EVIDENCE REVIEW

The Australian Prevention Partnership Centre
Systems and solutions for better health

A rapid review of evidence

Beyond overweight and obesity: HEAL targets for overweight and obesity and the six HEAL objectives

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An evidence review commissioned by the Centre for Population Health, NSW Ministry of Health, and brokered by the Sax Institute for The Australian Prevention Partnership Centre

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Abbreviations

CCS	case-control study
CS	cohort study
CVD	cardiovascular disease
DR	dose response
DRR	dose response relationship
HEAL	Healthy Eating Active Living Strategy 2013–2018
HR	hazard ratio
LDRR	linear dose response relationship
LO	lower odds
LR	lower risk
MA	meta-analysis
NCCS	nested case-control study
NLDRR	non-linear dose-response relationship
NR	not reported
NSW	New South Wales
OR	odds ratio
PAR	population attributable risk
RR	relative risk
SR	systematic review

Executive summary

This rapid review examined the broader health and other outcomes, beyond overweight and obesity, likely to accrue as a consequence of implementation of the NSW Healthy Eating Active Living Strategy 2013–2018. Specific questions were to:

• Identify the additional health conditions and wellbeing indicators (beyond overweight and obesity) where there is strong evidence that meeting the HEAL targets for overweight and obesity and six objectives will improve outcomes

• Provide information on the level of change in the targets and HEAL objectives that are associated with better outcomes for the health conditions and wellbeing indicators. The review was based on evidence retrieved from the main databases of biomedical and health economic peer-reviewed literature from January 2000 to current, supplemented with relevant local and international reports.

The review found:

1. Considerably less research has been conducted into the impact of overweight and obesity and associated health behaviours among children and adolescents compared with adults; nonetheless improved outcomes would occur for metabolic risk factors, dental health, pre-hypertension/hypertension, cardiovascular disease risk factors, depression, rates of mortality in hospitalised children, bullying and otitis media

2. A substantial number of direct and indirect health and other outcomes may occur as a result of stabilisation and/or reduction of the prevalence of overweight and obesity among adults. Non-health benefit areas include disability payments, absenteeism, worker productivity, workplace injuries, healthcare costs and insurance claims (see 'Findings at a glance')

3. A wide range of additional benefits may occur as a result of meeting HEAL objectives 1-5. The review found: a relatively limited amount of evidence for reducing intake of energy dense nutrient poor food [EDNP]; substantial evidence linking increased consumption of fruit and vegetables to a large number of reduced morbidities and mortality (although not to weight status); a large body of evidence linking the intake of sugar-sweetened beverages to a number of health outcomes, including type 2 diabetes, pancreatic cancer, coronary heart disease, stroke and hypertension; and an abundance of evidence supporting health and other outcomes from increased physical activity and reduced sedentary behaviours (see 'Findings at a glance')

4. There is a paucity of systematic review evidence linking community awareness (HEAL Objective 6) directly to outcomes beyond health behaviours and their antecedents. Conducting such a review across the domains of physical activity, healthy eating and obesity would be very worthwhile but the detailed additional analysis required was outside the scope of the current review. Selected population datasets which show community awareness levels are presented.

5. Estimation of lives saved and diseases prevented through addressing the HEAL strategy objectives suggest that every year in NSW, for five chosen case study examples alone, the following would be prevented:

• 37 cases of colorectal cancer (through increased physical activity)

• 1003 cases of coronary heart disease (through improved vegetable consumption) and 422 cases of coronary heart disease (through improved fruit consumption)

• 510 cases of type 2 diabetes (through reduced sugar-sweetened beverage consumption)

• 266 cases of type 2 diabetes in men and 200 cases in women (through a 5% reduction in overweight)

• 185 cases of type 2 diabetes in men and 186 cases in women (through a 5% reduction in obesity)

• 366 deaths from all causes (through reduced sedentary behaviour)

6. Although evidence regarding the effectiveness of the inter-sectoral approach to achieving changes in overweight and obesity is only beginning to emerge, this approach is strongly supported by prevailing global expert opinion. The impact of achieving the HEAL targets for overweight and obesity as well as other direct and indirect health and non-health impacts is likely to be substantial. In addition, although not additive, the impacts are likely to be cumulative.

Review findings at a glance

Meeting HEAL Target 1 – reduce overweight and obesity rates of children and young people (5–16 years)

Considerably less research has been conducted into the health impact of reducing overweight and obesity among children and adolescents compared with adults. Nevertheless, the findings indicate that reducing the prevalence of overweight and obesity among children and adolescents will result in additional benefits in the following areas:

- Metabolic risk factors in children
- Dental health
- Pre-hypertension and hypertension
- Cardiovascular disease risk factors
- Depression
- Higher mortality in hospitalised children
- Bullying
- Otitis media.

Meeting HEAL Target 2 – stabilise and then reduce overweight and obesity rates in adults

A substantial number of direct and indirect health and other outcomes were identified as accruing as a result of stabilisation and/or reduction of the prevalence of overweight and obesity. These include:

- Type 2 diabetes
- All-cause mortality
- Endometrial cancer
- Second primary breast and colorectal cancers
- Gastric cancer
- Lumbar radicular pain and sciatica
- Cataracts
- Biliary tract disease
- Osteoarthritis (knees)
- Quality of life
- Prostate cancer
- Colorectal cancer survival
- Cardiovascular disease
- Renal cell cancer
- Bladder cancer
- Thyroid cancer
- Infant death.

Non-health outcomes that may be improved through reductions in overweight and obesity include:

- Disability payments

- Absenteeism
- Productivity
- Workplace injuries
- Health care costs
- Insurance claims.

Meeting HEAL Objective 1 – reduce intake of energy dense nutrient poor (EDNP) food and drinks[a]

The review found a limited amount of evidence relating reduced intake of energy dense nutrient poor (ENDP) food to health and other outcomes, including and beyond overweight and obesity. Much of the literature is centred on the effects of specific nutrients that are likely to be high in EDNP foods, but such studies were excluded from this review as they were too extensive and complicated in their findings. Nevertheless, a number of studies indicated that reducing consumption of EDNP foods would be associated with improved outcomes in the following areas:

- Metabolic syndrome
- Type 2 diabetes
- Renal cell carcinoma
- Coronary heart disease mortality
- Prostate cancer
- Stroke.

Meeting HEAL Objective 2 – increase consumption of fruit and vegetables (F&V)

The review found substantial evidence linking increased consumption of F&V to reduced morbidities and mortality, although not weight status. Increased consumption of F&V was associated with the following health outcomes:

- All-cause mortality
- Cardiovascular mortality
- Type 2 diabetes
- Gastric cancer
- Colorectal cancer
- Breast cancer
- Oral cancer
- Lung cancer
- Oesophageal cancer
- Endometrial cancer
- Non-Hodgkin's lymphoma.

[a] EDNP drinks were not considered within this objective in this review as HEAL Objective 6 relates to sugar-sweetened beverages separately.

Evidence for non-health outcomes was less extensive and related to:

- Healthcare costs and
- Agricultural sector gains.

Meeting HEAL Objective 3 – increase intake of water in preference to sugar-sweetened drinks

The review found a large body of evidence linking the intake of sugar-sweetened beverages to a number of health outcomes, including:

- Type 2 diabetes
- Pancreatic cancer
- Coronary heart disease
- Gout
- Kidney stones
- Stroke
- Hypertension
- Dental erosion in children.

Meeting HEAL Objective 4 – increase incidental, moderate and vigorous physical activity

The review found an abundance of evidence supporting health and other outcomes of increased physical activity. The areas in which gains would accrue include the following:

- All-cause mortality
- Healthcare costs
- Absenteeism
- Cardiovascular mortality
- Life satisfaction
- Stroke
- Sleep duration and quality
- Coronary heart disease
- Self-esteem
- Hypertension
- Body image
- Metabolic syndrome
- Type 2 diabetes
- Asthma
- Hip fractures
- Dementia
- Cognitive decline
- Lung cancer

- Pancreatic cancer
- Breast cancer
- Colon cancer
- Gastroesophageal cancers
- Renal cancer
- Bladder cancer
- Prostate cancer
- Endometrial cancer
- Preeclampsia
- Gestational diabetes
- Ovarian cancer
- Erectile dysfunction
- Depression
- Anxiety
- Health-related quality of life.

Meeting HEAL Objective 5 – reduce time in sedentary behaviours

The review also found that reducing sedentary behaviours would likely address the following health and non-health outcomes:

- All-cause mortality
- Cardiovascular disease and mortality
- Type 2 diabetes
- Colon cancer
- Endometrial cancer
- Lung cancer
- Metabolic syndrome
- Breast cancer
- Ovarian cancer
- Prostate cancer
- Depressive symptoms
- High blood cholesterol
- Hypertension
- Physical fitness
- Academic achievement
- Cognitive performance
- Anti-social behaviour
- Eating habits.

Meeting HEAL Objective 6 – increase community awareness of healthy eating and physical activity as protective factors against chronic disease

The review found a paucity of evidence linking community awareness directly to outcomes beyond health behaviours and their antecedents. No relevant systematic reviews have been published to date in the peer-reviewed literature ('absence of evidence' rather than 'evidence of absence'). Conducting such a review across the domains of physical activity, healthy eating and obesity, whilst extensive, would be a very worthwhile undertaking; the detailed additional analysis required for this fell outside the scope of the current review. Nonetheless relevant data on community awareness in NSW are provided.

Estimation of lives saved and diseases prevented annually through a fully implemented HEAL strategy – selected case studies

The measures of association identified in addressing review question 1 (RQ1) were used as inputs to answer RQ2; i.e. to provide measures of the population impact of such changes. Analyses were undertaken to develop estimates of the number of cases of disease or deaths that could be avoided if the HEAL targets and objectives were met. One outcome was selected for each of the HEAL targets and objectives (excluding community awareness) addressed in RQ1; accordingly, five modelled studies of estimated diseases prevented or lives saved annually were prepared. The modelling from these theoretical case studies suggests that:

- 37 cases of colorectal cancer annually in NSW would be prevented through a 15% reduction in inadequate physical activity
- 1003 cases of coronary heart disease annually in NSW would be prevented through a 25% reduction in insufficient vegetable consumption and 422 cases would be prevented through a 25% reduction in insufficient fruit consumption
- 510 cases of type 2 diabetes annually in NSW would be prevented through a 25% reduction in consumption of sugar-sweetened beverages
- 366 deaths (from all causes) annually in NSW would be prevented through a 25% reduction in the proportion sitting for 10 or more hours a day
- 266 cases of type 2 diabetes in men and 200 cases in women in NSW per year would be prevented by a 5% reduction in overweight while 185 cases in men and 186 cases in women would be prevented by a 5% reduction in obesity.

Background

The NSW Healthy Eating and Active Living Strategy (HEAL): Preventing overweight and obesity in New South Wales 2013-2018[1] provides a whole-of-government framework to promote and support healthy eating and active living in NSW and reduce the impact of lifestyle-related chronic disease. It is the first time NSW has had a whole-of-government approach to address overweight and obesity. The Strategy aims to encourage and support the community to make healthy lifestyle changes at a personal level, and create an environment that supports healthier living, through better planning, built environments and transport solutions. The Strategy aims to ensure that everyone has opportunities to be healthy through the delivery of evidence-based, interactive and relevant programs and has four key strategic directions:

- Environments to support healthy eating and active living
- State-wide healthy eating and active living support programs
- Healthy eating and active living advice as part of routine service delivery
- Education and information to enable informed, healthy choices.

The Strategy has been designed to assist the NSW Government to implement policies and programs across a range of government agencies and health services and to work in partnership with the academic and non-government sectors. The strategy acknowledges that physical activity and healthy eating are important factors in individual and population health; and that they contribute to the prevention of many chronic diseases and may enhance an individual's quality of life. The Strategy recognises that actions to influence healthy eating and physical activity require a comprehensive approach. This recognises the interaction of individual, societal and environmental factors that impact directly and indirectly upon behaviours that have led to weight gain over the past 15–20 years in NSW.

Policy context

The Strategy is informed by and complements four key policy and strategic initiatives:

- *NSW 2021: A plan to make NSW number one*
- National Partnership Agreement on Preventive Health (direct Federal Government support ceased in June 2014)
- National Partnership Agreement for Closing the Gap in Indigenous Health Outcomes
- NSW Government Aboriginal Health Plan 2013–2023.

The NSW Government has developed a 10-year strategic plan that details the Government's commitment to improving opportunities to be healthy and quality of life for the NSW population. Goal 11 recognises the need to "keep people healthy and out of hospital". The Strategy directly contributes to this goal and in particular provides a framework of actions to achieve the following targets relating to levels of overweight and obesity (a focus of this review):

- Reduce overweight and obesity rates of children and young people (5–16 years) to 21% by 2015
- Stabilise overweight and obesity rates in adults by 2015, and then reduce by 5% by 2020.

The Strategy also incorporates a range of other actions that address the targets of *NSW 2021: A plan to make NSW number one* (these actions are not the focus of this review):

- Increase participation in sport, recreational, arts and cultural activities in rural and regional NSW by 10% and in Sydney by 10% from 2010 to 2016
- More than double the mode share of bicycle trips made in the Greater Sydney region, at a local and district level by 2016
- Increase the mode share of walking trips made in the Greater Sydney region at a local and district level to 25% by 2016
- Increase the proportion of total journeys to work by public transport in the Sydney Metropolitan Region to 28% by 2016

- Increase the percentage of the population living within 30 minutes by public transport of a city or major centre in metropolitan Sydney.

The NSW HEAL Strategy focuses on prevention, recognising that there is much to be gained by the prevention of chronic diseases for the individual, the community and the health system. There is also growing evidence on the effectiveness and cost-effectiveness of improving population health through prevention initiatives.

Six objectives (that are the focus of this review) within the NSW HEAL strategy are aimed at achieving improvements in nutrition and physical activity levels:

1. Reduce intake of energy dense nutrient poor food and drinks
2. Increase consumption of fruit and vegetables
3. Increase intake of water in preference to sugar-sweetened drinks (beverages)
4. Increase incidental, and moderate and vigorous physical activity
5. Reduce time in sedentary behaviours
6. Increase community awareness of healthy eating and physical activity as protective factors against chronic disease.

Although having a primary focus on the prevention of overweight and obesity, the HEAL Strategy is framed towards improving nutrition and physical activity. It is acknowledged that this work will have broader impacts than the health benefits associated with prevention of overweight and obesity. These impacts are expected to include impacts on other health conditions, such as cardiovascular disease, cancers, hypertension, mental health and arthritis. Non-health impacts are expected to include demand for health services and financial savings for government and individuals. Gaining an understanding of the broader impacts of implementing the HEAL Strategy is important to justify ongoing investment in these areas of prevention.

The aim of this rapid review is to articulate some of the potential broader health impacts of the NSW HEAL Strategy and demonstrate the benefits of this approach for the NSW government.

Evidence on inter-sectoral interventions

There is no definitive evidence of the effectiveness of broad-scale inter-sectoral interventions. Indeed, achieving full implementation of cross-government and sector intervention is challenging,[2-4] and many efforts do not move beyond the policy discussion stage. Nonetheless, there is agreement that broad, cross-government actions are needed to counteract the health and other impacts of poor nutrition, low levels of physical activity and sedentariness. Obesity is a complex ("wicked") public health problem that demands complex solutions.[5] As an example of the challenges in developing evidence in this area, global policy actions for obesity prevention were recently summarised in an OECD report.[6] This report focused predominantly on obesity prevention but indicated that a mix of strategies, financial incentives, health screening, and health insurance subsidies might be necessary parts of current inter-sectoral approaches to preventing obesity.

For at least two decades complex interventions to prevent non-communicable disease have required working with partners outside of the health sector. These inter-sectoral interventions were developed as part of broad strategic approaches to increasing physical activity at the population level, improving nutrition and healthy eating patterns, and preventing obesity at the population level.

The rationale behind inter-sectoral interventions is based on the premise that the conditions for poor health include causal factors outside the purview of the health system or health professionals. This means that, to create a healthy diet, action must not be limited to health and nutrition but also cover agriculture, food production, food distribution, food taxation and relevant food policy development. Similarly to create a more physically active society, work in partnership between health and sport, urban planning, public transportation, school education, crime prevention, local municipalities and other sectors is required. This approach is also important to obesity prevention efforts, as the causal factors leading to obesity are decreased energy expenditure and increased energy intake, and thus require a diverse and multifaceted approach.

Evaluations of inter-sectoral interventions and of the partnerships between sectors to improve healthy eating and increased physical activity are scarce. There are a few complex program evaluations that demonstrate co-partnership at the community level and some improvements in physical activity or dietary outcomes that result. There are no summary reviews or summary papers that synthesise this evidence in a way that provides guidance to policymakers on the net effects of this kind of inter-sectoral intervention. Given the complex causal chains leading to the behavioural and societal risk factors for chronic disease, it is not surprising that there is limited evidence in this space. For this reason there are no systematic reviews, research syntheses or meta-analyses that describe inter-sectoral action in a summative way in relation to the HEAL indicators.

Review methods and report development

The Prevention Research Centre at The University of Sydney produced a rapid overview to articulate some of the potential broader health and non-health impacts of the NSW HEAL Strategy and demonstrate the benefits of this approach for the NSW Government.

Project brief

The brief required the review to address two specific questions:

Review Question 1

- What are the additional health conditions and wellbeing indicators (beyond overweight and obesity) for which there is strong evidence that meeting the HEAL targets and six objectives (see Background, page 12) will improve outcomes?

Review Question 2

- What is the level of change in the targets and HEAL objectives that are associated with better outcomes for the health conditions and wellbeing indicators identified in Question 1?

The scope of this review indicated the inclusion of evidence from whole-of-population, inter-sectoral interventions. A number of countries have adopted inter-sectoral approaches and policies to address the promotion of physical activity, nutrition and obesity prevention. While reports document these approaches and the rationale for this approach, these initiatives are only recent and evaluations of the effectiveness of these approaches are not available. Thus the methods of inter-sectoral action, partnerships and cross-agency policy are assumed to be useful in the context of chronic disease prevention, based on public health practice and experience, rather than on systematic scientific evidence.

It was proposed that population-level research evidence demonstrating the consistency of associations between physical activity and dietary behaviours and health and other outcomes be used as the basis of this review. This review therefore provides evidence of the health and other outcomes that might be achieved through the HEAL strategies and via achievement of the HEAL target 1 and objectives, as its main purpose.

The rapid nature of this review and the lack of available evidence around the effectiveness of inter-sectoral approaches to identify associations between overweight and obesity and physical activity and nutrition behaviours and other health outcomes, mean that this report may not report on all possible associations. It does mean, however, that the measures of association identified in the review may be an underestimate of the cumulative effect of inter-sectoral action.

The project was undertaken between August and October 2014.

Review Question 1

Searches were undertaken to identify articles reporting on an association with health conditions and other (direct or indirect) outcomes associated with:

- Reducing overweight and obesity
- Consumption of energy dense nutrient poor foods
- Consumption of fruit and vegetables
- Consumption of sugar-sweetened drinks
- Incidental, moderate and vigorous physical activity
- Sedentary behaviours
- Community awareness of healthy eating and physical activity as protective factors against chronic disease.

Searches were undertaken to identify articles reporting on associations with any of (but not limited to) the following direct health outcomes:

- Cardiovascular disease

- Cancer
- Hypertension
- Anxiety or depression
- Arthritis
- Type 2 diabetes
- Morbidities
- Mortality
- Wellbeing indicators (e.g. quality of life, falls).

The searches also aimed to identify associations with other direct or indirect outcomes, e.g. performance at school or economic benefits. The searches were restricted to studies published between 2000 and September 2014. Searches of Medline, Pre-Medline and PsychINFO were undertaken using Medical Subject Headings (MeSH) terms and key word (phrase searching) to identify recent reviews of reviews, systematic reviews of population studies and individual population studies. The search strategies were slightly different for each target/objective and the most recent data of the strongest evidence were extracted wherever possible and a brief description provided with the findings. Details of the number of articles identified in the search strategies are in Appendix 1. The articles retrieved were screened on the basis of title and abstract and, where required, full articles were retrieved. Only those articles that reported on the association using appropriate statistical methods were included. Articles reporting no association were excluded.

Unless otherwise specified, the associations represent risk (or odds) of contracting the disease.

The searches yielded a significant number of returns for overweight and obesity, physical activity, sedentary behaviour and fruit and vegetables. The searches were less productive in identifying studies reporting on health outcomes associated with reducing intake of energy dense nutrient poor food, increasing intake of water in preference to sugar-sweetened drinks, and increasing community awareness of healthy eating and physical activity as protective factors against chronic disease.

