundamental concepts).
Promotes positive energy balance (weight gain)
<b>Sedentary behaviours; Screen time</b>
<b>Decreased total energy expenditure:</b> Physical activity is the main variable contributor to total energy expenditure. If physical activity level is low (through increased sedentary time) then total energy expenditure will decrease; this can lead to positive energy balance (assuming insufficient compensation by decreased energy intake). <b>Appetite dysregulation:</b> Lack of physical activity (through increased time spent sedentary) impairs satiety sensitivity and appetite signals [65]. At low levels of energy expenditure (and when food and drink are freely available), adequate suppression of appetite to maintain energy balance may be compromised [66, 296] (also see Section 3 on fundamental concepts). <b>Exposure to marketing and promotions:</b> Time spent watching television or using other screen devices may increase exposure to marketing of foods and drinks that promote weight gain, leading to increased preference for, purchasing of and intake of such foods, at least in children and adolescents [318, 319]. <b>Pattern of behaviours:</b> Time spent watching television or using other screen devices may be accompanied by relatively uninhibited consumption of energy-dense foods, for example through distraction, which may be eaten in large portion sizes [320-322], and can occur in the absence of advertising or marketing [343]. <b>Displacement:</b> Time spent watching television or using other screen devices may displace opportunities for more active pursuits [316, 317, 344].
<b>Sugar sweetened drinks; Refined grains; ‘Fast foods’</b>
<b>High energy density foods:</b> Consuming foods and drinks with higher energy densities increases the likelihood of passive overconsumption. In general, people tend to consume roughly the same amount of food from day to day, measured by bulk and weight, indicating that appetite is more influenced by mass of food (weight and volume) than the intrinsic amount of energy, at least in the short to medium term [67, 145]. <b>Lack of compensation:</b> Energy from sugars may not be compensated for in the same way when consumed in a soft drink as when consumed as part of a solid meal: energy in liquid form appears to be less effective in inducing satiation or satiety [71], and so may promote excess energy intake. <b>Modified fat deposition:</b> It is hypothesised that consumption of high fructose corn syrup or sucrose, the key sweetening agents of many soft drinks, may promote the deposition of liver, muscle and visceral fat and an increase in serum lipids independently of an effect on body weight (reviewed in Malik and Hu (2015) [206]). <b>Altered hedonics:</b> Increased intake of high-sugar foods and drinks has been associated with greater reward response and decreased inhibitory response to such foods and drinks [207, 208]. <b>High glycaemic index:</b> Refined grain products frequently have a high glycaemic index, provoking high insulin responses and a fast glucose decline [151]. It is hypothesized that these properties could increase hunger and enhance lipogenesis (see next point), thereby promoting obesity (for a summary, see Fogelholm <i>et al.</i> (2012) [160]). <b>Fat tissue synthesis:</b> Animal feeding studies suggest that consumption of refined grain products can promote fat synthesis even when total energy intake is unchanged [180]. <b>Displacement:</b> It is possible that higher intakes of refined grains reflect lower consumption of other dietary factors that might promote energy balance and protect against weight gain (see also Section 5.2). <b>Degree of processing:</b> Highly processed foods, such as those typically served at ‘fast foods’ outlets (for example, French fries (chips) and nuggets), have generally undergone industrial processing and may be unrecognisable from their original plant or animal source. They are frequently high in energy (see point above). Data reported from the EPIC cohort show that high levels of trans fatty acids in the blood were associated with a lower likelihood of weight loss and increased risk of weight gain [229]; plasma trans fatty acids were interpreted as a biomarker of dietary exposure to industrially processed foods. <b>Cluster of characteristics:</b> Excess energy intake is also promoted through a cluster of characteristics embodied by ‘fast foods’, such as being highly palatable, served in large portions, high in energy density (see above point), affordable and easy to access. ‘Fast foods’ are also frequently consumed alongside sugar sweetened drinks, which have their own positive energy balance promoting effects. <b>Preparation and service:</b> Increased intake of energy is observed when eating in ‘fast food’ outlets and restaurants [230-232]. This may be mediated by environmental cues which prompt increased energy intake [233] such as offers to increase portion size or add more food items, or lack of control over initial portion size [234] or ingredients (see <b>Sections 7.5, 7.6</b> and <b>7.8</b> ).
<b>Sedentary behaviours; Screen time</b>
<b>High energy density foods:</b> <ul style="list-style-type: none"><li>Eating foods with higher energy density increases the likelihood of passive overconsumption. In general, people tend to consume roughly the same amount of food from day to day, measured by bulk and weight, indicating that appetite is more influenced by mass of food (weight and volume) than the intrinsic amount of energy, at least in the short to medium term [67, 145].</li><li>Meat, and some meat products in particular, may be energy dense, especially if high in fat, and thereby may increase total energy intake [256].</li></ul> <b>Unfavourable influences on appetite:</b> <ul style="list-style-type: none"><li>Prolonged consumption of a high-fat diet may desensitise individuals to a number of appetite signals, such as release of gastrointestinal hormones [257].</li><li>Increased intake of high-sugar and high-fat foods has been associated with greater reward response and decreased inhibitory response to such foods [207, 208].</li><li>The orosensory properties of fat, and foods high in fat, improve palatability [207, 258, 259] and may lead to voluntary overconsumption [260]. Similar preferences have been observed for palatable foods high in sugars [207, 261]. However, replication of these results in human studies is limited.</li><li>Dietary protein has a stronger satiating effect than other macronutrients (fats and carbohydrates) [262]; as meat is high in protein it is possible that diets containing meat low in fat may have a beneficial impact on appetite cues. However, some small human trials suggest that meat- or vegetarian-based sources of protein do not differ in their satiating effects [263-265].</li></ul>