For each health outcome (or other indirect or direct outcome) for which an association was identified key articles were selected for information on the level of association. Meta-analyses (MA) and systematic reviews (SRs) were used where available. If more than one MA or SR was identified, the most recent article was used. For some of the areas of focus, there were no MAs or SRs so the measure of association was determined from good-quality population studies. General non-database searches were also undertaken to identify key reports providing information relevant to the review.

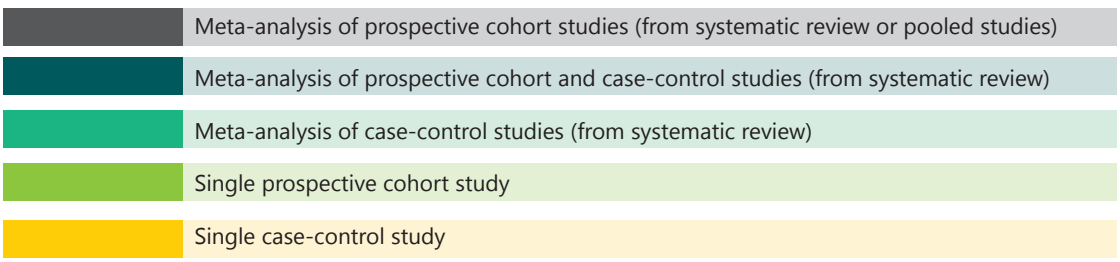
Caution is advised in use of the data on economic outcomes as many of these data come from outside of Australia. Different findings may be expected if similar studies were carried out in Australia.

Tabulations of the included articles

Details of the articles used to provide information on the level of association is contained in Appendix 2.

Graphical representation of findings

For each HEAL objective, a figure is used to illustrate the strength of association between the relevant behaviour (HEAL target or objective) and direct health outcome. The figures include shading to indicate the type of evidence upon which the association is based, with the increasingly darker shading indicating that the measure of association comes from 'stronger' evidence. The legend is shown below:



Review Question 2

Calculations of Population Attributable Risk (PAR) were undertaken to answer Review Question 2 using quantified associations identified when answering Review Question 1, and using NSW population health data where available. PAR provides an estimate of the number or proportion of cases that is attributable in a population to a particular risk factor; for example, the proportion of lung cancer that is attributable to smoking. It is used in epidemiology to show how the reduction or elimination of a risk factor would benefit a population.

A complete description of the methodology, including the formulas used, is included in Appendix 3.

SECTION 1 – REVIEW QUESTION 1

Meeting the HEAL targets and objectives: Overweight and obesity

Introduction

Identified overviews and syntheses of multiple health and other outcomes were initially searched for in relation to meeting the targets for overweight and obesity in children and adults. However, very few such articles were identified in the peer-reviewed literature. The most recent evidence from meta-analyses and systematic reviews on individual health outcomes was identified from 2012 onwards; the publication period restriction was applied due to the vast amount of literature for the association between overweight and obesity and health outcomes.

Health outcomes – Adults

Evidence from overview paper (Dixon 2010)[7]

Obesity is associated with a broad range of health issues ranging from specific diseases such as type 2 diabetes and hypertension, to impaired quality of life, psychosocial disturbance, and limited access to quality care.[7] The magnitude of the associations is indicated in 'Table 1[c]' (from Dixon 2010).[7]

Table 1: The relative risk of some of the comorbidities, conditions and risks associated with obesity

Relative risk >5	Relative risk 2-5	Relative risk 1-2
Type 2 diabetes	All-cause mortality	Cancer mortality
Dyslipidaemia	Hypertension	Breast cancer
Obstructive sleep apnoea	Myocardial infarction and stroke	Prostate and colon cancer in men
Breathlessness	Endometrial carcinoma in women and hepatoma in men	Impaired fertility
Excessive daytime sleepiness	Gallstones and complications, including cancer	Obstetric complications, including fetal abnormalities
Obesity hypoventilation syndrome	Polycystic ovary syndrome	Asthma
Idiopathic intracranial hypertension	Osteoarthritis (knee)	Gastroesophageal reflux
Non-alcoholic steatohepatitis	Gout	Aesthetic risk

- Increasing levels of obesity have a major impact on patients' physical, mental, psychosocial and economic health. It has been estimated that obesity has a greater negative impact on quality of life than 20 years of ageing (single study cited in Dixon 2010).[7] The negative impact on quality of life remains after accounting for demographics, health habits, medical conditions and depression.
- In Australia, as measured with DALYs, deaths and burden of disease related to overweight and obesity are largely related to co-morbidities, with diabetes (40%), coronary heart disease (34%) and stroke (11%) leading conditions contributed to by obesity, and with cancer and osteoarthritis additional contributors
- Indirect societal costs:
  Absenteeism, workplace injuries, disability payments and loss of productivity.

c. Dixon (2010) does not identify the sources of the relative risks (measures of association) in Table 1; in addition the table is presented in the background to the article rather than the summary, hence it is not clear if the findings in Table 1 are indeed a summary of the papers included in the review

Cost of obesity in Australia

- The economic cost was estimated in 2008 to be $8.283 billion. Of this, productivity costs were estimated as $3.6 billion (44%), health system costs were $2 billion (24%) and carer costs were $1.9 billion (23%). Deadweight losses from transfers (taxation revenue forgone, welfare and other government payments) were $727 million (9%) and other indirect costs were $76 million (1%). The net cost of lost wellbeing (the dollar value of the burden of disease, netting out financial costs borne by individuals) was valued at a further $49.9 billion, bringing the total cost of obesity in 2008 to $58.2 billion. Among States and Territories, total costs were largest in NSW at $19.0 billion – including $2.7 billion (14%) in financial costs and $16.3 billion (86%) in net costs of lost wellbeing[8] (cited in Dixon 2010).[7]
- Other estimates ($21 billion) of the economic burden for the year 2005 were published in 2010 by Colagiuri and colleagues. These cost estimates included overweight as well as obesity (refer to Appendix 5).

Evidence from other articles (all meta-analyses)

- A meta-analysis of overweight and obesity and all-cause mortality indicated that the summary Hazard Ratios (HRs) indicated a protective decrease in risk, 0.94 (95% CI, 0.91-0.96) for overweight, and an increased risk, HR=1.18 (95% CI, 1.12–1.25) for obesity (all grades combined), 0.95 (95% CI, 0.88–1.01) for grade 1 obesity, and 1.29 (95% CI, 1.18–1.41) for grades 2 and 3 obesity.[9] These findings persisted when limited to studies with measured weight and height that were considered to be adequately adjusted. Relative to normal weight, both obesity (all grades) and grades 2 and 3 obesity were associated with significantly higher all-cause mortality. Grade 1 obesity overall was not associated with higher mortality, and overweight was associated with significantly lower all-cause mortality.
- Metabolically healthy obese individuals (relative risk [RR], 1.24; 95% CI, 1.02–1.55) had increased risk for all-cause mortality and CVD events or both compared with metabolically healthy normal-weight individuals, when only studies with 10 or more years of follow-up were considered.[10] Much greater risks were seen in all metabolically unhealthy groups: normal weight (RR, 3.14; CI, 2.36–3.93), overweight (RR, 2.70; CI, 2.08–3.30), and obese (RR, 2.65; CI, 2.18–3.12).
- Overweight and obesity and incident type 2 diabetes in all adults: Overweight men (RR 2.40; 95% CI 2.12 –2.72) and women (3.92; 3.10–4.97) were found to be at increased risk of type 2 diabetes.[11] Similarly, obese men (6.74; 5.55–8.19) and women (12.41; 8.23–14.96) were at increased risk.
- Obesity and incident type 2 diabetes in metabolically healthy and unhealthy obese adults: The pooled adjusted RR for incident type 2 diabetes was 4.03 (95% CI 2.66–6.09) in healthy obese adults and 8.93 (6.86–11.62) in unhealthy obese compared with healthy normal-weight adults.[12] Although there was between-study heterogeneity in the size of effects (I2=49.8%; P=0.03), RR for healthy obesity exceeded one in every study, indicating a consistently increased risk across study populations. Metabolically healthy obese adults show a substantially increased risk of developing type 2 diabetes compared with metabolically healthy normal-weight adults.
- BMI and knee osteoarthritis: In males, the RRs were 1.39 (95% CI: 0.99–1.92), 3.41 (95% CI: 2.07–5.48), and 5.71 (95% CI: 3.12–9.95) when BMI was at the point of 25, 30, and 32.5 kg/m^2 compared with reference, respectively.[13] In females, the corresponding RRs were 1.72 (95% CI: 1.51–1.99), 3.51 (95% CI: 2.65–4.51), and 4.72 (95% CI: 3.25–6.91). Modest publication bias was found in the meta-analysis. However, sensitivity analysis showed a high stability for the result.
- BMI and endometrial cancer: Excess body weight (EBW defined as body mass index [BMI] ≥25 kg/m^2), obesity (BMI ≥30 kg/m^2) and overweight (25≤BMI<30 kg/m^2) were associated with an increased risk of endometrial cancer (relative risk [RR] for EBW=1.62, 95% confidence interval [CI], 1.39–1.89; for obesity RR=2.54, 95% CI, 2.11–3.06; for overweight RR=1.32, 95% CI, 1.16–1.50).[14]
- Obesity (but not overweight) and overall survival of colorectal cancer patients: The association between obesity and worse survival was strengthened when BMI was assessed before diagnosis (HR=1.30, 95 % CI, 1.17–1.44).[15]
- Obesity and risk of renal cell cancer: pooled RRs were 1.28(1.24–1.33) for pre-obesity and 1.77(1.68–1.87) for obesity, respectively. A nonlinear dose-response relationship was also found for risk of RCC with BMI (p = 0.000), and the risk increased by 4% for each 1 kg/m^2 increment in BMI.[16]

- In categorical meta-analysis of BMI, obesity was associated with significantly increased risks of contralateral breast (RR = 1.37, 95 % CI: 1.20–1.57), breast (RR = 1.40, 95 % CI: 1.24–1.58), endometrial (RR = 1.96, 95 % CI: 1.43–2.70), and colorectal (RR = 1.89, 95 % CI: 1.28–2.79) second primary cancers. For a BMI increase of 5 kg/m^2, dose-response meta-analyses resulted in significantly increased RRs of 1.12 (95 % CI: 1.06–1.20) and 1.14 (95 % CI: 1.07–1.21) for contralateral breast and breast second primary cancers, respectively. The summary RR for endometrial second primary cancers was 1.46 (95 % CI: 1.17–1.83) for a 5-unit increment.[17]
- In a meta-analysis of BMI and gastric cancer, obesity was associated with an increased risk of gastric cancer (odds ratio = 1.13, 95% CI 1.03–1.24) compared with normal weight, while overweight showed no association (OR = 1.04, 95% CI 0.96–1.12).18 Specifically, a stratified analysis showed there were associations between obesity and the increased risk of gastric cancer for males (OR = 1.27, 95% CI 1.09–1.48), non-Asians (OR = 1.14, 95% CI 1.02–1.28) and both cohort studies (odds ratio = 1.10, 95% CI 1.00–1.22) and case-control studies (odds ratio = 1.29, 95% CI 1.03–1.60). Both overweight (OR = 1.22, 95% CI 1.05–1.42) and obesity (OR = 1.61, 95% CI 1.15–2.24) were associated with the increased risk of gastric cancer.
- A total of 11 cohort studies were included in a meta-analysis of the association between obesity and risk of bladder cancer.[19] It showed that obesity was associated with an increased risk for bladder cancer in all subjects (RR=1.10, 95% CI=1.06–1.16). Among the nine studies that controlled for cigarette smoking, the pooled RR was 1.09 (95% CI 1.01–1.17). No significant publication bias was detected (p = 0.244 for Egger's regression asymmetry test).
- Cumulative data are compelling for a strong positive association between obesity and fatal prostate cancer incidence.[20]
- Obesity is linked with increased thyroid cancer risk in males and females, the strength of the association increasing with increasing BMI.[21] The combined RR of thyroid cancer was 1.18 (95% CI 1.11, 1.25) for excess body weight (overweight and obesity combined). Being overweight was associated with a significant increase in thyroid cancer risk among non-Asians, but not among Asians. Overweight, obesity and excess body weight were all associated with papillary thyroid carcinoma risk.
- Overweight and obesity are risk factors for lumbar radicular pain and sciatica in men and women, with a dose-response relationship. Both overweight (pooled odds ratio (OR) = 1.23, 95% CI, 1.14–1.33; n = 19,165) and obesity (OR = 1.40, 95% CI 1.27–1.55; n = 19,165) were associated with lumbar radicular pain.[22] The pooled OR for physician-diagnosed sciatica was 1.12 (95% CI, 1.04–1.20; n = 109,724) for overweight and 1.31 (95% CI, 1.07–1.62; n = 115,661) for obesity. Overweight (OR = 1.16, 95% CI, 1.09–1.24; n = 358,328) and obesity (OR = 1.38, 95% CI, 1.23–1.54; n = 358,328) were associated with increased risk of hospitalisation for sciatica, and overweight/obesity was associated with increased risk of surgery for lumbar disc herniation (OR = 1.89, 95% CI, 1.25–2.86; n = 73,982). Associations were similar for men and women and were independent of the design and quality of included studies. There was no evidence of publication bias.
- BMI and biliary tract disease: combined RRs (95% CIs) comparing the top with bottom categories were 1.40 (1.15–1.65) for biliary tract cancer and 2.75 (2.35–3.15) for non-cancer biliary tract disease (P for difference<0.001).[23] For non-cancer biliary tract disease, combined RRs (95% CIs) comparing the top with bottom categories were 3.21 (2.48–3.93) for women and 2.01 (1.66–2.37) for men (P for difference=0.04).
- Obesity was associated with an increasing risk of nuclear cataract (pooled RR, 1.12; 95% CI, 1.02–1.25), cortical cataract (pooled RR, 1.34; 95% CI, 1.07–1.66), and PSC cataract (pooled RR, 1.52; 95% CI, 1.31–1.77).[24] Overweight was only associated with an increasing risk of PSC cataract (pooled RR, 1.23; 95% CI, 1.09–1.40).
- Obese mothers (BMI >/=30) had greater odds of having an infant death (odds ratio 1.42; 95% CI, 1.24–1.63; P < .001; 11 studies); these odds were greatest for the most obese (BMI >35) (odds ratio 2.03; 95% CI 1.61–2.56; P < .001; 3 studies).[25] [residual confounding may explain findings so causality not assured].

Protective effects of obesity

- Obesity may be protective against incidence of non-smoking-related lung cancer.[26]
- This meta-analysis of prospective cohort studies suggests that obesity significantly decreases the risk of hip fracture in adults.[27]

Direct economic costs of obesity – Adults

- In an examination of 32 articles published between 1990 and June 2009, obesity was estimated to account for between 0.7% and 2.8% of a country's total healthcare expenditure.[28] Furthermore, obese individuals were found to have medical costs that were approximately 30% greater than their normal weight counterparts. Although variations in inclusion/exclusion criteria, reporting methods and included costs varied widely between the studies, a lack of examination of how and why the excess costs were being accrued appeared to be common in most studies. Accordingly, future studies must better explore how costs accrue among obese populations, to best facilitate health and social policy interventions.

Indirect costs of obesity – Adults

- There is a gradient between increasing BMI and direct healthcare costs and indirect costs due to reduced productivity and early premature mortality.[29] In the four (out of five) studies which presented separate estimates for direct and indirect costs of overweight and obesity, the indirect costs were higher, accounting for between 54% and 59% of the estimated total costs.
- As more people in a country become obese, a larger share of total annual national healthcare expenditure is spent on obesity and obesity-related health problems.[30] In addition to escalating healthcare costs, obesity increases indirect costs through decreases in workforce productivity. The empirical evidence has shown that obesity negatively impacts individuals, healthcare systems, employers, and the economy as a whole. This review provides a brief overview of selected economic consequences associated with excess weight.

Employer/productivity costs – Adults

- Obesity is associated with large employer costs from direct health care and insurance claims and indirect costs from lost productivity owing to work days lost because of illness and disability in the US.[31] Normal weight employees cost on average $3830 per year in covered medical, sick day, short-term disability and workers' compensation claims combined; morbidly obese employees cost more than twice that amount, or $8067, in 2011 dollars.
- Among 39,696 (BMI < 27), 14,281 (27 ≤ BMI < 30), and 18,801 (BMI ≥ 30) eligible employees in the US, per-employee adjusted total annual costs were $4258, $4873 and $6313, respectively.[32] Medical, pharmacy, sick leave, workers' compensation costs and days were higher for higher-BMI cohorts ($P < 0.01$). Employees with BMI ≥ 30 kg/m had the most short-term disability costs and days and least productivity ($P < 0.001$).
- Obese [RR=1.53] and, to a lesser extent, overweight (RR=1.16) individuals had an increased likelihood of exit from paid employment through disability pension, but were not at statistically significant increased risk for unemployment or early retirement.[33]
- Compared with non-obese workers, obese workers miss more work days due to illness, injury or disability. Costs of premature mortality vary substantially across countries. The results for presenteeism and workers' compensation were mixed.[34]

Health outcomes – Children

- A recent review (2013) indicated that the major medical co-morbidities associated with childhood obesity in the current literature are metabolic risk factors, asthma and dental health issues. Major psychological co-morbidities include internalising and externalising disorders, attention-deficit hyperactivity disorder and sleep problems.[35]
- Obesity has been identified as a primary risk factor for pre-hypertension and hypertension in childhood.[36] As many as 13% of youth with obesity have elevated systolic blood pressure and 9% have elevated diastolic blood pressure. The presence of obesity increases the likelihood that a hypertensive child will become a hypertensive adult, thus compounding the risk for CVD these children already face.

- Overweight and obese youths are more likely to be victims of bullying.[37] The results were not moderated by gender: overweight and obese boys and girls were equally likely to be victimised. Results remained significant after adjustment for publication bias.
- Studies reported a worsening of risk parameters for cardiovascular disease in overweight and obese children.[38] Compared with normal weight children, systolic blood pressure was higher by 4.54 mmHg (99% CI, 2.44–6.64; n=12,169, eight studies) in overweight children, and by 7.49 mmHg (CI, 3.36–11.62; n=8074, 15 studies) in obese children. Similar associations were found between groups in diastolic and 24-hour ambulatory systolic blood pressure. Obesity adversely affected concentrations of all blood lipids; total cholesterol and triglycerides were 0.15 mmol/L (CI, 0.04–0.25, n=5072) and 0.26 mmol/L (CI, 0.13 to 0.39, n=5138) higher in obese children, respectively. Fasting insulin and insulin resistance were significantly higher in obese participants but not in overweight participants. Obese children had a significant increase in left ventricular mass of 19.12 g (CI, 12.66–25.59, n=223), compared with normal weight children.
- Relationships were found between lack of physical exercise, heightened sedentary behaviour, poor diet quality, obese or overweight and depression in adolescence.[39] However, the finding that obesogenic risk factors are associated with poor adolescent mental health should be interpreted with caution as data typically come from non-representative samples with less than optimal study design and methodology.
- The available literature on the relationship between obesity and clinical outcomes is limited by subject heterogeneity, variations in criteria for defining obesity and outcomes examined.[40] Childhood obesity may be a risk factor for higher mortality in hospitalised children with critical illness, oncologic diagnoses or transplants. Further examination of the relationship between obesity and clinical outcomes in this subgroup of hospitalised children is needed.
- There is relatively strong evidence showing a relationship between obesity and otitis media in children.[41]

Meeting HEAL Objective: Reduce consumption of energy dense nutrient poor food

Introduction

As there is a separate HEAL objective relating to reducing the consumption of sugar-sweetened drinks; the search for this objective was restricted to energy dense nutrient poor (EDNP) food and did not include drinks. The search strategy included the following terms: 'energy dense nutrient poor AND food', 'processed AND food', 'packaged AND food', 'non-core AND food', 'takeaway AND food', 'fast food' and 'fried food'. It is acknowledged that there may be other foods/terms that could be used to identify information on associations between ENDP foods and health outcomes. Additionally the large literature that relates to the specific negative nutrients contained within EDNP foods (particularly salt, sugar and saturated fat) were outside the scope of this review.

Description of the evidence

No systematic reviews or meta-analyses of population studies were identified. The evidence on associations of EDNP foods with major health outcomes is from single population studies (cohort and case control studies). Details of the included articles reporting associations with major health outcomes and the consumption of EDNP food are contained in Appendix 2.

Summary of the findings

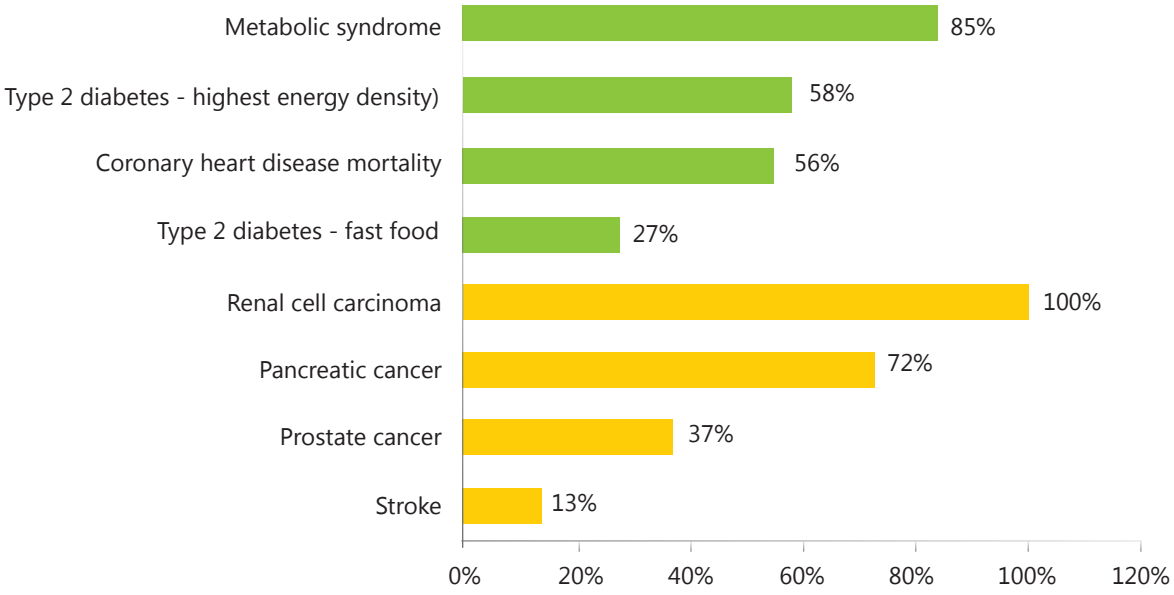
There is evidence of an association between consumption of ENDP food and an increased risk of:

- Metabolic syndrome (85% for highest quintile of fast food consumption)[42]
- Type 2 diabetes (27% for ≥2 serves per week of fast food[43]; 58% for diet with the highest energy density)[44]
- Renal cell carcinoma (100% for highest quintile of consumption of dietary fat)[45]

- Coronary heart disease mortality (56% for ≥2 serves per week of fast food)[43]
- Prostate cancer (≥1 serve per week; 37% OR for French fries; 30% OR for fried chicken; 35% OR for doughnuts)[46]
- Stroke (13% for highest versus lowest distribution of fast food restaurants)[47]
- Pancreatic cancer (72% for diet with highest energy density).[48]

A comparison of effect sizes of the prospective associations between EDNP food and major health outcomes is presented in Figure 1.1.

Figure 1.1 – Comparisons of increased risk of various health outcomes related to [d]consumption of energy dense nutrient poor food



d: As indicated in the dot points in this section, increased risk of the various morbidities is related to different levels of consumption and different types of EDNP food.

Meeting HEAL Objective: Increase consumption of fruit and vegetables

Introduction

Search terms included 'fruit' OR 'vegetables' and 'mortality', 'morbidity' and 'health' in addition to a variety of specific health outcomes as listed in the general methods.

Description of the evidence

A total of 24 articles were identified in the systematic searches consisting of seven studies undertaking systematic reviews and meta-analysis with prospective cohorts reporting outcomes for:

- All-cause mortality
- CVD mortality
- Type 2 diabetes
- Gastric cancer
- Colorectal cancer
- Breast cancer.

A further five studies undertaking systematic reviews and meta-analysis with a combination of cohort and case-control studies were identified reporting outcomes on:

- Oral cancer
- Lung cancer
- Oesophageal cancer
- Endometrial cancer
- Non-Hodgkin's lymphoma.

The non-systematic searches to identify other relevant literature identified two papers reporting on the:

- Health care costs associated with an increase in fruit and vegetable consumption
- Economic gains in the agricultural sector.

Eleven health outcomes were identified that were associated with fruit and vegetables consumption. Details of the articles used to provide information on the level of association is provided in Table A2.2, Appendix 2. Two articles modelling the impact on health care costs and the agriculture sectors were also located. Publications reporting an association with specific fruits and vegetables (or vegetable groups) were excluded as their examination was beyond the scope of this review.

The majority of studies reported on the association with a health outcome for fruit and vegetables separately. Possible limitations reported in the reviews include high heterogeneity in the populations, designs and analysis of the included studies. The reviews also identified differences between the studies in the way in which fruit and vegetable intake was assessed (e.g. self-report or interview) and reported (serves per day, week or month) and variation in the definition of a serve (portion) of fruit or vegetables. Most of the studies undertook statistical analysis to identify and address the heterogeneity.

All studies undertaking meta-analyses indicated they used the relative risk score adjusted for the greatest number of confounders in the original studies. Only two systematic reviews assessed the quality of the included studies using a quality score, however all investigated whether specific study characteristic such as duration of follow-up, number of cases, and adjustment for confounders, which are indicators of study quality to determine if they influenced the results in sub-group analysis.

Summary of findings

There is evidence of an association with increased fruit and/or vegetables (F&V) consumption and reduced risk for a number of major health outcomes, including:

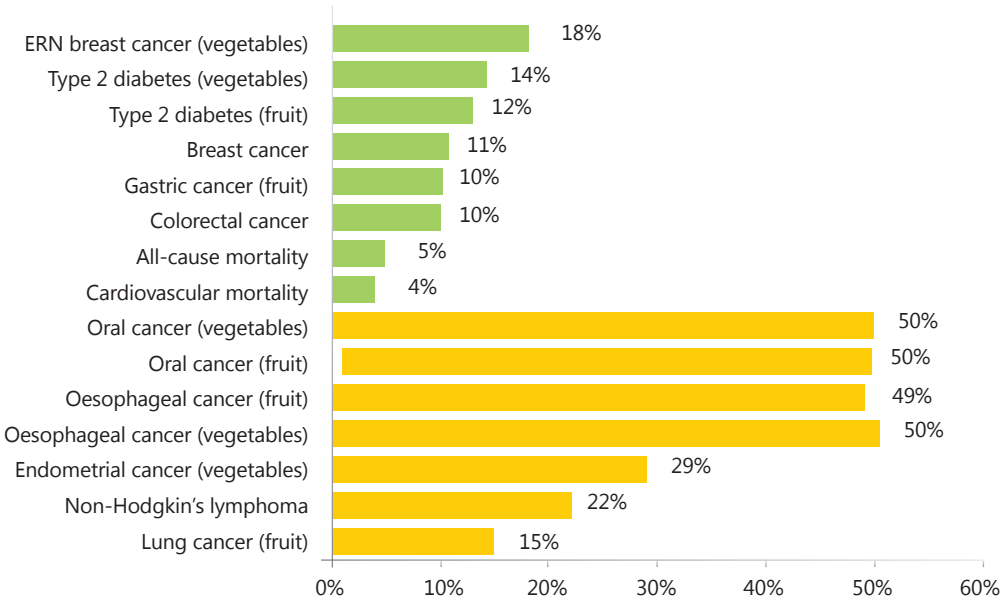
- All-cause mortality (5% for 5-6 serves/day of F&V combined)[49]
- Cardiovascular mortality (4% for 5-6 serves/day of F&V combined)[49]
- Type 2 diabetes (12% for ≥3 serves/day of fruit[50] and 14% for high vegetable consumption)[51]
- Gastric cancer (10% for high fruit and 4% for high vegetable consumption)[52]
- Colorectal cancer (10% for high fruit and 9% for high vegetable consumption)[53]
- Breast cancer (11% for high F&V consumption)[54]
- Oestrogen receptor-negative[e] breast cancer (6% for high fruit, 18% for high vegetable consumption)[55]
- Oral cancer (49% for each additional serve of fruit/day and 50% for each additional serve of vegetable/day)[56]
- Lung cancer[f] (15% for high fruit consumption and 8% for each additional serve of fruit/day)[57]
- Oesophageal cancer[g] (49% for high fruit and 50% for high vegetable consumption)[58]
- Endometrial cancer (10% for high fruit and 29% for high vegetable consumption)[59]
- Non-Hodgkin's lymphoma (22% for high F%V consumption).[60]

There is some evidence of economic benefits associated with increased fruit and/or vegetables consumption, including:

• Decreased health care costs up to 70 years of age[61,] and economic gains in the agricultural sector.[62]

There was no evidence for any other direct or indirect benefits (including wellbeing indicators) associated with increased fruit and/or vegetables consumption.

Figure 1.2 – Comparisons of risk reduction of various health outcomes associated with high fruit and/or vegetables consumption



Meeting HEAL Objective: Increase intake of water in preference to sugar-sweetened drinks

Introduction

The HEAL objective is to 'Increase intake of water in preference to sugar-sweetened drinks'. However, no articles were identified from an initial search for studies specifically examining the behaviours of 'replacing sugar-sweetened drinks with water' and 'consumption of water' and the impact on health or other outcomes. Subsequent searches concentrated on the impact of consumption of sugar-sweetened beverages only.

Description of the evidence

Eight health outcomes were found to be associated with consumption of sugar-sweetened drinks. Detail of the articles used to provide information on the level of association is contained in Table A2.3, Appendix 2. A total of nine articles were identified in the systematic searches consisting of three undertaking systematic reviews and meta-analysis with prospective cohorts reporting outcomes for:

• Type 2 diabetes

• Coronary heart disease

• Pancreatic cancer.

e Oestrogen receptor-negative breast cancer; accounts for approximately 20% of all breast cancers

f 8 of the 10 studies included in the analysis controlled for confounding by smoking (7) or limited analysis to never-smoke (1)

g Oesophageal squamous cell carcinoma only

A further six single cohort studies were identified reporting outcomes on:

- Gout (men) and gout (women)
- Dental erosion in children
- Hypertension
- Kidney stones
- Stroke.

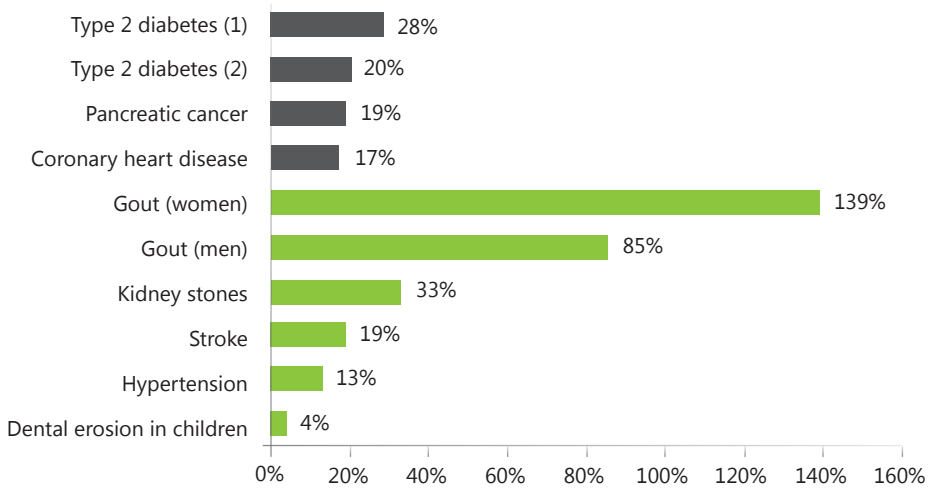
Summary of findings

There is evidence of an association between intake of sugar-sweetened drinks and increased risk of:

- Type 2 diabetes (20% increased risk for each 330mL serve/day)[63]
- Coronary heart disease (17% for high intake, compared to low)[64]
- Pancreatic cancer (19% for high intake, compared to low)[65]
- Gout (45% in males and 74% in females for 1 serve/day, compared to <1 serve/month)[66, 67]
- Dental erosion in children (4% for any consumption, compared to none)[68]
- Hypertension (13% ≥1 serve/day, compared to <1 serve/month)[69]
- Kidney stones (33% for ≥1 serve/day, compared to <1 serve/week)[70]
- Stroke (11% ≥2 serves/day, compared to <0.5 serve/day)[71]

Table A2.3 in Appendix 2 contains further details of the details of the categorisation for high and low intake.

Figure 1.3: Increased risks of major health outcomes associated with high consumption of sugar-sweetened beverages



(1) Sugar-sweetened fruit juice only
(2) Sugar-sweetened soft drinks only

Meeting HEAL Objective: Increase incidental, moderate and vigorous physical activity

Introduction

All relevant search terms were used. Many of the articles did not specify the type of physical activity linked to the health outcome.

Description of the evidence

A total of 27 reviews consisting of 15 systematic reviews and/or meta-analytical reviews of prospective cohorts was used to present information on level of association between:

- All-cause mortality
- Cardiovascular mortality
- Cognitive decline
- Dementia
- Stroke
- Asthma
- Metabolic syndrome
- Hypertension
- Type 2 diabetes
- Coronary heart disease
- Hip fractures
- Pancreatic cancer
- Lung cancer
- Breast cancer.

A further 10 articles undertaking a systematic review and/or meta-analytical with a combination of cohort and case-control studies were identified reporting outcomes on:

- Pre-eclampsia
- Renal cancer
- Gastroesophageal cancer
- Colon cancer
- Colorectal cancer
- Bladder cancer
- Endometrial cancer
- Breast cancer
- Prostate cancer
- Gestational diabetes.

In addition, two systematic reviews and/or meta-analytical reviews were identified that reported pooled results of case-control studies on the association between physical activity and:

- Breast cancer
- Ovarian cancer.

Finally, one meta-analytical review was identified that reported pooled results of cross-sectional studies on physical activity and erectile dysfunction.

The non-systematic searches to identify other relevant literature identified two papers reporting on the:

- Association of physical activity and depression (systematic review)
- Association of physical activity and anxiety (systematic review)

- Healthcare costs associated with physical inactivity in Canada (analytical review)
- Healthcare costs associated with physical inactivity in Australia (report)
- Influence of physical activity on long-term sickness absence (prospective cohort study)
- Influence of physical activity on global well-being/life satisfaction (two longitudinal studies);
- Association of physical activity and health-related quality of life (systematic review)
- Influence of physical activity on sleep duration and quality (prospective cohort study)
- Effects of exercise on global self-esteem (meta-analytical review)
- Effects of exercise interventions on body image (meta-analytical review)
- Various health outcomes of physical activity among children and adolescents (two systematic reviews).

Most meta-analytical reviews tested for the heterogeneity between effect sizes, particularly when reporting pooled results from cohort and case-control studies. Approximately half of the included studies reported on health outcomes of non-specific physical activity. The other half of the studies reported exclusively or separately on walking (four studies), leisure-time physical activity (five studies), recreational physical activity (five studies), occupational physical activity (seven studies), transport-related physical activity (one study), activities of daily living (one study), moderate-intensity physical activity (five studies), vigorous-intensity physical activity (four studies), moderate to vigorous-intensity physical activity (two studies) and light-intensity physical activity (one study).

Several reviews have shown heterogeneous effects of different domains and intensity-levels of physical activity on health outcomes. For example, leisure-time and recreational physical activity showed stronger association with all-cause mortality,[72] coronary heart disease,[73] hypertension[74] and bladder cancer[75] when compared to occupational physical activity. Conversely, occupational physical activity showed stronger association with prostate cancer[76] and pancreatic cancer[77] when compared to recreational physical activity. Vigorous-intensity physical activity showed somewhat stronger association with all-cause mortality when compared to moderate-intensity physical activity.[72]

These data show that pooled estimates for health outcomes of non-specific physical activity may be biased, as they combine potentially heterogeneous effect sizes. Additional sources of bias may include the use of different methods of physical activity assessment and inconsistencies in categorizing physical activity levels across the studies. Furthermore, approximately half of the reviews about health outcomes of physical activity did not assess the quality of the included studies. Inclusion of potentially low-quality studies may have also reduced the validity of pooled estimates.

The estimate of total direct healthcare costs that can potentially be saved by increasing population physical activity levels are based on Canadian data.[78] The Australian estimate is based on seven medical outcomes only.[79] It could be assumed that this estimate would be even higher, if it took account of all of the above-mentioned health outcomes of physical activity.

Summary of findings

There was evidence of a strong association for 24 health conditions and moderate evidence of prospective association for a number of other health outcomes with physical activity. Details of the articles used to provide information on the level of association is in Table A2.4, Appendix 2.

There is evidence of an association between physical activity and the following health conditions, including a lower risk (LR) or lower odds (LO) of:

- All-cause mortality (29% for most active group, compared to least)[80]
- Cardiovascular mortality (30% LR)[80]
- Stroke (21% LR)[81]
- Coronary heart disease (up to 20%)[82]
- Hypertension (19% LR)[74]
- Metabolic syndrome (20% LR)[83]
- Type 2 diabetes (31% LR)[84]
- Asthma (13% LO)[85]

- Hip fractures (38% [women], 45% [men] LR)[86]
- Dementia (14% LR)[87]
- Cognitive decline (35% LR)[87]
- Lung cancer (23% LR)[88]
- Pancreatic cancer (up to 28% LR)[77]
- Breast cancer (19% LR)[89]
- Proximal colon cancer (27% LR)[90]
- Distal colon cancer (26% LR)[90]
- Gastroesophageal cancer (18% LR)[91]
- Renal cancer (12% LR)[92]
- Bladder cancer (up to 20% LR)[75]
- Prostate cancer (up to 19% LR)[76]
- Endometrial cancer (18% LR)[93]
- Pre-eclampsia (up to 36% LR)[94]
- Gestational diabetes (55% LO)[95]
- Ovarian cancer (24% LR).[96]

Additionally, there is moderately strong evidence about the prospective associations of physical activity and reduced risk of depressive[97, 98] and anxiety disorders[98] and cross-sectional evidence about an inverse association of physical activity and erectile dysfunction (47% LO).[99]

This review identified the following evidence about economic benefits associated with physical activity:

- By increasing population physical activity levels total direct healthcare costs can potentially be reduced by 2.5%.[78]
- An older estimate indicate that the annual direct healthcare costs that could be saved by increasing population physical activity level in Australia are around $377 million a year.[100]
- Taking into account possible reductions of direct healthcare costs of seven medical conditions (coronary heart disease, stroke, type 2 diabetes, breast cancer, colon cancer, depression and falls), it was more recently estimated that, by increasing population physical activity levels, Australian healthcare system could save up to $1.5 billion a year[79].
- Australian estimates indicate that a 10% increase in prevalence of sufficient physical activity would result in lifetime potential cost savings of $71 million in domestic-based production, $12 million in workforce production, and $79 million in leisure-based production, which is in total $162 million.[101]
- Participation in leisure-time physical activities reduces odds of long-term (>14 days) sick leave by up to 29%.[102]

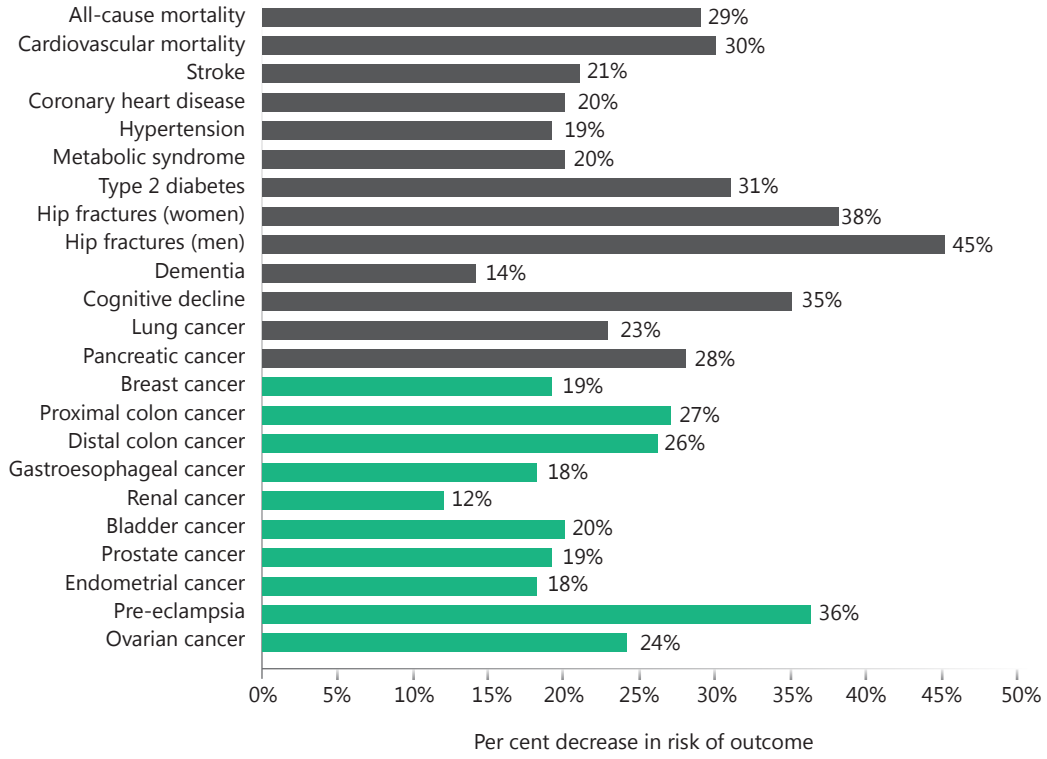
Some evidence was also identified for other direct or indirect benefits associated with physical activity, including:

- Improved global well-being/life satisfaction[103, 104]
- Improved health-related quality of life[105]
- Improved sleep quality[106]
- Improved global self-esteem[107]
- Improved body image.[108]

Two recent reviews provide a synthesis of evidence on health outcomes of physical activity among children and adolescents.[109, 110] In summary, they found high-level evidence for the positive association between physical activity and the following health outcomes:

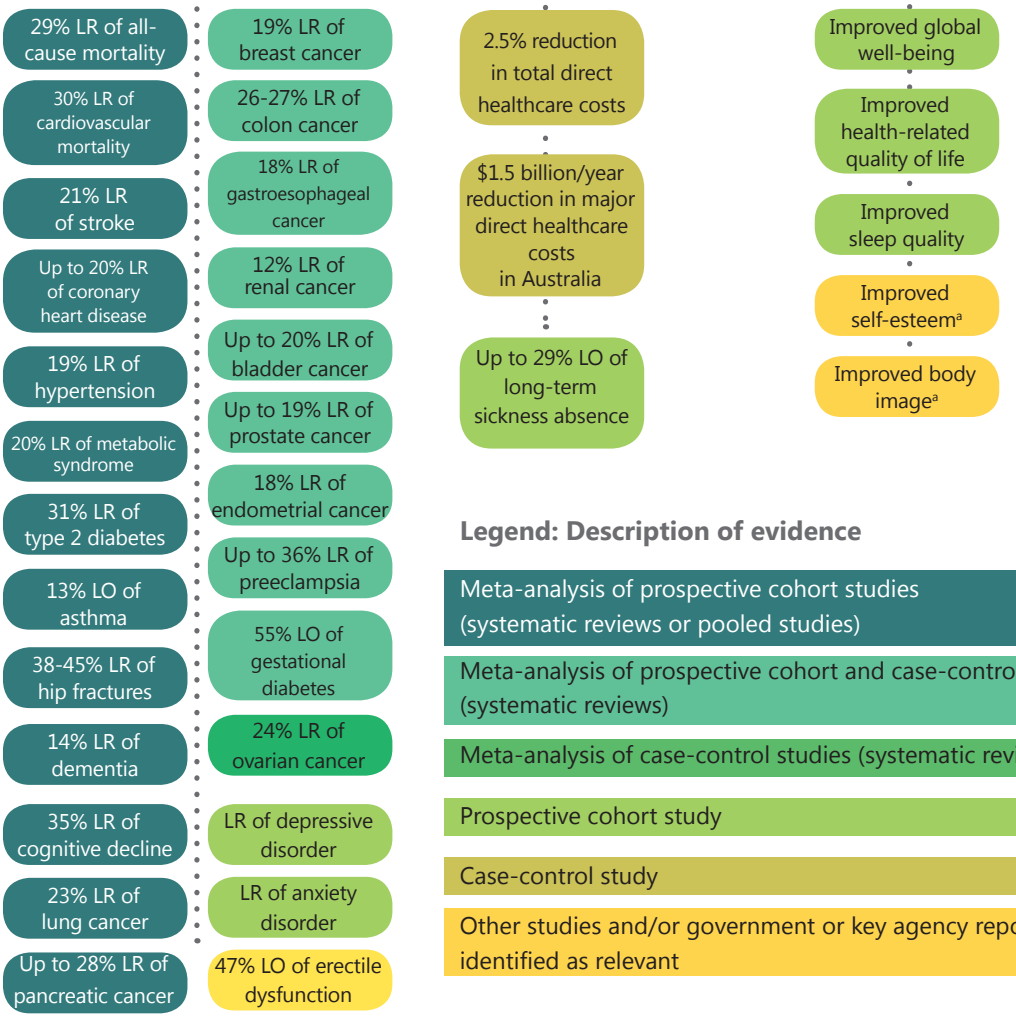
- Cardiometabolic health
- Musculoskeletal health
- Mental health
- Cardiorespiratory fitness.

Figure 1.4a: A comparison of risk reductions of health outcomes by participation in physical activity among adults



An overview of the major health, economic and other benefits of physical activity and the nature of evidence supporting these associations is presented in Figure 1.4b on the next page.

Figure 1.4B: Outcomes associated with physical activity



Legend: Description of evidence

- Meta-analysis of prospective cohort studies (systematic reviews or pooled studies)
- Meta-analysis of prospective cohort and case-control studies (systematic reviews)
- Meta-analysis of case-control studies (systematic reviews)
- Prospective cohort study
- Case-control study
- Other studies and/or government or key agency reports identified as relevant

NOTE:

a: The evidence based on review and meta-analysis of exercise intervention trials

Meeting HEAL Objective: Reduce time in sedentary behaviours

Introduction

All relevant search terms were used. Relevant exposures and therefore search terms included TV viewing, sitting time, and occupational sitting. Nearly half of the articles identified did not differentiate between the various types of sedentary behaviour.

Description of the evidence

A total of seven reviews were identified in the systematic searches on health outcomes of sedentary behaviour consisting of four systematic reviews and/or meta-analysis of prospective cohorts reporting on:

- All-cause mortality
- Cardiovascular mortality
- Cardiovascular disease
- Type 2 diabetes.

Three systematic reviews and/or meta-analysis with a combination of cohort and case-control studies reporting on:

- Type 2 diabetes
- Colon cancer
- Endometrial cancer
- Lung cancer.

One systematic and meta-analytical review reported pooled results of cross-sectional studies on metabolic syndrome.

The non-systematic searches to identify other relevant literature identified a recently published article[111] that provided an overview of findings from 27 systematic reviews about health and non-health outcomes of sedentary behaviour including:

- Breast cancer
- Ovarian cancer
- Prostate cancer
- Depressive symptoms
- High blood cholesterol
- Hypertension
- Physical fitness
- Academic achievement
- Cognitive performance
- Anti-social/pro-social behaviour
- Eating habits.

All meta-analytical reviews included in the current report tested for the heterogeneity of effect sizes. For approximately half of the health outcomes, meta-analyses were conducted on all available sedentary behaviour studies, regardless of the type/domain of sedentary behaviour. The other half of the meta-analyses were conducted specifically for TV viewing/screen time (five meta-analyses), total sitting time (four meta-analyses), and occupational sitting (two meta-analyses).

It seems that specific types of sedentary behaviour may have distinct effects on health outcomes. For example, screen time showed stronger association with cardiovascular disease, colon cancer and endometrial cancer, when compared to total sitting time.[112] Hence, combining results for different types of exposure (e.g. TV viewing, total sitting time, occupational sitting), i.e. analysing health outcomes of non-specific sedentary behaviour, may have increased heterogeneity of effect sizes and induced bias in pooled estimates. Additional sources of bias may include the use of different methods of sedentary behaviour assessment and inconsistencies in defining sedentary behaviour categories across the included studies.

Furthermore, two reviews did not assess the quality of the included studies. Inclusion of potentially

low-quality studies may have also reduced the validity of pooled estimates. In a recent review of methodological quality of 54 sedentary behaviour studies, Pedisic[112] identified the following key limitations:

- Most studies used measures of sedentary behaviour that had not been previously validated.
- In the large majority of studies, the adjustments for physical activity were not thorough enough to support the conclusion about the independent association between sedentary behaviour and health outcomes.
- Almost none of the studies adjusted their analyses for sleep duration, which may have also caused residual confounding.[112]

These limitations may have led to biased estimates and therefore should be taken into account when interpreting the findings of the present report.

Summary of findings

There is evidence of an association between reducing time spent in sedentary behaviours and an increased risk of:

- All-cause mortality (33% for highest sedentary behaviour, compared to lowest[113]; 12% per two hours/day reduced TV viewing time[114]; 2% LHR per two hours/day reduced total sitting time)[115]
- Cardiovascular mortality (47% for highest sedentary behaviour, compared to lowest)[113]
- Cardiovascular disease (13% per two hours/day reduced TV viewing time;[114] 5% per two hours/day reduced total sitting time)[111]
- Type 2 diabetes (53% for highest sedentary behaviour, compared to lowest)[113]
- Metabolic syndrome (42% for highest sedentary behaviour, compared to lowest)[116]
- Colon cancer (23% for highest sedentary behaviour, compared to lowest[117]; 35% for highest TV viewing time, compared to lowest[118]; 19% for highest occupational sitting time, compared to lowest; 19% for highest total sitting time, compared to lowest)
- Endometrial cancer (26% for highest sedentary behaviour, compared to lowest; 40% for highest TV viewing time, compared to lowest; 24% for highest total sitting time, compared to lowest)[118]
- Lung cancer (17% for highest sedentary behaviour, compared to lowest).[118]

Additionally, a recent article gave an overview of findings from 27 systematic reviews about health outcomes of sedentary behaviour.[119] In addition to the above-mentioned potential benefits of reduced time spent in sedentary behaviours, the article reported some evidence for lower risk of the following outcomes among adults:

- Breast cancer (reduced TV viewing, total sitting time or sitting outside work)
- Ovarian cancer (reduced TV viewing, total sitting time or sitting outside work)
- Prostate cancer (reduced TV viewing, total sitting time or sitting outside work)
- Depressive symptoms (reduced TV viewing time or other sedentary behaviours)
- Unhealthy eating habits (reduced screen time).

This review also presented some evidence for potential benefits of reducing time spent in sedentary behaviour among children and adolescents, in terms of lower risk of the following health and non-health outcomes:

- High blood cholesterol
- Hypertension
- Depressive symptoms (reduced screen time)
- Low physical fitness (reduced TV viewing time/computer use)
- Poor academic achievement (reduced screen time)
- Low cognitive performance (reduced TV viewing time)
- Anti-social behaviour (reduced TV viewing time)
- Unhealthy eating habits (reduced screen time).

A comparison of effect sizes of the prospective associations between sedentary behaviour and major health outcomes identified in the current review is presented in Figures 1.5a and 1.5b.

Figure 1.5a: Comparison of potential risk reduction of health outcomes by reducing non-specific sedentary behaviour/total sitting time

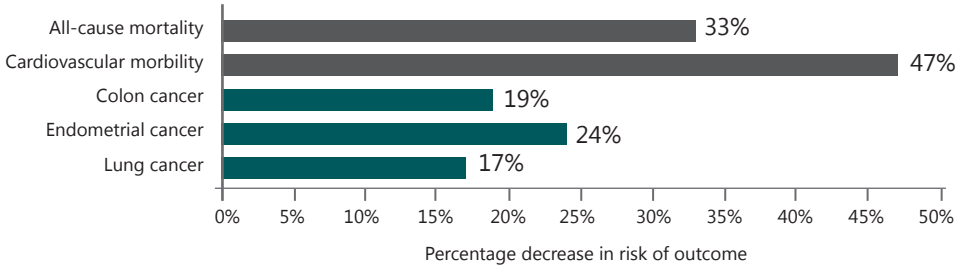
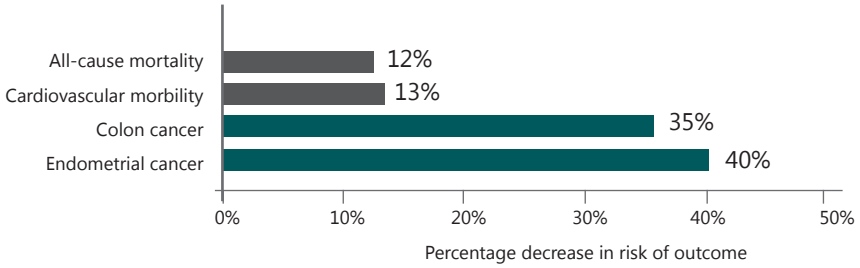


Figure 1.5b – Comparisons of potential risk reduction of health outcomes by reducing TV viewing/screen time



Meeting HEAL Objective: Increase community awareness of healthy eating and physical activity as protective factors against chronic disease

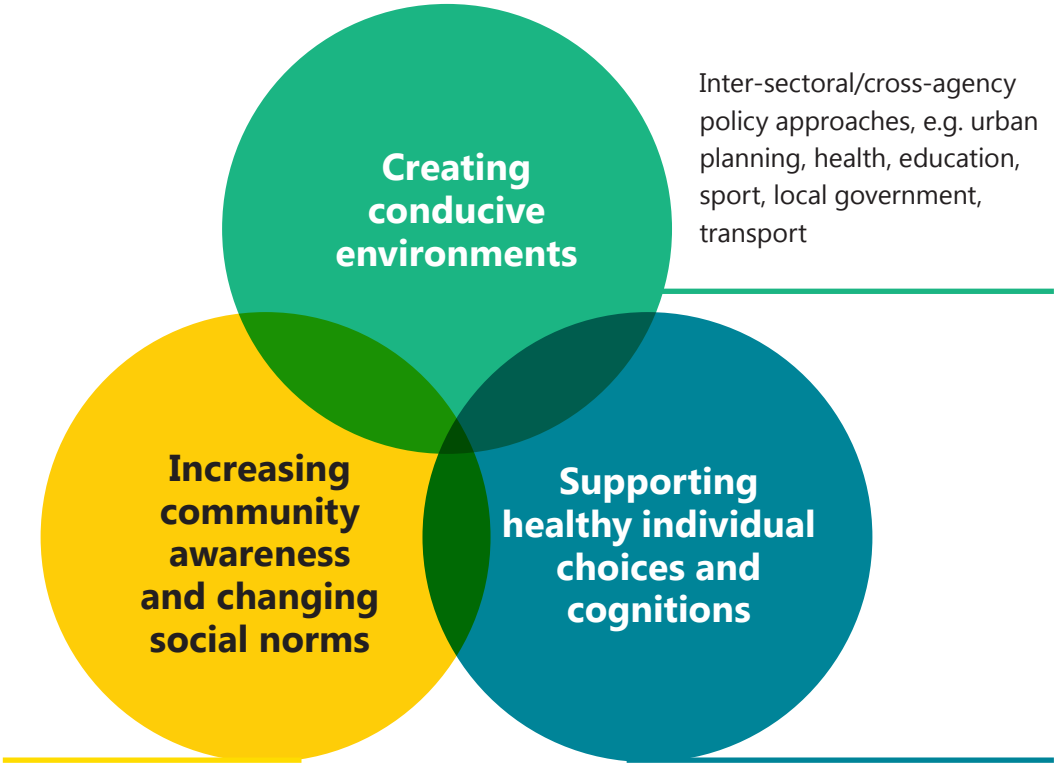
Introduction

The review found a paucity of evidence linking community awareness directly to outcomes beyond health behaviours and their antecedents. No relevant systematic reviews have been published to date in the peer-reviewed literature ('absence of evidence' rather than 'evidence of absence'). Conducting such a review across the domains of physical activity, healthy eating and obesity, while extensive, would be a very worthwhile undertaking. The detailed additional analysis required for this fell outside the scope of the current review. Nonetheless relevant population data on community awareness in NSW are presented in this section.

Effective approaches to the primary prevention of non-communicable diseases require a comprehensive range of public health strategies and interventions. An integrated program will engage with the health sector, work in partnerships outside the health sector, and develop healthy and supportive environments and policies to facilitate healthy choices by individuals and communities. Such an integrated public health framework is characterised by three major areas of public health planning and action, shown in Figure 1.6. Work in all three areas is required for a comprehensive prevention approach, and the role of communications and public education, including mass media campaigns, is a catalyst for action and contributor overall to a public health strategy.

This approach is not new, but it is a development from traditional 'integrated health promotion' program approaches that date back two decades.[120] A comprehensive approach is optimal, as shown in Figure 1.6 (next page) and described elsewhere.[121] Mass media campaigns are related to the left hand side of the diagram, changing community awareness and making health-enhancing behaviours more strongly socially normative.

Figure 1.6: The place of community awareness strategies in a comprehensive approach to non-communicable diseases prevention



Awareness in NSW of prevention-related information

To discuss the issue further, 'community awareness' may be defined as the proportion of the population aware of specific information or themes regarding the relationship between healthy diet and physical activity and chronic disease. This could in turn be conceptualised in diverse ways. One method is to focus on the proportion of the population aware that healthy eating and physical activity are related to chronic disease, CVD, diabetes or other specific outcomes. This is explored below using data from the Cancer Institute NSW as well as data from the Measure up Campaign baseline survey.

Community awareness of healthy lifestyle NSW 2006–2009

In 2006, the Cancer Institute conducted the lifestyle cancers baseline survey to measure knowledge, attitudes and behaviour in respect to a range of lifestyle indicators.[122] This survey was repeated annually until 2009 to enable any changes over time to be monitored and to inform future policy and practice in NSW. Participants were asked how many serves (fruit and vegetables) they understood was recommended for a well-balanced diet.

In 2009, as in previous years, people tended to slightly overestimate the amount of fruit required for good health, and to underestimate the amount of vegetables. In terms of fruit, the average number of serves believed to be recommended has increased significantly since the 2008 survey, from 2.6 to 2.8 serves per day ($p<0.05$). Encouragingly, the significant improvement in perceived requirements for vegetables that occurred in 2008 was maintained in 2009. Participants believed an average of 3.4 serves of vegetables were required each day, up from 2.8 serves in 2006.

About one-quarter (24%) of participants correctly stated that it is recommended an adult eat five serves of vegetables each day, while 38% knew that it is recommended an adult eat two serves of fruit. Only 10%

were able to correctly state both recommendations. Overall, the proportions who correctly stated two serves of fruit, five serves of vegetables or both have all increased since baseline (from 31%, 12%, and 4% respectively; all $p<0.01$).

However, there has been a decline since 2008 in both the proportion correctly stating two serves of fruit (down from 43%; $p<0.05$) and the proportion correctly stating five serves of vegetables (down from 29%; $p<0.05$). The decrease may be attributable to the fact that the 'Go for 2 & 5' campaign had not been aired since 2007. Participants were asked about how much exercise is needed to maintain good health: both the frequency of moderate exercise, and the duration of moderate exercise on each day. Close to three-tenths (29.6%) of participants correctly identified the NSW Health physical activity recommendation of 30 minutes, five to seven times per week. This level has not increased significantly since the baseline in 2006.

Another example below uses data re-analysed from the 'Measure Up' phase 1 mass media campaign, and confined to NSW residents. This is shown in the table below; although population-based surveys generated these data, they are not included in routine NSW Population health surveys. For example from Table A below, a quarter of adults knew the recommended serves of vegetables, over a third knew the amount of fruit, but very few recognised that waist circumference was associated with chronic disease.

Table A: Data from the Measure Up Campaign baseline, indicators of community understanding of prevention

Knowledge	
Chronic diseases caused by an unhealthy lifestyle (% mentioning at least one of type 2 diabetes, heart disease, cancer)	90.9%
Recommended serves of vegetables/day (5)	26.6%
Recommended serves of fruit/day (2)	38.5%
Recommended minutes of physical activity/day (30)	52.3%
Waist measurement associated with increased risk of chronic disease for men (% correct men only, n=994)	0.2%
Waist measurement associated with increased risk of chronic disease for women (% correct women only, n=1018)	6.4%
Proportion of Australian adults who are overweight (% 1 in 2)	42.9%

Data: Measure Up NSW, n=1006; In [123]

Summary of findings

The review found a paucity of evidence linking community awareness directly to outcomes beyond health behaviours and their antecedents. No relevant systematic reviews have been published to date in the peer-reviewed literature ('absence of evidence' rather than 'evidence of absence'). Conducting such a review across the domains of physical activity, healthy eating and obesity, while extensive, would be worthwhile; the detailed additional analysis required for this fell outside the scope of the current review.

SECTION 2 – REVIEW QUESTION 2

Estimated lives saved and diseases prevented annually through a fully implemented HEAL strategy – selected case studies

Introduction

The following section provides estimates the number of cases or deaths that could be avoided if the HEAL targets and objectives were met. One outcome was selected for each of the HEAL targets and objectives (excluding community awareness) addressed in review question 1. The outcomes were selected based on the burden of disease in Australia, as identified by Begg et al.[125] Accordingly, five modelled studies of estimated diseases prevented or lives saved annually were prepared, as described in the following sections.

Case study 1: Physical activity and colorectal cancer

In 2012, 44% of NSW adults were estimated to be inadequately physically active, according to Australian guidelines in place at the time.[126] Based on this, a 15% reduction in the proportion of adults that are physically inactive in NSW (i.e. to 37.4%) would be estimated to prevent 37 cases of colorectal cancer per year (Table 2.1).

Table 2.1: The estimated number of avoided cases of colorectal cancer (CRC) in NSW per year that would result from a 15% reduction in inadequate physical activity

NSW prevalence of inadequate physical activity (2012)[127]	RR of CRC[128]	PAR	NSW incidence of CRC (2009)[129]	Number attributable to inadequate physical activity	Avoided cases per year
44%	1.14	5.3%	4744	249	37

RR = relative risk; relative to adequate physical activity

Case study 2: Fruit and vegetables consumption and coronary heart disease

In 2012, 47% of NSW adults were estimated to not be eating a sufficient number of serves of fruits per day, while 90% were estimated to not be eating a sufficient number of serves of vegetables per day, according to Australian guidelines.[130] Based on this, a 25% reduction in insufficient fruit consumption (i.e. to 35%) would be estimated to prevent 422 cases of coronary heart disease (Table 2.2, see next page).

The corresponding figure for a 25% reduction in insufficient vegetable consumption (i.e. to 68%) would be 1003 cases of coronary heart disease. It should be noted that, as a result of the complex relationships between fruit and vegetables consumption and coronary heart disease, the estimated number of avoided events are not cumulative.

Table 2.2: The estimated number of avoided cases of coronary heart disease (CHD) in NSW per year that would result from a 25% reduction in insufficient fruit and vegetables consumption

	NSW prevalence of insufficient consumption (2012)a,[127]	RR of CHD[131]	PAR	Estimated NSW incidence of CHD (2009)[132]	Number attributable to insufficient consumption	Avoided cases per year
Fruit	47%	1.15	6.1%	27,851	1687	422
Vegetables	90%	1.19	14.4%	27,851	4011	1003

RR = relative risk; relative to sufficient consumption of fruit or vegetables
a Insufficient consumption of fruit = less than two serves per day; insufficient consumption of vegetables = less than five serves per day

Case study 3: Sugar-sweetened beverages and type 2 diabetes

In 2011–12, 29% of Australian adults were estimated to consume any soft drink. Based on this, a 25% reduction (i.e. to 22%) would be estimated to prevent 510 cases of type 2 diabetes per year (Table 2.3).

Table 2.3: The estimated number of avoided cases of type 2 diabetes in NSW per year that would result from a 25% reduction in consumption of sugar-sweetened beverages (SSB)

Any consumption of SSB (2011-12)[133]	RR of type 2 diabetes[63]	PAR	Estimated NSW incidence of type 2 diabetes[i]	Number attributable to sugar-sweetened beverages	Avoided cases per year
29%	1.20	4.9%	42,080	2041	510

RR = relative risk; relative to no consumption of sugar-sweetened beverages

Case study 4: Sitting time and all-cause mortality

In a recent study[h], 12% of Australian adults were estimated to sit for 10 or more hours per day. Based on this, a 25% reduction (i.e. to 9%) would be estimated to prevent 366 deaths per year (Table 2.4).

Table 2.4: The estimated number of avoided cardiovascular disease deaths in NSW per year that would result from a 25% reduction in the proportion sitting for 10 or more hours a day

Sitting for ≥10 hours per day (2011-12)	RR of all-cause mortality[115]	PAR	NSW incidence of all-cause deaths (2012) [135]	Number attributable to sitting for ≥10 hours per day	Avoided cases per year
12%	1.34	3%	49,314	1464	366

RR = relative risk; relative to sitting <7 hours per day, adjusted for physical activity

Case study 5: Overweight and obesity and type 2 diabetes

In 2013, 39% of NSW male adults and 25% of NSW female adults were estimated to be overweight. As a result, approximately 266 cases of type 2 diabetes per year could be avoided in men and 200 cases in women if the prevalence of overweight was reduced by 5% (Table 2.5a). A similar 5% reduction in the prevalence of obesity would prevent 185 cases of type 2 diabetes in obese men and 186 cases in obese women per year (Table 2.5b).

Table 2.5a: The estimated number of avoided cases of type 2 diabetes in NSW per year that would result from a 5% reduction in overweight

	Prevalence of overweight (2013)[127]	RR of type 2 diabetes[11]	PAR	Estimated NSW incidence of type 2 diabetes[i]	Number attributable to overweight	Avoided cases per year	Total avoided cases
Men	39%	2.40	22.5%	23,671	5316	266	466
Women	25%	3.92	18.7%	21,368	3995	200	

Table 2.5b: The estimated number of avoided cases of type 2 diabetes in NSW per year that would result from a 5% reduction in obesity

	Prevalence of over-weight (2013)[127]	RR of type 2 diabetes[11]	PAR	Estimated NSW incidence of type 2 diabetes[i]	Number attributable to obesity	Avoided cases per year	Total avoided cases
Men	18%	6.74	15.7%	23,671	3709	185	371
Women	19%	12.41	17.4%	21,368	3713	186	

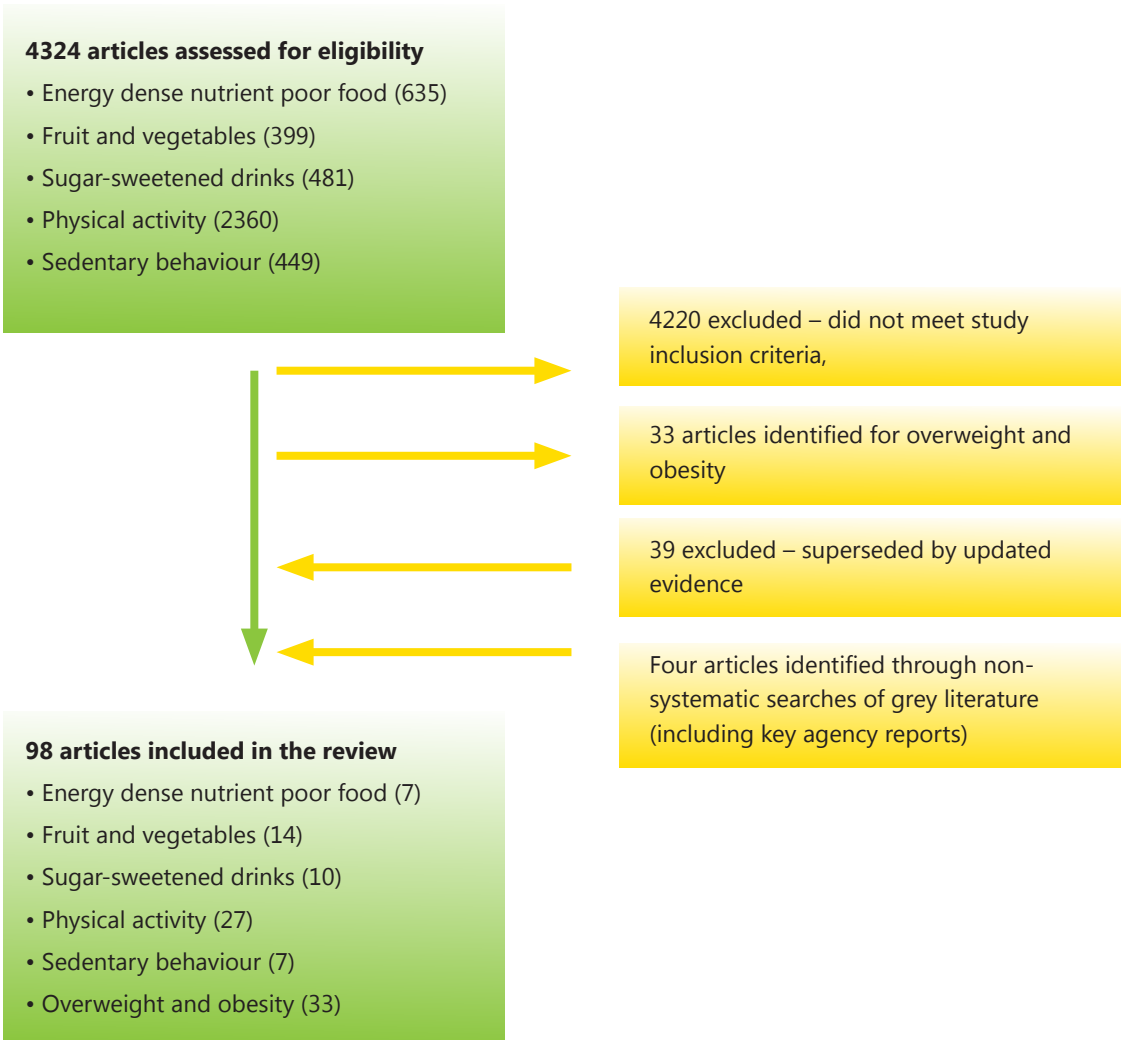
RR = relative risk; relative to healthy weight

[h] Bauman A, Ainsworth BE, Sallis JF, Hagstromer M, Craig CL, Bull FC, et al. The descriptive epidemiology of sitting. A 20-country comparison using the International Physical Activity Questionnaire (IPAQ). Am J Prev Med. 2011; 41(2): 228-35

[i] Estimates were based on Magliano DJ, Barr ELM, Zimmet PZ, Cameron AJ, Dunstan DW, Colagiuri S, et al. Glucose indices, health behaviors, and incidence of diabetes in Australia: the Australian Diabetes, Obesity and Lifestyle Study. Diabetes Care. 2008; 31(2): 267-72

APPENDICES

Appendix 1: Results of the searches



Appendix 2: Details of studies used in the review

Details of studies used to present information on level of association are presented in the tables. Additional references identified are listed below the table.

Table A2.1 Energy dense nutrient poor food

Reference	Study design	Length of follow-up	Outcome	No. of individuals	Countries (no. of studies)	Food type	Categorisation	HR/OR/RR; CI
Bahadoran et al (2013)[42]	COH	At least 3 years (1999–2008)	**Metabolic syndrome**	1476	Iran	Fast food	Quartiles of fast food consumption (a) Q1 – lowest consumption (ref) (b) Q2 (c) Q3 (d) Q4 – highest consumption	OR: (d) 1.85; 1.17–2.95
Brock et al (2009)[45]	C-C		**Renal cell carcinoma**	323 cases 1820 controls	USA	Dietary fat	Percentage of total energy from fat consumption (a) <32% (ref) (b) 32–35% (c) 36–40% (d) >40%	OR: (b) 1.4; 1.0–2.5 (c) 2.3; 1.6–4.2 (d) 2.0; 1.3–3.0
Morgenstern et al (2009)[47]	Ecological		**Stroke**	64 neighbourhoods	USA	Fast food	Distribution of fast food restaurants – highest vs lowest (ref)	RR: 1.13; 1.02–1.25
Odegaard et al (2012)[43]	COH	Up to 11 years (1993–2004)	**Type 2 diabetes**	43,176	Singapore	Fast food	Intake of fast food (a) None (ref) (b) 1–3 time/month (c) 1/week (d) ≥2/week	HR: (d) 1.27; 1.03–1.54
			Coronary heart disease mortality	52,584				HR: (d) 1.56; 1.18–2.06

Stott-Miller et al (2013)[13,6]	C-C		**Prostate cancer**	1549 cases 1492 controls	USA	Deep fried foods	Frequency of consumption of selected foods (a) <1/month (ref) (b) 1–3 time/month (c) ≥1/week DRR - 1 unit increase in DED	OR French fries: (c) 1.37; 1.11–1.69 Fried chicken: (c) 1.30; 1.04–1.62 Fried fish: (c) 1.32; 1.05–1.66 Doughnuts: (c) 1.35; 1.11–1.66
Wang et al (2008)[44]	COH	Up to 11 years (1993-2004)	**Type 2 diabetes**	21,919	UK	Dietary energy density	Quintiles of dietary energy density (a) Q1 – least energy dense (ref) (b) Q2 (c) Q3 (d) Q4 (e) Q5 – most energy dense DRR - 1 unit increase in DED	OR: (e) 1.58; 1.18–2.12 DRR – 1.20; 1.05–1.37
Wang et al (2013)[48]	C-C		**Pancreatic cancer**	908 cases 1067 controls	China	Dietary energy density	Quintiles of dietary energy density (a) Q1 – least energy dense (ref) (b) Q2 (c) Q3 (d) Q4 (e) Q5 – most energy dense DRR - 1 unit increase in DED	RR: (d) 1.51; 1.11–2.05 (e) 1.72; 1.25–2.35 DRR – 1.16; 1.07–1.27

Abbreviations: SB: sedentary behaviour; SR: systematic review; MA: meta-analysis; COH: cohort study; C-C: case-control study; CSS: cross-sectional study; DRR: Dose response relationship; HR: hazard ratio; OR: odds ratio; RR: relative risk

Table A2.2 Fruit and vegetables

Reference (Nature of evidence)	Study design	Search period	Outcome	No. of individuals in analysis	Countries (no. of studies)	Categorisation	Association with fruit consumption			Association with vegetables consumption			Association with fruit and vegetables consumption
							RR; 95% CI	Dose response relationship		RR; 95% CI	Dose response relationship		
								Linear (Y/N)	Non-linear (Y/N)		Linear (Y/N)	Non-linear (Y/N)	RR; 95% CI
Aune et al. (2011)[53]	SR & MA (19 COH)	Up to 2010	**Colorectal cancer**	Fruit & Veg: >1.52m	Asia (4) Europe (4) North America (11)	High vs. low intake (ref) and 100g/day increments for assessing DRR	0.90; 0.83–0.98	N	Y	0.91; 0.86–0.96	Y (Colon cancer only)	Y	0.92; 0.86–0.99 (Colon cancer only)
Chen et al. (2013)[60]	SR & MA (6 COH & 11 C-C)	1966-2012	**Non-Hodgkin's lymphoma**		Europe (4) North America (11) South America (2)	High vs. low intake (ref) and 1 serve (80g)/day for assessing DRR	NA	Y 0.92; 0.87-0.96	N	0.81; 0.71–0.92	N	N	0.78; 0.66–0.92
Pavia et al. (2006)[56]	SR & MA (1 COH & 15 C-C)	Up to 2005	**Oral cancer**	Fruit: 65,802 Veg: 57,993	Asia (4) Europe (7) North America (2) South America (3)	Per portion of fruit or vegetable/day	0.51; 0.40–0.65	NA	NA	0.50; 0.38–0.65	NA	NA	NA
Wang, Q. et al. (2014)[52]	SR & MA (17 COH)	1990-2013	**Gastric (stomach) cancer**	>2.4m	Asia (8) Europe (4) North America (5)	High vs. low intake (ref) and 100g/day increments for assessing DRR	0.90; 0.83–0.98	Y	Y	N	N	N	NA
Liu et al. (2013)[58]	SR & MA (5 COH & 27 C-C)	Up to 7/2012	**Oesophageal cancer ***	10,037	Asia (18) Europe (7) North America (3) South America (4)	High vs. low intake (ref) 100g/day increments for assessing DRR	0.53; 0.44–0.64	Y	Y	0.56; 0.45-0.69	Y	Y	NA

Table A2.2 Fruit and vegetables

Reference (Nature of evidence)	Study design		Outcome	No. of individuals in analysis	Countries (no. of studies)	Categorisation	Association with fruit consumption			Association with vegetables consumption			Association with fruit and vegetables consumption
							RR; 95% CI	Dose response relationship		RR; 95% CI	Dose response relationship		RR; 95% CI
								Linear (Y/N)	Non-linear (Y/N)		Linear (Y/N)	Non-linear (Y/N)	
Bandera et al. (2007)[59]	SR & MA (1 COH & 20 C-C)	Up to 2006	**Endometrial cancer**	61,618	Asia (3) Europe (10) North America (8)	High vs low intake (ref) 100g/day increments for assessing DRR	N	N	NA	0.71; 0.55–0.91	Y	NA	NA
Wakai et al. (2011)[57]	SR & MA (6 COH & 4 C-C)	1980–2009	**Lung cancer**	517,956	Japan (10)	Highest vs lowest intake (ref) and 1 serve (80g) per day for assessing DRR	0.85; 0.75–0.96	NA	Y	NR	NR	NA	NA
Carter et al. (2010)[51]	SR & MA (6 COH)	1950–2009	**Type 2 diabetes**	F&V: 155,017 F: 213,217 V: 213,217 GLV: 177,229	Asia (1) Europe (1) North America (4)	Highest vs lowest intake (ref)	NA	NA	NA	0.86; 0.77–0.97	NA	NA	NA
He et al. (2006)[137]	SR & MA (9 COH)	1966–2005	**Stroke**	F&V: 257,551	Asia (1) Europe (3) North America (5)	<3 serves/d (ref) vs (a) 3-5 serves/d (b) >5 serves/d	NA	NA	NA	NA	NA	NA	(a) 0.89; 0.83–0.97 (b) 0.74; 0.69–0.79
He et al. (2007)[131]	SR & MA (13 COH)	1966–2005	**Coronary heart disease**	F&V: 278,459	Europe (4) North America (9)	>5 serves/day vs <3 serves/ day (ref)	NA	NA	NA	NA	NA	NA	0.83; 0.77-0.89
Jung et al. (2013)[55]	MA (20 COH)	/	**Estrogen receptor-negative breast cancer**	Total: 993,466	Asia (1) Australia (1) Europe (4) North America (14)	Highest vs lowest quintile (ref)	N	NA	NA	0.82; 0.74–0.90	NA	NA	Y (*P trend* across quintiles = 0.03)

Table A2.2 Fruit and vegetables

Reference (Nature of evidence)	Study design		Outcome	No. of individuals in analysis	Countries (no. of studies)	Categorisation	Association with fruit consumption			Association with vegetables consumption			Association with fruit and vegetables consumption
							RR; 95% CI	Dose response relationship		RR; 95% CI	Dose response relationship		
								Linear (Y/N)	Non-linear (Y/N)		Linear (Y/N)	Non-linear (Y/N)	RR; 95% CI
Muraki et al. (2013)[50]	MA (3 COH)	/	**Type 2 diabetes**	F: 187,382	North America	<4 serves/w (ref) vs (a) 5–6 serves/w (b) 1 serve/d (c) 2 serves/d (d) ≥3 serves/d DRR assessed by modelling medians across categories	HR: (a) 0.91; 0.85-0.99 (b) 0.88; 0.82-0.95 (c) 0.87; 0.81-0.94 (d) 0.88; 0.81-0.96	Y	HR: 0.98; 0.96–0.99	NA	NA	NA	
Wang, X. et al. (2014)[49]	SR & MA (16 COH)	Up to 2013	**All-cause mortality**	833,234	Asia (4) Europe (6) North America (6)	(a) <1 serves/d (ref) (b) 1–2 serves/d (c) 3–4 serves/d (d) 5–6 serves/d (e) 7+ serves/d	HR: (d) 0.94; 0.90–0.98	NA	NA	HR: (d) 0.95; 0.92–0.99	NA	NA	HR: (d) 0.95; 0.92–0.98
			Cancer mortality				HR: (d) 0.99; 0.97–1.00			N			N
			Cardiovascular mortality				HR: (d) 0.95; 0.91–1.00			HR: (d) 0.96; 0.93–0.99			HR: (d) 0.96; 0.92–0.99
Aune et al. (2010)[54]	SR&MA (14 COH)	Up to April, 2011	**Breast cancer**	F&V combined: 233,036 F: 785,668 V: 751,965	Asia (3) Europe (5) North America (7)	High vs low intake 200g/day increments for DRR	RR: 0.92; 0.86–0.98	Y RR: 0.94; 0.89–1.00	N	N	N	N	RR: High vs low -0.89; 0.80–0.99 DRR – 0.96; 0.93–1.00

Abbreviations: SR: systematic review; MA: meta-analysis; COH: prospective cohort study; C-C: case-control study; NC-C: nested case-control study; RR: relative risk; DRR: dose response relationship; NR: not reported; Y: yes; N: no; NA: not assessed; ref: reference group; d: day; w: week;

*specifically oesophageal squamous cell carcinoma (OSCC)

Table A2.3 Sugar-sweetened beverages

Reference	Study design (no, studies included)	Search period or length of follow-up	Outcome	No. of individuals in analysis	Countries (no. of studies)	Categorisation	Association with sugar-sweetened beverage consumption		
							RR; 95% CI	Dose response relationships	
								Linear (Y/N)	Non-linear (Y/N)
Greenwood et al. (2014)[63]	SR & MA (9 COH)	January 1990 to November 2009	**Type 2 diabetes**	≈280,000	Asia (2) Europe (2) North America (5)	330ml/day increments for assessing DRR	NA	N	Y RR: 1.20; 1.12–1.29
Huang et al. (2014)[64]	SR & MA (4 COH)	Up to February 2014	**Coronary heart disease**	173,743	Asia (1) North America (3)	High vs. low (ref) consumption 1 serving per day increase for DRR	RR: 1.17; 1.07–1.28	Y RR: 1.16; 1.10–1.23	NR
Xi et al. (2014)[138]	SR & MA (10 COH)	Up to December 2013	**Type 2 diabetes**	375,261	Asia (3) Europe (3) North America (4)	High vs. low (ref) consumption of sugar-sweetened fruit juice	RR: 1.28; 1.04–1.59	NA	NA
Genkinger (2012)[65]	MA (14 COH)	–	**Pancreatic cancer**	853,894	Australia (1) Europe (3) North America (6) Unclear (4)	>250g/day vs 0g/day (ref) 177.5g per day increment for DDR	RR: 1.19; 0.98–1.46	Y RR: 1.06; 1.02–1.12	N
Choi & Curhan (2008)[66]	COH	12 years follow-up (1986–1998)	**Gout (men)**	46,393	USA	<1 serving/month (ref) vs (a) ≥1 serving a month to 1 serving a week, (b) 2-4 servings/ week (c) 5-6 servings/week (d) 1 serving/day (e) ≥2 servings/day	RR: (c) 1.29; 1.00–1.68 (d) 1.45; 1.02–2.08 (e) 1.85; 1.08–3.16	NA	NA
Choi et al. (2010)[67]	COH	22 years follow-up (1984–2004)	**Gout (women)**	78,906	USA	<1 serving/month (ref) vs (a) ≥1 serving a month to 1 serving a week, (b) 2-4 servings/ week (c) 5-6 servings/week (d) 1 serving/ day (e) ≥2 servings/day	RR: (d) 1.74; 1.19–2.55 (e) 2.39; 1.34–4.26	NA	NA
Aidi et al. (2011)[68]	COH	3 years follow-up	**Dental erosion in children**	572	Netherlands	Any consumption vs no consumption (ref)	OR: 1.04; 1.01–1.07	NA	NA
Cohen et al. (2012)[69]	COH	28 years follow-up in women (1980–2008) and 22 years in men (1986–2008)	**Hypertension**	186,531 women 37,360 men	USA	<1 serving/month (ref) vs. (a) 1–4/month (b) 2–6/week (c) ≥1/day	RR: (b): 1.06; 1.03–1.08 c): 1.13; 1.09–1.17	NA	NA

Table A2.3 Sugar-sweetened beverages

Reference	Study design (no, studies included)	Search period or length of follow-up	Outcome	No. of individuals in analysis	Countries (no. of studies)	Categorisation	Association with sugar-sweetened beverage consumption		
							RR; 95% CI	Dose response relationships	
								Linear (Y/N)	Non-linear (Y/N)
Ferraro et al. (2013)[70]	COH	Median follow-up of >8 years	**Kidney stones**	194,095	USA	Cola and non-cola <1 serving/week (ref) vs. (a) 1/week (b) 2–4/week (c) 5–6/week, (d) ≥1/day	Cola (d): 1.23; 0.98–1.55 Non-cola (d): 1.33; 1.01–1.74	NA	NA
Larsson et al. (2014)[71]	COH	10 years (1998–2008)	**Stroke**	32,575 women 35,884 men	Sweden	0.1 to <0.5 servings/day (ref) vs. (a) none (b) 0.5 to <1.0/day (c) 1.0 to <2.0 (d) ≥2/day	(d): 1.19; 1.19–1.36		

Abbreviations: SR: systematic review; MA: meta-analysis; Coh: prospective cohort study; C-C: case-control study; NC-C: nested case-control study; RR: relative risk; DRR: dose response relationship; NR: not reported; Y: yes; N: no; NA: not assessed; ref: reference group; d: day; w: week; OR: odds ratio

Table A2.4 Physical activity

Reference	Study design (no. studies included)[a]	Search period	Outcome	No. of individuals	Countries (no. of studies)	PA type/domain/ intensity	Categorisation	Association with total PA	
								Y/N	HR/OR/RR; CI
Aune et al. (2014)[94]	SR & MA (11 COH & 4 C-C)	Up to 11/2012	**Pre-eclampsia**	190,749	Asia (1) Europe (7) North America (7)	(a) Prepregnancy PA (b) PA in early pregnancy (c) combined a and b (d) walking (e) occupational PA	High vs Low (ref) PA	(a) Y (b) Y (c) Y (d) Y (e) N	RR: (a) 0.65, 0.47–0.89 (b) 0.79, 0.70–0.91 (c) 0.64, 0.44–0.93 (d) 0.68, 0.51–0.89 (e) 0.82, 0.66–1.03
Behrens and Leitzmann (2013)[92]	SR & MA (11 COH & 8 C-C)	Up to 09/2012	**Renal cancer**	2,327,322	Asia (2) Europe (7) North America (10)	Not specified	High vs Low (ref) PA	Y	RR: 0.88, 0.79–0.97
Behrens et al. (2014)[91]	SR & MA (9 COH & 15 C-C)	Up to 12/2013	**Gastroesophageal cancer**	1,698,207	Asia (10) Europe (6) North America (7) Australia (1)	Not specified	High vs Low (ref) PA	Y	RR: 0.82, 0.74–0.90
Blondell et al. (2014)[87]	SR & MA (17 COH)	Up to 12/2013	**Cognitive decline**	NR	NR	Not specified	Highest vs Lowest (ref) PA category	Y	RR: 0.65, 0.55–0.76
	SR & MA (21 COH)		**Dementia**	NR	NR	Not specified	Highest vs Lowest (ref) PA category	Y	RR: 0.86, 0.76–0.97
Boyle et al. (2012)[90]	SR & MA (12 COH & 9 C-C)	1946-2012	**Proximal colon cancer**	NR	Asia (3) Europe (8) North America (9) Australia (1)	Not specified	Highest vs Lowest (ref) PA category	Y	RR: 0.73, 0.66-0.81
			Distal colon cancer	NR		Not specified	Highest vs Lowest (ref) PA category	Y	RR: 0.74, 0.68–0.80
Cheng et al. (2007)[99]	MA (7 CSS)	1990-2006	**Erectile dysfunction**	11,844	Asia (2) Europe (2) South America (2) International (1)	Not specified	Above average PA vs no PA (ref)	Y	OR: 0.53, 0.31–0.91
Diep et al. (2010)[81]	MA (13 COH)	1986-2005	**Stroke outcome (incidence and/or mortality)**	255,873	Asia (1) Europe (3) North America (9)	Not specified	Low PA (ref) vs. (a) moderate PA (b) high PA	Y	RR: (a) 0.89, 0.85–0.94 (b) 0.79, 0.74–0.85
Eijkemans et al. (2012)[85]	SR & MA (4 COH)	Up to 06/2011	**Asthma**	85,117	Europe (3) North America (1) Australia (1)	Not specified	High vs Low (ref) PA	Y	OR: 0.87, 0.77–0.99

Table A2.4 Physical activity

Reference	Study design (no. studies included)[a]	Search period	outcome	No. of individuals	Countries (no. of studies)	PA type/domain/ intensity	Categorisation	Association with total PA	
								Y/N	HR/OR/RR; CI
Gonçalves et al. (2014)[139]	SR & MA (7 COH & 14 C-C)	2000-2010	**Breast cancer**	COH 743,201 C-C 64,265	Asia (4) Europe (8) North America (9)	Not specified	Not specified	Y	OR: COH 0.84, 0.81–0.88 C-C 0.61, 0.59–0.63
Hamer and Chida (2008)[140]	SR & MA (18 COH)	1970-2007	**Cardiovascular disease**	459,833	Asia (4) Europe (4) North America (10)	Walking	Highest vs Lowest (ref) walking category	Y	HR 0.69, 0.61–0.77
			All-cause mortality					Y	HR: 0.68, 0.59–0.78
He et al. (2014)[83]	MA (14 COH)	Up to 06/2013	**Metabolic syndrome**	64,353	Asia (1) Europe (8) North America (4) South America (1)	Leisure-time PA	Low PA level (ref) vs. (a) moderate PA level (b) high PA level	Y	RR: (a) 0.95, 0.91–1.00 (b) 0.80, 0.75–0.85
Huai et al. (2013)[74]	MA (13 COH)	Up to 11/2013	**Hypertension**	136,846	Asia (2) Europe (6) North America (5)	Recreational PA Occupational PA	Low PA level (ref) vs. (a) moderate PA level (b) high PA level	Recreational PA Y Occupational PA N	RR: Recreational PA (a) 0.89, 0.85–0.94 (b) 0.81, 0.76–0.85 Occupational PA (a) 0.96, 0.87–1.06 (b) 0.93, 0.81–1.08
Jeon et al. (2007)[84]	SR & MA (10 COH)	Up to 03/2006	**Type 2 diabetes**	301,221	Asia (1) Europe (3) North America (6)	(a) Moderate-intensity PA (b) Walking	(a) insufficient PA (ref) vs regular PA (b) <2.5 h/w (ref) vs ≥2.5 h/w	Y	RR: (a) 0.69, 0.58–0.83 (b) 0.70, 0.58–0.84
Johnson et al. (2013)[128]	MA (12 COH & 9 C-C)	1966–2010	**Colorectal cancer**	NR	NR	Not specified	An increase of 2 in standardized PA score	Y	RR: 0.88, 0.86–0.91
Keimling et al. (2014)[75]	SR & MA (11 COH & 4 C-C)	1975–2013	**Bladder cancer**	5,402,369	Asia (1) Europe (6) North America (8)	(a) Non-specific PA (b) Recreational PA (c) Occupational PA (d) Moderate-intensity PA (e) Vigorous-intensity PA	Highest vs Lowest (ref) PA category	(a) Y (b) Y (c) N (d) Y (e) N	RR: (a) 0.85, 0.74–0.98 (b) 0.81, 0.66–0.99 (c) 0.90, 0.76–1.00 (d) 0.85, 0.75–0.98 (e) 0.80, 0.64–1.00

Table A2.4 Physical activity

Reference	Study design (no. studies included)[a]	Search period	Outcome	No. of individuals	Countries (no. of studies)	PA type/domain/ intensity	Categorisation	Association with total PA	
								Y/N	HR/OR/RR; CI
Keum et al. (2014)[93]	MA (10 COH & 10 C-C)	Up to 09/2013	**Endometrial cancer**	659,662	Asia (2) Europe (8) North America (10)	Leisure-time PA	Highest vs Lowest (ref) PA category	Y	RR: 0.82, 0.75–0.90
Lageross et al. (2004)[89]	MA (4 COH & 19 C-C)	1966–2002	**Breast cancer**	28,079	Asia (2) Europe (5) North America (16)	PA in adolescence and young adulthood	Highest vs Lowest (ref) PA category	Y	RR: 0.81, 0.73–0.89
Li et al. (2012)[73]	SR & MA (21 COH)	1980–2010	**Coronary heart disease**	>650,000	Europe (5) North America (16)	(a) Leisure-time PA (b) Occupational PA	Highest vs Lowest (ref) PA category	Women (a) Y (b) Y Men (a) Y (b) Y	RR: Women (a) 0.71, 0.65–0.77 (b) 0.80, 0.71–0.91 Men (a) 0.79, 0.73–0.85 (b) 0.91, 0.83–0.99
			Stroke					Women (a) Y (b) N Men (a) Y (b) N	RR: Women (a) 0.78, 0.66–0.92 (b) 0.89, 0.78–1.03 Men (a) 0.71, 0.60–0.84 (b) 0.90, 0.80–1.03
Liu et al. (2011)[76]	SR & MA (19 COH & 24 C-C)	Up to 05/2011	**Prostate cancer**	2,123,799	Asia (5) Europe (15) North America (23)	(a) Non-specific PA (b) Recreational PA (c) Occupational PA	Highest vs Lowest (ref) PA category	(a) Y (b) Y (c) Y	RR: (a) 0.90, 0.84–0.95 (b) 0.95, 0.89–1.00 (c) 0.81, 0.73–0.91
Moayyeri (2008)[86]	MA (13 COH)	Up to 06/2008	**Hip fractures**	286,756	NR	Moderate to vigorous-intensity PA	NR	Y	RR: Women 0.62, 0.56–0.69 Men 0.55, 0.44–0.69
Nocon et al. (2012)[80]	SR & MA (16 COH)	Up to 05/2007	**Cardiovascular disease mortality**	550,224	NR	Not specific	The least active (ref) vs the most active population subgroups	Y	RR: 0.70, 0.66–0.74
	(21 COH)		**All-cause mortality**	640,073				Y	RR: 0.71, 0.66–0.76

Table A2.4 Physical activity

Reference	Study Design (no. studies included)[a]	Search period	Outcome	No. of individuals	Countries (no. of studies)	PA type/domain/ intensity	Categorisation	Association with total PA	
								Y/N	HR/OR/RR; CI
O'Rorke et al. (2009)[77]	SR & MA (28 COH)	Up to 07/2009	**Pancreatic cancer morbidity/ mortality**	>1.7m	NR	(a) Total PA (b) Occupational PA (c) Recreational PA (d) Transport PA (e) Light-intensity PA (f) Moderate-intensity PA (g) Vigorous-intensity PA	Highest vs Lowest (ref) PA category	RR: (a) Y (b) Y (c) N (d) N (e) N (f) N (g) N	RR: (a) 0.72, 0.52–0.99 (b) 0.75, 0.59–0.96 (c) 0.94, 0.88–1.01 (d) 0.77, 0.55–1.09 (e) 1.01, 0.77–1.34 (f) 0.79, 0.52–1.20 (g) 0.97, 0.88–1.07
Samitz et al. (2011)[72]	SR & MA (80 COH)	Up to 09/2010	**All-cause mortality**	1,338,143	Asia/Australia (12) Europe (42) North America (26)	(a) Total PA (b) Leisure-time PA (c) Activities of daily living (d) Occupational PA (e) Moderate-intensity PA (f) Vigorous-intensity PA (g) Moderate to vigorous-intensity PA	(a-d) Highest vs Lowest (ref) PA category (e-f) Per 1-h increment per week (g) Meeting MVPA recommendation vs not meeting MVPA recommendation (ref)	Y	RR: (a) 0.65, 0.60–0.71 (b) 0.74, 0.70–0.77 (c) 0.64, 0.55–0.75 (d) 0.83, 0.71–0.97 (e) 0.96, 0.93–0.98 (f) 0.91, 0.87–0.94 (g) 150 min/w 0.86, 0.80–0.92 (g) 300 min/w 0.74, 0.65–0.85
Sattelmair et al. (2011)[82]	MA (33 COH)	1995–2009	**Coronary heart disease**	NR	NR	Moderate-intensity leisure-time PA	No moderate-intensity leisure-time PA vs (a) ≥150 min/w (b) ≥300 min/w	Y	RR: (a) 0.86, 0.77–0.96 (b) 0.80, 0.74–0.88
Sun et al. (2012)[88]	SR & MA (14 COH)	Up to 05/2012	**Lung cancer**	1,644,305	NR	Not specified	Low PA level (ref) vs. (a) moderate PA level (b) high PA level	Y	RR: (a) 0.87, 0.83–0.90 (b) 0.77, 0.73–0.81

Table A2.4 Physical activity

Reference	Study Design (no. studies included)[a]	Search period	Outcome	No. of individuals	Countries (no. of studies)	PA type/domain/ intensity	Categorisation	Association with total PA	
								Y/N	HR/OR/RR; CI
Tobias et al. (2011)[95]	SR & MA (5 COH, 2 C-C & 2 CSS)	Up to 03/2010	**Gestational diabetes**	34,929	Europe (1) North America (7)	(a) Total PA (b) Walking (c) Stair climbing (d) Vigorous-intensity PA	Highest vs Lowest (ref) PA category	Prepregnancy (a) Y (b) N (c) Y (d) Y Early pregnancy (a) Y (b) N (c) NA (d) N	OR: Prepregnancy (a) 0.45, 0.28–0.75 (b) 0.95, 0.50–1.83 (c) 0.49, 0.26–0.72 (d) 0.47, 0.19–0.75 Early pregnancy (a) 0.76, 0.70–0.83 (b) 0.77, 0.51–1.16 (c) NA (d) 0.55, 0.21–1.43
Zhou et al. (2014)[96]	SR & MA (3 COH & 3 CSS)	Up to 02/2014	**Ovarian cancer**	435,398	Europe (1) North America (5)	Recreational PA	High vs Low (ref) PA	COH: N C-C: Y Total: N	RR: COH 1.12, 0.88–1.42 C-C 0.76, 0.64–0.90 Total 0.90, 0.72–1.12

Abbreviations: SR: systematic review; MA: meta-analysis; COH: cohort study; C-C: case-control study; CSS: cross-sectional study; PA: physical activity; MVPA: moderate to vigorous-intensity physical activity; w: week; HR: hazard ratio; OR: odds ratio; RR: relative risk

Table A2.5 Sedentary behaviour

Reference	Study Design (no. studies included)[a]	Search period	Outcome	No. of individuals	Countries (no. of studies)	SB type	Categorisation	Association with SB Y/N	HR/OR/RR; CI
Chau et al. (2014)[115]	MA (6 COH)	1989–2013	**All-cause mortality**	595,086	Asia (1) Australia (2) Europe (1) North America (2)	Total sitting	Per hour/day increase	Y	HR: 1.02, 1.01–1.03
Cong et al. (2014)[117]	MA (12 COH & 9 C-C)	Up to 05/2013	**Colon cancer**	4,324,462	Asia (1) Australia (2) Europe (12) North America (4) International (2)	Not specified	Various definitions in original studies	Y	RR: 1.30, 1.22–1.39
Edwardson et al. (2014)[116]	SR & MA (10 CSS)	Up to 01/2011	**Metabolic syndrome**	21,393	NR	Not specified	Highest vs Lowest (ref) sedentary behaviour category	Y	OR: 1.73, 1.55–1.94
Ford and Caspersen (2012)[111]	MA (7 COH)	NR	**Cardiovascular disease**	352,198	Australia (1) Europe (2) North America (4)	(a) Total sitting (b) Screen time	Per 2 hours/day increase	Y	HR: (a) 1.05, 1.01–1.09 (b) 1.17, 1.13–1.20
Grøntved and Hu (2011)[114]	SR & MA (4 COH)	1970–2011	**Type 2 diabetes**	175,938	Europe (1) North America (3)	TV viewing	Per 2 hours/day increase	Y	RR: 1.20, 1.14–1.27
	SR & MA (4 COH)		**Cardiovascular disease**	34,253	Australia (1) Europe (2) North America (1)			Y	RR: 1.15, 1.06–1.23
	SR & MA (3 COH)		**All-cause mortality**	26,509	Australia (1) Europe (2)			Y	RR: 1.13, 1.07–1.18
Schmid and Leitzmann (2014)[118]	SR & MA (5 COH & 3 C-C)	Up to 02/2014	**Colon cancer**	2,220,421	Asia (1) Australia (1) Europe (5) North America (1)	(a) TV viewing (b) Occupational sitting (c) Total sitting (d) Non-specific	Highest vs Lowest (ref) sedentary behaviour category	(a) Y (b) Y (c) Y (d) Y	RR: (a) 1.54, 1.19–1.98 (b) 1.24, 1.09–1.41 (c) 1.24, 1.03–1.50 (d) 1.28, 1.13–1.45
	SR & MA (3 COH & 5 C-C)		**Endometrial cancer**	155,804	Asia (2) Europe (2) North America (4)			(a) Y (b) Y (c) N (d) Y	RR: (a) 1.66, 1.21–2.28 b) 1.11, 0.88–1.39 (c) 1.32, 1.08–1.61 (d) 1.36, 1.15–1.60

Table A2.5 Sedentary behaviour

Reference	Study Design (no. studies included)[a]	Search period	Outcome	No. of individuals	Countries (no. of studies)	SB type	Categorisation	Association with SB Y/N	HR/OR/RR; CI
	SR & MA (2 COH & 1 C-C)		**Lung cancer**	185,466	Asia (1) Europe (1) North America (1)			(d) Y	RR: (d) 1.21, 1.03–1.43
Wilmot et al. (2014)[141]	SR & MA (5 COH & 5 CSS)	Up to 01/2012	**Type 2 diabetes**	505,634	Australia (2) Europe (3) North America (5)	TV viewing / Screen based entertainment	Highest vs Lowest (ref) sedentary behaviour category	Y	RR: 2.12, 1.61–2.78
	SR & MA (8 COH)		**Cardiovascular disease mortality**	421,921	Australia (1) Europe (2) North America (5)	Not specified		Y	RR: 1.90, 1.36–2.66
	SR & MA (8 COH)		**All-cause mortality**	497,211	Asia (1) Australia (1) Europe (2) North America (4)	Not specified		Y	RR: 1.49, 1.14–2.03

Abbreviations: SB: sedentary behaviour; SR: systematic review; MA: meta-analysis; COH: cohort study; C-C: case-control study; CSS: cross-sectional study; HR: hazard ratio; OR: odds ratio; RR: relative risk

Appendix 3: Methodology for Review Question 2

Population attributable risks (PAR) were estimated to quantify the level of benefit that could be expected if the HEAL targets and objectives were met for selected health outcomes. PARs provide an estimate of the proportion of events (i.e. cases, deaths, hospitalisations etc.) that can be attributed to a particular risk factor; for example, the proportion of lung cancer cases that are attributable to smoking.

PARs were calculated using the following formula:

$$PAR = \frac{P_e (RRe - 1)}{RRe}$$

Where P_e is the prevalence of the risk factor in the population and is the relative risk of the event, adjusted for confounding factors.

Relative risks were sourced from meta-analyses included in this review. Prevalence of inadequate physical activity (less than 150 minutes of physical activity per week), insufficient consumption of fruit (less than two serves per day) and vegetables (less than five serves per day), and overweight and obesity were sourced from the NSW Population Health Survey.[127]

Prevalence of any soft drink consumption and the proportion sitting for 10 or more hours a day (a proxy for sedentary behaviour) were unavailable for NSW and so Australia-wide data were used. Soft drink consumption was sourced from the Australian Health Survey (AHS),[133] while prevalence of sitting time was calculated by the Prevention Research Collaboration using unpublished data from the Australian Health Survey.

After calculating the PAR for each risk factor and associated outcome, we then calculated the proportion of events that would be avoided if the HEAL targets and objectives are met. This was done using same formula as used in Lee (2012)[142:]

$$\text{Avoided events} = I_e \times PAR \times \triangle$$

Where I_e is the incidence of the outcome in the NSW population and $\triangle$ is the targeted change in the risk factor.

The targeted change in the prevalence of overweight and obesity has been set by the HEAL Strategy at 5% by 2020. For the other risk factors, targeted change was set at 25% as this was felt to be realistic over the longer term while still representing a meaningful level of change. Incidence data were available for colorectal and breast cancer from the NSW Cancer Registry.[129] All-cause mortality data were sourced from the Australian Bureau of Statistics,[135] while estimates of type 2 diabetes and coronary heart disease[132] incidence were extrapolated from data available from the AusDiab study and the Australian Institute of Health and Welfare, respectively.

Appendix 4: Specifications for the review

Introduction

An Evidence Check review is a rapid review of existing evidence tailored to the individual needs of an agency. Evidence Check reviews answer specific policy questions and are presented as a short report in a policy-friendly format. Reviewers identify gaps in the evidence but do not undertake new research to fill these gaps.

Background and context

The NSW Healthy Eating and Active Living (HEAL) Strategy is a five-year whole-of-government plan linked to achievement of a number of goals within the state plan NSW 2021. The overarching goal of the HEAL Strategy is to keep people healthy and out of hospital by achieving seven targets related to improving nutrition and physical activity levels within the NSW population. Two of these targets relate specifically to overweight and obesity rates in the NSW population (see HEAL Strategy page 5: http://www.health.nsw.gov.au/obesity/Publications/nsw-healthy-eating-strategy.pdf). The plan has four strategic directions: environments to support healthy eating and active living, state-wide support programs, advice as a part of routine service delivery and education and information to enable informed healthy choices.

The six objectives within the HEAL Strategy to achieve these improvements in nutrition and physical activity levels are to:

1. Reduce intake of energy dense nutrient poor food and drinks
2. Increase consumption of fruit and vegetables
3. Increase intake of water in preference to sugar-sweetened drinks
4. Increase incidental, moderate and vigorous physical activity
5. Reduce time in sedentary behaviours
6. Increase community awareness of healthy eating and physical activity as protective factors against chronic disease.

Although having a primary focus on the prevention of overweight and obesity, the Strategy is framed towards improving nutrition and physical activity. It is acknowledged that this work will have broader impacts than health benefits associated with prevention of overweight and obesity. These impacts are expected to include impacts on other health conditions, such as CVD, cancers, hypertension, mental health and arthritis. Non-health impacts are expected to include demand for health services and financial savings for government and individuals.

Purpose and audience

The aim of this rapid review is to articulate the broader health impacts of the HEAL Strategy and demonstrate the benefits of this approach for the NSW government.

The primary audience for this review is senior decision makers in the NSW Ministry of Health.

Review questions

The review will address the following questions:

Question 1: What are the additional health conditions and wellbeing indicators (beyond overweight and obesity) where there is strong evidence that meeting the HEAL targets and six objectives (see Background) will improve outcomes?

Scope of Question 1

- The relevant HEAL targets are (a) reduction in overweight and obesity rates of children and young people (5–16 years) to 21% by 2015; and (b) stabilisation of overweight and obesity rates in adults by 2015, then reduction by 5% by 2020
- Only include evidence from whole of population, inter-sectoral interventions
- Health conditions include (but are not limited to) those that have a demonstrated risk association with overweight and/or obesity
- Outcomes of health conditions is broadly defined to include mortality, morbidity, disability or other direct or indirect outcomes (for example, reduced use of services) commonly associated with each health condition
- Wellbeing is broadly defined, and may include for example school or social participation, childhood development and health-related quality of life
- Evidence is restricted to studies where change can be attributed either wholly or in part to HEAL objectives
- Include information on important contextual factors (for example, length of time between implementation and outcomes, co-interventions, health system configuration, inter-sectoral components and arrangements, geographical and population characteristics)
- Where possible, include evidence for population age subgroups (infants, children, young adults, adults and older adults

Question 2: What is the level of change in the targets and HEAL objectives that is associated with better outcomes for the health conditions and wellbeing indicators identified in Question 1?

Scope of Question 2

- Assessment of improvement must be demonstrated using reliable and valid population-level measures.

Depth and scope of the review

The review should:

- Provide a brief summary of existing reviews of the evidence or key research papers where there is strong evidence in relation to the review questions
- Include non-health benefits (for example, improved transport infrastructure) associated with improvements in outcomes where this information is available
- Include indirect health outcomes (for example, effect on use of health services) where this information is available
- Include economic evaluations where this information is available
- Include information on the timeframe in which benefits are achieved where this information is available
- Include peer reviewed literature from 2000 to current
- Examine local and international agency reports
- Provide a glossary defining key terms
- Be suitable for use, without modification, by senior executives.

Format of the review

The review should be in the range of 10–20 pages, not including tables and references. The language of the review should be appropriate for policy makers. The review should include the following elements:

- Executive plain English summary with key points
- Background and introduction
- Description of method of searching and selecting papers for inclusion
- Tabulation of the relevant papers indicating the methods, findings and critical commentary for each study

• Analysis of evidence in relation to the review questions
• Reference list.

The table of individual papers included in the review will include:
• Study methods, including design, sample size, setting
• Study findings, i.e. statement of evidence
• Level of evidence, as defined by the researcher.

Proofreading and copy-editing

The Sax Institute provides basic proofreading of the final report to correct misspellings and other obvious errors. However, Evidence Check costs do not include copy-editing or ensuring that the report adheres to the agency's style.

Publication

By the Sax Institute:

The Sax Institute may publish the Final Report subject to consent from the commissioning agency.

By the researcher in a journal or other format:

The commissioning agency may consent to publication of work based on the review in a journal or other format after perusing a draft of the work to be submitted for publication.

Researchers are advised that they are required to submit a draft for consideration within three months of consent being granted.

Appendix 5 - The economic costs of overweight and obesity in Australia

Access Economics Report, 2008

In August 2008, Access Economics published the report: *The growing cost of obesity in 2008: three years on*. The report is available at:

https://www.diabetesaustralia.com.au/PageFiles/7830/FULLREPORTGrowingCostOfObesity2008.pdf

Using recent obesity prevalence estimates, attributable fractions (AFs) and unit cost data, the financial cost of obesity in 2008 was estimated as $8.283 billion. Of this, productivity costs were estimated as $3.6 billion (44%), health system costs were $2.0 billion (24%) and carer costs were $1.9 billion (23%). Deadweight losses (DWLs) from transfers (taxation revenue forgone, welfare and other government payments) were $727 million (9%) and other indirect costs were $76 million (1%).

The net cost of lost wellbeing (the dollar value of the burden of disease, netting out financial costs borne by individuals) was valued at a further $49.9 billion, bringing the total cost of obesity in 2008 to $58.2 billion. Of the financial costs, 29.4% are borne by individuals, 19.2% by family and friends, 34.3% by Federal Government ($2.8 billion per annum), 5.1% by State Governments, less than 0.1% by employers and 11.8% by the rest of society. However, if the cost of lost wellbeing is included, the individual's share rises markedly to 90.0% of the total.

In 2005, the economic costs were significantly lower at $21.0 billion, including $3.8 billion in financial costs and $17.2 billion in net cost of lost wellbeing. The increase of economic costs is due to a combination of factors such as cost inflation, population growth and change in methodology in relation to value per statistical life-year (VSLYs) and AFs. For instance, when the old VSLY is applied, the net cost of lost wellbeing (in 2008 dollars) would have been $32.7 billion (compared with $49.9 billion based on the new VSLY, with total economic costs of obesity amounting to $41.0 billion.

Costs of obesity in Australia (2008) by State and Territory

In line with population shares, economic costs of obesity for 2008 were largest in NSW at $19.0 billion – including $2.7 billion (14%) in financial costs and $16.3 billion (86%) in net costs of lost wellbeing) – followed by Victoria at $14.4 billion and Queensland at $11.6 billion (Table B3.1)

COSTS OF OBESITY BY STATE/TERRITORY ($M), 2008

	NSW	VIC	QLD	WA	SA	TAS	ACT	NT	Australia
% Population	32.7%	24.8%	20.0%	10.1%	7.5%	2.3%	1.6%	1.0%	100.0%
BoD	16,318	12,358	9,961	5,020	3,750	1,168	803	513	**49,896**
Health System	641	485	391	197	147	46	32	20	**1,959**
Productivity	1,187	899	724	365	273	85	58	37	**3,629**
Carers	619	469	378	190	14	44	30	19	**1,893**
DWL	238	180	145	73	55	17	12	7	**727**
Other indirect	25	19	15	8	6	2	1	1	**76**
Total financial	**2,709**	**2,052**	**1,654**	**833**	**623**	**194**	**133**	**85**	**8,283**
Total inc. BoD	**19,027**	**14,410**	**11,614**	**5,853**	**4,373**	**1,362**	**936**	**598**	**58,179**

Table B3.1 The costs of obesity in Australia in 2008, by State and Territory

Table B3.1 The costs of obesity in Australia in 2008, by State and Territory

Medical Journal of Australia – Colagiuri et al. estimates for the year 2005

This 2010 journal paper [Colagiuri S, Lee, C. M., Colagiuri, R., Magliano, D., Shaw, J. E., Zimmet, P. Z., Caterson, I. D. The cost of overweight and obesity in Australia. The Medical journal of Australia 2010; 192(5): 260–264.] described a study which was designed to assess and compare health care costs for normal-weight, overweight and obese Australians. The researchers conducted an analysis of five-year follow-up data from the Australian Diabetes, Obesity and Lifestyle study, collected in 2004–2005. Data were available for 6140 participants aged >or= 25 years at baseline. The main outcome measures were direct health care cost, direct non-health care cost and government subsidies associated with overweight and obesity, defined by both body mass index (BMI) and waist circumference (WC).

The study found that annual total direct cost (health care and non-health care) per person increased from $1472 (95% CI, $1204–$1740) for those of normal weight to $2788 (95% CI, $2542-$3035) for the obese, however defined (by BMI, WC or both). In 2005, the total direct cost for Australians aged >or= 30 years was $6.5 billion (95% CI, $5.8–$7.3 billion) for overweight and $14.5 billion (95% CI, $13.2–$15.7 billion) for obesity. The total excess annual direct cost due to overweight and obesity (above the cost for normal-weight individuals) was $10.7 billion. Overweight and obese individuals also received $35.6 billion (95% CI, $33.4–$38.0 billion) in government subsidies. Comparing costs by weight change since 1999–2000, those who remained obese in 2004–2005 had the highest annual total direct cost. Cost was lower in overweight or obese people who lost weight or reduced WC compared with those who progressed to becoming, or remained, obese.

The researchers concluded that total annual direct cost of overweight and obesity in Australia in 2005 was $21 billion, substantially higher than previous estimates.

References

1. NSW Health. NSW Healthy Eating and Active Living Strategy: preventing overweight and obesity in New South Wales 2013-2018. North Sydney: NSW Ministry of Health, 2013.

2. Hawkes C, Ahern A, Jebb S. A stakeholder analysis of the perceived outcomes of developing and implementing England's obesity strategy 2008-2011. BMC Public Health 2014; 14(1): 441.

3. Lobstein T, Brinsden H. Symposium report: the prevention of obesity and NCDs: challenges and opportunities for governments. Obesity Reviews 2014; 15(8): 630-9.

4. Sautkina E, Goodwin D, Jones A, et al. Lost in translation? Theory, policy and practice in systems-based environmental approaches to obesity prevention in the Healthy Towns programme in England. Health & Place 2014; 29(0): 60-6.

5. Butland B, Jebb S, Kopelman P, et al. Tackling obesities: future choices - project report (2nd edition). United Kingdom: Department of Innovation Universities and Skills, 2007.

6. Organisation for Economic Cooperation and Development. Obesity update, 2014.

7. Dixon JB. The effect of obesity on health outcomes. Mol. Cell. Endocrinol 2010; 316(2): 104-8.

8. Access Economics. The growing cost of obesity in 2008: three years on. Canberra: Diabetes Australia, 2008.

9. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta- analysis. JAMA 2013; 309(1): 71-82.

10. Kramer CK, Zinman B, Retnakaran R. Are metabolically healthy overweight and obesity benign conditions?: A systematic review and meta-analysis. Ann Intern Med 2013; 159(11): 758-69.

11. Guh D, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis A. The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. BMC Public Health 2009; 9(1): 88.

12. Bell JA, Kivimaki M, Hamer M. Metabolically healthy obesity and risk of incident type 2 diabetes: a meta-analysis of prospective cohort studies. Obes Rev 2014; 15(6): 504-15.

13. Zhou ZY, Liu YK, Chen HL, Liu F. Body mass index and knee osteoarthritis risk: A dose-response meta-analysis. Obesity 2014; 3(10): 20835.

14. Zhang Y, Liu H, Yang S, Zhang J, Qian L, Chen X. Overweight, obesity and endometrial cancer risk: results from a systematic review and meta-analysis. Int J Biol Markers 2014; 29(1): e21-9.

15. Wu S, Liu J, Wang X, Li M, Gan Y, Tang Y. Association of obesity and overweight with overall survival in colorectal cancer patients: a meta-analysis of 29 studies. Cancer Causes Control 2014; 29: 29.

16. Wang F, Xu Y. Body mass index and risk of renal cell cancer: a dose-response meta-analysis of published cohort studies. Int J Cancer 2014; 135(7): 1673-86.

17. Druesne-Pecollo N, Touvier M, Barrandon E, et al. Excess body weight and second primary cancer risk after breast cancer: a systematic review and meta-analysis of prospective studies. Breast Cancer Res Treat 2012; 135(3): 647-54.

18. Lin XJ, Wang CP, Liu XD, et al. Body Mass Index and Risk of Gastric Cancer: A Meta-analysis. Jpn J Clin Oncol 2014; 44(9): 783-91.

19. Qin Q, Xu X, Wang X, Zheng XY. Obesity and risk of bladder cancer: a meta-analysis of cohort studies. Asian Pac J Cancer Prev 2013; 14(5): 3117-21.

20. Golabek T, Bukowczan J, Chlosta P, Powroznik J, Dobruch J, Borowka A. Obesity and prostate cancer incidence and mortality: a systematic review of prospective cohort studies. Urol Int 2014; 92(1): 7-14.

21. Zhao ZG, Guo XG, Ba CX, et al. Overweight, obesity and thyroid cancer risk: a meta-analysis of cohort studies. J Int Med Res 2012; 40(6): 2041-50.

22. Shiri R, Lallukka T, Karppinen J, Viikari-Juntura E. Obesity as a risk factor for sciatica: a meta-analysis. AJE 2014; 179(8): 929-37.

23. Park M, Song da Y, Je Y, Lee JE. Body mass index and biliary tract disease: a systematic review and meta-analysis of prospective studies. Prev Med 2014; 65: 13-22.

24. Pan CW, Lin Y. Overweight, obesity, and age-related cataract: a meta-analysis. Optom Vis Sci 2014; 91(5): 478-83.

25. Meehan S, Beck CR, Mair-Jenkins J, Leonardi-Bee J, Puleston R. Maternal Obesity and Infant Mortality: A Meta-Analysis. Pediatrics 2014; 7: 7.

26. Yang Y, Dong J, Sun K, et al. Obesity and incidence of lung cancer: a meta-analysis. Int J Cancer 2013; 132(5): 1162-9.

27. Tang X, Liu G, Kang J, et al. Obesity and risk of hip fracture in adults: a meta-analysis of prospective cohort studies. PLoS One 2013; 8(4): e55077.

28. Withrow D, Alter DA. The economic burden of obesity worldwide: a systematic review of the direct costs of obesity. Obes Rev 2011; 12(2): 131-41.

29. Dee A, Kearns K, O'Neill C, et al. The direct and indirect costs of both overweight and obesity: a systematic review. BMC Res Notes 2014; 7(242): 242.

30. Lehnert T, Sonntag D, Konnopka A, Riedel-Heller S, Konig HH. Economic costs of overweight and obesity. Best Pract Res Clin Endocrinol Metab 2013; 27(2): 105-15.

31. Van Nuys K, Globe D, Ng-Mak D, Cheung H, Sullivan J, Goldman D. The association between employee obesity and employer costs: evidence from a panel of U.S. employers. Am J Health Promot 2014; 28(5): 277-85.

32. Kleinman N, Abouzaid S, Andersen L, Wang Z, Powers A. Cohort analysis assessing medical and nonmedical cost associated with obesity in the workplace. J Occup Environ Med /American College of Occupational and Environmental Medicine 2014; 56(2): 161-70.

33. Robroek SJ, Reeuwijk KG, Hillier FC, Bambra CL, van Rijn RM, Burdorf A. The contribution of overweight, obesity, and lack of physical activity to exit from paid employment: a meta-analysis. Scand J Work Environ Health 2013; 39(3): 233-40.

34. Trogdon JG, Finkelstein EA, Hylands T, Dellea PS, Kamal-Bahl SJ. Indirect costs of obesity: a review of the current literature. Obes Rev 2008; 9(5): 489-500.

35. Pulgaron ER. Childhood obesity: a review of increased risk for physical and psychological comorbidities. Clin Ther 2013; 35(1): A18-32.

36. Estrada E, Eneli I, Hampl S, et al. Children's Hospital Association Consensus Statements for Comorbidities of Childhood Obesity. Child Obes 2014; 10(4): 304-17.

37. van Geel M, Vedder P, Tanilon J. Are overweight and obese youths more often bullied by their peers? A meta-analysis on the relation between weight status and bullying. Int J Obes 2014;8(10): 117.

38. Friedemann C, Heneghan C, Mahtani K, Thompson M, Perera R, Ward AM. Cardiovascular disease risk in healthy children and its association with body mass index: systematic review and meta-analysis. Br Med J 2012; 25(345): e4759.

39. Hoare E, Skouteris H, Fuller-Tyszkiewicz M, Millar L, Allender S. Associations between obesogenic risk factors and depression among adolescents: a systematic review. Obes Rev 2014; 15(1): 40-51.

40. Bechard LJ, Rothpletz-Puglia P, Touger-Decker R, Duggan C, Mehta NM. Influence of obesity on clinical outcomes in hospitalized children: a systematic review. JAMA Pediatr 2013; 167(5):476-82.

41. Ahmed S, Arjmand E, Sidell D. Role of obesity in otitis media in children. Curr Allergy Asthma Rep 2014; 14(11): 469.

42. Bahadoran Z, Mirmiran P, Hosseini-Esfahani F, Azizi F. Fast food consumption and the risk of metabolic syndrome after 3-years of follow-up: Tehran Lipid and Glucose Study. Eur J Clin Nutr 2013; 67(12): 1303-9.

43. Odegaard AO, Koh WP, Yuan J-M, Gross MD, Pereira MA. Western-style fast food intake and cardiometabolic risk in an Eastern country. Circulation 2012; 126(2): 182-8.

44. Wang J, Luben R, Khaw K-T, Bingham S, Wareham NJ, Forouhi NG. Dietary energy density predicts the risk of incident type 2 diabetes: the European Prospective Investigation of Cancer (EPIC)-Norfolk Study. Diabetes Care 2008; 31(11): 2120-5.

45. Brock KE, Gridley G, Chiu BCH, Ershow AG, Lynch CF, Cantor KP. Dietary fat and risk of renal cell carcinoma in the USA: a case-control study. Br J Nutr 2009; 101(8): 1228-38.

46. Stott-Miller M, Neuhouser ML, Stanford JL. Consumption of deep-fried foods and risk of prostate cancer. Prostate 2013; 73(9): 960-9.

47. Morgenstern LB, Escobar JD, Sanchez BN, et al. Fast food and neighborhood stroke risk. Ann Neurol 2009; 66(2): 165-70.

48. Wang J, Zhang W, Sun L, et al. Dietary energy density is positively associated with risk of pancreatic cancer in urban Shanghai Chinese. J Nutr 2013; 143(10): 1626-9.

49. Wang X, Ouyang Y, Liu J, et al. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. Br Med J 2014; 349: g4490.

50. Muraki I, Imamura F, Manson JE, et al. Fruit consumption and risk of type 2 diabetes: results from three prospective longitudinal cohort studies. Br Med J 2013; 347: f5001.

51. Carter P, Gray LJ, Troughton J, Khunti K, Davies MJ. Fruit and vegetable intake and incidence of type 2 diabetes mellitus: systematic review and meta-analysis. Br Med J 2010; 341: c4229.

52. Wang Q, Chen Y, Wang X, Gong G, Li G, Li C. Consumption of fruit, but not vegetables, may reduce risk of gastric cancer: results from a meta-analysis of cohort studies. Eur J Cancer 2014;50(8): 1498-509.

53. Aune D, Lau R, Chan DSM, et al. Nonlinear reduction in risk for colorectal cancer by fruit and vegetable intake based on meta-analysis of prospective studies. Gastroenterology 2011;141(1): 106-18.

54. Aune D, Chan DSM, Vieira AR, et al. Fruits, vegetables and breast cancer risk: a systematic review and meta-analysis of prospective studies. Breast Cancer Res Treat 2012; 134(2): 479-93.

55. Jung S, Spiegelman D, Baglietto L, et al. Fruit and vegetable intake and risk of breast cancer by hormone receptor status. J Natl Cancer Inst 2013; 105(3): 219-36.

56. Pavia M, Pileggi C, Nobile CGA, Angelillo IF. Association between fruit and vegetable consumption and oral cancer: a meta-analysis of observational studies. Am J Clin Nutr 2006;83(5): 1126-34.

57. Wakai K, Matsuo K, Nagata C, et al. Lung cancer risk and consumption of vegetables and fruit: an evaluation based on a systematic review of epidemiological evidence from Japan. Jpn J Clin Oncol 2011; 41(5): 693-708.

58. Liu J, Wang J, Leng Y, Lv C. Intake of fruit and vegetables and risk of esophageal squamous cell carcinoma: a meta-analysis of observational studies. Int J Cancer 2013; 133(2): 473-85.

59. Bandera EV, Kushi LH, Moore DF, Gifkins DM, McCullough ML. Fruits and vegetables and endometrial cancer risk: a systematic literature review and meta-analysis. Nutr Cancer 2007; 58(1): 6-21.

60. Chen G-C, Lv D-B, Pang Z, Liu Q-F. Fruits and vegetables consumption and risk of non-Hodgkin's lymphoma: a meta-analysis of observational studies. Int J Cancer 2013; 133(1): 190-200.

61. Gundgaard J, Nielsen JN, Olsen J, Sørensen J. Increased intake of fruit and vegetables: estimation of impact in terms of life expectancy and healthcare costs†. Public Health Nutr 2003; 6(01): 25-30.

62. Swenson D. The economic impacts of increased fruit and vegetable production and consumption in Iowa: Phase II. United States: Leopold Center for Sustainable Agriculture, 2006.

63. Greenwood DC, Threapleton DE, Evans CEL, et al. Association between sugar-sweetened and artificially sweetened soft drinks and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. Br J Nutr 2014; 112(5): 725-34.

64. Huang C, Huang J, Tian Y, Yang X, Gu D. Sugar sweetened beverages consumption and risk of coronary heart disease: a meta-analysis of prospective studies. Atherosclerosis 2014; 234(1):11-6.

65. Genkinger JM, Li R, Spiegelman D, et al. Coffee, tea, and sugar-sweetened carbonated soft drink intake and pancreatic cancer risk: a pooled analysis of 14 cohort studies. Cancer Epidemiol Biomarkers Prev 2012; 21(2): 305-18.

66. Choi HK, Curhan G. Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. Br Med J 2008; 336(7639): 309-12.

67. Choi HK, Willett W, Curhan G. Fructose-rich beverages and risk of gout in women. JAMA 2010; 304(20): 2270-8.

68. Aidi HE, Bronkhorst EM, Huysmans MCDNJM, Truin G-J. Factors associated with the incidence of erosive wear in upper incisors and lower first molars: a multifactorial approach. J Dent 2011; 39(8): 558-63.

69. Cohen L, Curhan G, Forman J. Association of sweetened beverage intake with incident hypertension. J Gen Intern Med 2012; 27(9): 1127-34.

70. Ferraro PM, Taylor EN, Gambaro G, Curhan GC. Soda and other beverages and the risk of kidney stones. Clin J Am Soc Nephrol 2013; 8(8): 1389-95.

71. Larsson SC, Akesson A, Wolk A. Sweetened beverage consumption is associated with increased risk of stroke in women and men. J Nutr 2014; 144(6): 856-60.

72. Samitz G, Egger M, Zwahlen M. Domains of physical activity and all-cause mortality: systematic review and dose-response meta-analysis of cohort studies. Int J Epidemiol 2011; 40(5): 1382-400.

73. Li J, Siegrist J. Physical activity and risk of cardiovascular disease-a meta-analysis of prospective cohort studies. Int J Environ Res Public Health 2012; 9(2): 391-407.

74. Huai P, Xun H, Reilly KH, Wang Y, Ma W, Xi B. Physical activity and risk of hypertension: a meta-analysis of prospective cohort studies. Hypertension 2013; 62(6): 1021-6.

75. Keimling M, Behrens G, Schmid D, Jochem C, Leitzmann MF. The association between physical activity and bladder cancer: systematic review and meta-analysis. Br J Cancer 2014; 110(7): 1862-70.

76. Liu Y, Hu F, Li D, et al. Does physical activity reduce the risk of prostate cancer? A systematic review and meta-analysis. Eur Urol 2011; 60(5): 1029-44.

77. O'Rorke MA, Cantwell MM, Cardwell CR, Mulholland HG, Murray LJ. Can physical activity modulate pancreatic cancer risk? a systematic review and meta-analysis. Int J Cancer 2010; 126(12): 2957-68.

78. Katzmarzyk PT, Janssen I. The economic costs associated with physical inactivity and obesity in Canada: an update. Can J Appl Physiol = Revue canadienne de physiologie appliquee 2004; 29(1): 90-115.

79. Medibank Private. The cost of physical inactivity: what is the lack of participation in physical activity costing Australia? Medibank Private Ltd., 2007.

80. Nocon M, Hiemann T, Muller-Riemenschneider F, Thalau F, Roll S, Willich SN. Association of physical activity with all-cause and cardiovascular mortality: a systematic review and meta-analysis. Eur J Cardiovasc Prev Rehabil 2008; 15(3): 239-46.

81. Diep L, Kwagyan J, Kurantsin-Mills J, Weir R, Jayam-Trouth A. Association of physical activity level and stroke outcomes in men and women: a meta-analysis. J Womens Health (Larchmt) 2010; 19(10): 1815-22.

82. Sattelmair J, Pertman J, Ding EL, Kohl HW, 3rd, Haskell W, Lee IM. Dose response between physical activity and risk of coronary heart disease: a meta-analysis. Circulation 2011; 124(7): 789-95.

83. He D, Xi B, Xue J, Huai P, Zhang M, Li J. Association between leisure time physical activity and metabolic syndrome: a meta-analysis of prospective cohort studies. Endocrine 2014; 46(2): 231-40.

84. Jeon CY, Lokken RP, Hu FB, van Dam RM. Physical activity of moderate intensity and risk of type 2 diabetes: a systematic review. Diabetes Care 2007; 30(3): 744-52.

85. Eijkemans M, Mommers M, Draaisma JMT, Thijs C, Prins MH. Physical activity and asthma: a systematic review and meta-analysis. PLoS ONE [Electronic Resource] 2012; 7(12): e50775.

86. Moayyeri A. The association between physical activity and osteoporotic fractures: a review of the evidence and implications for future research. Ann Epidemiol 2008; 18(11): 827-35.

87. Blondell SJ, Hammersley-Mather R, Veerman JL. Does physical activity prevent cognitive decline and dementia?: A systematic review and meta-analysis of longitudinal studies. BMC Public Health 2014; 14: 510.

88. Sun J-Y, Shi L, Gao X-D, Xu S-F. Physical activity and risk of lung cancer: a meta-analysis of prospective cohort studies. Asian Pac J Cancer Prev 2012; 13(7): 3143-7.

89. Lagerros YT, Hsieh SF, Hsieh CC. Physical activity in adolescence and young adulthood and breast cancer risk: a quantitative review. Eur J Cancer Prev 2004; 13(1): 5-12.

90. Boyle T, Keegel T, Bull F, Heyworth J, Fritschi L. Physical activity and risks of proximal and distal colon cancers: a systematic review and meta-analysis. J Natl Cancer Inst 2012; 104(20): 1548-

91. Behrens G, Jochem C, Keimling M, Ricci C, Schmid D, Leitzmann MF. The association between physical activity and gastroesophageal cancer: systematic review and meta-analysis. Eur J Epidemiol 2014; 29(3): 151-70.

92. Behrens G, Leitzmann MF. The association between physical activity and renal cancer: systematic review and meta-analysis. Br J Cancer 2013; 108(4): 798-811.

93. Keum N, Ju W, Lee DH, et al. Leisure-time physical activity and endometrial cancer risk: dose-response meta-analysis of epidemiological studies. Int J Cancer 2014; 135(3): 682-94.

94. Aune D, Saugstad OD, Henriksen T, Tonstad S. Physical activity and the risk of preeclampsia: a systematic review and meta-analysis. Epidemiology 2014; 25(3): 331-43.

95. Tobias DK, Zhang C, van Dam RM, Bowers K, Hu FB. Physical activity before and during pregnancy and risk of gestational diabetes mellitus: a meta-analysis. Diabetes Care 2011;34(1): 223-9.

96. Zhou L-M. Recreational Physical Activity and Risk of Ovarian Cancer: a Meta-analysis. Asian Pac J Cancer Prev 2014; 15(13): 5161-6.

97. Mammen G, Faulkner G. Physical activity and the prevention of depression: a systematic review of prospective studies. Am J Prev Med 2013; 45(5): 649-57.

98. Strohle A. Physical activity, exercise, depression and anxiety disorders. J Neural Transm 2009; 116(6): 777-84.

99. Cheng JYW, Ng EML, Ko JSN, Chen RYL. Physical activity and erectile dysfunction: meta-analysis of population-based studies. Int J Impot Res 2007; 19(3): 245-52.

100. Stephenson J, Bauman A, Armstrong T, Smith B, Bellew B. The costs of illness attributable to physical inactivity in Australia: a preliminary study. Canberra: Department of Health and Ageing, 2000.

101. Cadilhac DA, Cumming TB, Sheppard L, Pearce DC, Carter R, Magnus A. The economic benefits of reducing physical inactivity: an Australian example. Int J Behav Nutr Phys Act 2011; 8: 99.

102. Eriksen W, Bruusgaard D. Physical leisure-time activities and long-term sick leave: a 15-month prospective study of nurses' aides. J Occup Env Med /American College of Occupational and Environmental Medicine 2002; 44(6): 530-8.

103. Waller K, Kujala UM, Kaprio J, Koskenvuo M, Rantanen T. Effect of physical activity on health in

twins: a 30-yr longitudinal study. Med Sci Sports Exerc 2010; 42(4): 658-64.

104. Wang F, Orpana HM, Morrison H, de Groh M, Dai S, Luo W. Long-term association between leisure-time physical activity and changes in happiness: analysis of the Prospective National Population Health Survey. AJE 2012; 176(12): 1095-100.

105. Bize R, Johnson JA, Plotnikoff RC. Physical activity level and health-related quality of life in the general adult population: a systematic review. Prev Med 2007; 45(6): 401-15.

106. Tsunoda K, Kai Y, Kitano N, et al. Influence of physical activity on sleep duration and quality: A prospective cohort study. Bulletin of the Physical Fitness Research Institute 2014; 112: 10.

107. Spence JC, McGannon KR, Poon P. The effect of exercise on global self-esteem: a quantitative review. J Sport Exerc Psychol 2005; 27(3): 24.

108. Campbell A, Hausenblas HA. Effects of exercise interventions on body image: a meta-analysis. J Health Psychol 2009; 14(6): 780-93.

109. Okely AD, Salmon J, Vella SA, et al. A Systematic Review to update the Australian Physical Activity Guidelines for Children and Young People. Report prepared for the Australian Government Department of Health. Canberra: Commonwealth of Australia, 2012.

110. Janssen I, Leblanc AG. Systematic review of the health benefits of physical activity and fitness in school-aged children and youth. Int J Behav Nutr Phys Act 2010; 7: 40.

111. Ford ES, Caspersen CJ. Sedentary behaviour and cardiovascular disease: a review of prospective studies. Int J Epidemiol. 2012; 41(5): 1338-53.

112. Pedišić Ž. Measurement issues and poor adjustments for physical activity and sleep undermine sedentary behaviour research: the focus should shift to the balance between sleep, sedentary behaviour, standing and activity. Kinesiology 2014; 46(1): 12.

113. Wilmot EG, Edwardson CL, Achana FA, et al. Sedentary time in adults and the association with diabetes, cardiovascular disease and death: systematic review and meta-analysis. Diabetologia 2012; 55(11): 2895-905.

114. Grontved A, Hu FB. Television viewing and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a meta-analysis. JAMA 2011; 305(23): 2448-55.

115. Chau JY, Grunseit AC, Chey T, et al. Daily sitting time and all-cause mortality: a meta-analysis. PLoS ONE [Electronic

Resource] 2013; 8(11): e80000.

116. Edwardson CL, Gorely T, Davies MJ, et al. Association of sedentary behaviour with metabolic syndrome: a meta-analysis. PLoS ONE 2012; 7(4): e34916.

117. Cong YJ, Gan Y, Sun HL, et al. Association of sedentary behaviour with colon and rectal cancer: a meta-analysis of observational studies. Br J Cancer 2014; 110(3): 817-26.

118. Schmid D, Leitzmann MF. Television viewing and time spent sedentary in relation to cancer risk: a meta-analysis. J Natl Cancer Inst 2014; 106(7).

119. Rezende LFMd, Rodrigues Lopes M, Rey-López JP, Matsudo VKR, Luiz OdC. Sedentary Behavior and Health Outcomes: An Overview of Systematic Reviews. PLoS ONE 2014; 9(8): e105620.

120. Green LW, McAlister AL. Macro-intervention to support health behavior: some theoretical perspectives and practical reflections. Health Educ Q 1984; 11(3): 322-39.

121. Global Advocacy Council for Physical Activity and International Society for Physical Activity and Health. The Toronto Charter for Physical Activity: A Global Call for Action. J Phys Act Health 2010; 7 Suppl 3: S370-85.

122. Cancer Institute NSW. Lifestyle and cancer: knowledge, attitudes and behaviour in NSW. Sydney: Cancer Institute NSW, 2009.

123. King E, Grunseit A, O'Hara B, Bauman A. Evaluating the effectiveness of an Australian obesity mass-media campaign: how did the 'Measure-Up' campaign measure up in New South Wales? Health Educ Res 2013: 1029-39.

124. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organization technical report series 2000; 894: i-xii, 1-253.

125. Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez A. The burden of disease and injury in Australia 2003. Cat. no. PHE 82. Canberra, Australia: Australian Institute of Health and Welfare, 2007.

126. Department of Health and Ageing. National Physical Activity Guidelines for Australia. Canberra: Commonwealth of Australia, 1999.

127. Centre for Epidemiology and Evidence. Health statistics New South Wales. 2014. http://www.healthstats.nsw.gov.au (accessed 4 September 2014).

128. Johnson CM, Wei C, Ensor JE, et al. Meta-analyses of colorectal cancer risk factors. Cancer Causes Control 2013; 24(6): 1207-22.

129. Cancer Institute NSW. Cancer in NSW: incidence report 2009. Sydney: Cancer Institute NSW, 2009.

130. Department of Health and Ageing. Dietary guidelines for Australians: a guide to healthy eating. Canberra: Commonwealth of Australia, 2005.

131. He FJ, Nowson CA, Lucas M, MacGregor GA. Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. J Hum Hypertens 2007; 21(9): 717-28.

132. Australian Institute of Health and Welfare. Trends in cardiovascular disease. 2010. http://www.aihw.gov.au/cardiovascular-health/trends/ - t1 (accessed 3 September 2014).

133. Australian Bureau of Statistics. Australian Health Survey: Nutrition First Results - Food and Nutrients, 2011-12: Commonwealth of Australia, 2014.

134. Australian Institute of Health and Welfare. Incidence of insulin-treated diabetes in Australia 2000-2009. 2012. http://www.aihw.gov.au/diabetes/incidence/ - t4 (accessed 3 September 2014).

135. Australian Bureau of Statistics. Deaths, Australia, 2012. 2013. http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/3302.0Main+Features12012?OpenDocument (accessed 4 September 2014).

136. Stott-Miller M, Neuhouser ML, Stanford JL. Consumption of deep-fried foods and risk of prostate cancer. The Prostate 2013; 73(9): 960-9

137. He FJ, Nowson CA, MacGregor GA. Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. Lancet 2006; 367(9507): 320-6.

138. Xi B, Li S, Liu Z, et al. Intake of fruit juice and incidence of type 2 diabetes: a systematic review and meta-analysis. PLoS ONE 2014; 9(3): e93471.

139. Goncalves AK, Dantas Florencio GL, de Atayde Silva MJM, Cobucci RN, Giraldo PC, Cote NM. Effects of physical activity on breast cancer prevention: A systematic review. J Phys Act Health 2014; 11(2): 445-54.

140. Hamer M, Chida Y. Walking and primary prevention: a meta-analysis of prospective cohort studies. BJSM online 2008; 42(4): 238-43.

141. Wilmot EG, Edwardson CL, Achana FA, et al. Sedentary time in adults and the association with diabetes, cardiovascular disease and death: systematic review and meta-analysis. Diabetologia 2012; 55(11): 2895-905.

142. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. The Lancet; 380(9838): 219-29.

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