Curr Obes Rep (2015) 4:1–10
DOI 10.1007/s13679-014-0134-7

The Influence on Population Weight Gain and Obesity of the Macronutrient Composition and Energy Density of the Food Supply

Michelle Crino · Gary Sacks · Stefanie Vandevijvere · Boyd Swinburn · Bruce Neal

Published online: 14 January 2015

Abstract Rates of overweight and obesity have increased dramatically in all regions of the world over the last few decades. Almost all of the world's population now has ubiquitous access to low-cost, but highly-processed, energy-dense, nutrient-poor food products. These changes in the food supply, rather than decreases in physical activity, are most likely the primary driver of population weight gain and obesity. To-date, the majority of prevention efforts focus on personalised approaches targeting individuals. Population-wide food supply interventions addressing sodium and *trans* fat reduction have proven highly effective and comparable efforts are now required to target obesity. The evidence suggests that strategies focusing upon reducing the energy density and portion size of foods will be more effective than those targeting specific macronutrients. Government leadership, clearly specified targets, accountability and transparency will be the key to achieving the food supply changes required to address the global obesity epidemic.

Keywords Obesity · Nutrition transition · Macronutrient composition · Energy density · Food supply · Ultra-processed food products

This article is part of the Topical Collection on *Etiology of Obesity*

M. Crino (✉) · B. Neal
The George Institute for Global Health, University of Sydney, Level 10, King George V Building, 83-117 Missenden Rd, Camperdown, NSW 2050 Sydney, Australia
e-mail: mcrino@georgeinstitute.org.au

B. Neal
e-mail: bneal@georgeinstitute.org.au

G. Sacks · B. Swinburn
WHO Collaborating Centre for Obesity Prevention, Deakin University, 221 Burwood Highway, Burwood, VIC 3125, Australia

G. Sacks
e-mail: gary.sacks@deakin.edu.au

B. Swinburn
e-mail: boyd.swinburn@auckland.ac.nz

S. Vandevijvere · B. Swinburn
School of Population Health, University of Auckland, Private Bag 92019, Auckland Mail Centre, Auckland 1142, New Zealand

S. Vandevijvere
e-mail: s.vandevijvere@auckland.ac.nz

B. Neal
The Royal Prince Alfred Hospital, Sydney, Australia

B. Neal
Imperial College, London, UK

Introduction

The prevalence of overweight and obesity has dramatically increased across the world from approximately 29 % of adults being overweight or obese in 1980 (857 million individuals) to 38 % (2.1 billion individuals) in 2013 [1•]. Being overweight or obese predisposes affected individuals to a plethora of premature health risks caused both by the direct effects of overweight on conditions such as arthritis and low back pain [2] and indirectly through a series of associated metabolic abnormalities [3]. The effects of overweight and obesity on high blood pressure, dyslipidaemia and diabetes are particularly important because these are the leading causes of cardiovascular diseases, which in turn are now the leading causes of death in most countries around the world [1•, 4, 5]. As a consequence, raised body mass index (BMI; BMI>25 kg/m^2) due to excess weight is now a leading risk factor for death worldwide [5]. It was estimated that in 2010, overweight and obesity caused 3.4 million deaths globally (7 % of total deaths that year), 4 % of all years of life lost and 4 % of all disability adjusted life years (DALYS) [1•, 6].

The global burden of ill health attributable to poor diet has always been high, but for most of human history the primary diet-related health issues have been those caused by micronutrient deficiencies and the consumption of insufficient calories [7, 8]. Until about 50 years ago poor health caused by overweight and obesity was the preserve of a wealthy few [8, 9]. Now, however, the adverse effects of "over-nutrition" far exceed the problems caused by "under-nutrition" and create a double burden in low- and middle-income countries [10]. This 'nutrition transition' [8] parallels profound economic, demographic and epidemiologic changes that have by now affected most high- and low-income regions across the world [7, 9, 11]. The predominant dietary shift has been away from traditional foods prepared within the community towards ultra-processed and packaged food products, manufactured and heavily marketed by large national, multi-national, and trans-national corporations [7, 9]. Very widely marketed ultra-processed food products are typically formulated from refined ingredients, which have been extracted from raw materials, and transformed into imitations of their original constituents [12, 13]. These types of food products are often energy dense and nutrient poor [12, 13].

The nutrition transition has not only modified the average composition of food products but has also changed the entire food environment including all aspects of marketing, price, food availability and accessibility [14]. In addition, the globalization of the food supply has enabled overseas entities to significantly adversely affect the healthiness of domestic food environments by increasing the availability of ultra-processed foods [11, 15, 16].

Energy In and Energy Out

At the most basic level, weight gain and obesity are problems of energy balance — if energy intake exceeds energy expenditure, then weight will accrue [17••, 18, 19]. It follows that if either energy intake can be reduced or physical activity increased, weight gain and obesity should attenuate [17••, 18–20]. As well as changes in the food supply, the broad societal changes of the last 50 years have also had effects on the physical activity levels of much of the world's population, both while at work and during transport and leisure time [21–23]. What has remained unclear for some time has been the relative contribution of each to the world's obesity epidemic.

A recent report using small area models to analyse data from the United States has provided further insight into the comparative effects of the two sides of energy balance. The primary observation was that, while the analyses identified small average *increases* in moderate to vigorous physical activity over the decade of study, there were much larger *increases* in average levels of obesity [17••]. This was despite there being the anticipated weak inverse associations of physical activity with BMI [17••]. While somewhat counter-intuitive, the findings are directly in line with prior work addressing the likely contribution of changes in physical activity, compared to changes in the food supply, to the evolution of the obesity epidemic [14, 18]. A broad range of analyses using diverse sources of data have now identified energy intake as the major driver with a key observation being that over the past four decades physical activity has remained generally stable whereas energy intake has gradually increased [18].

Thus, while physical activity is to be encouraged for the health benefits that it will produce, even if it is not accompanied by weight loss [5, 24, 25], interventions targeting physical activity alone are unlikely to be sufficient to address the global obesity epidemic [14, 18–20]. The focus needs instead to be on the food supply factors [14].

The Food Supply, Weight Gain and Obesity

The types of food consumed impact upon obesity, health and well-being through a range of different mechanisms [5, 9, 18, 26••, 27]. Many studies have assessed the effects of the intake levels of different dietary components on both weight loss and weight gain. The most robust of those studies are randomised controlled trials examining the impact of different types of diets on weight loss at the individual-level, [14, 18, 26••, 27, 28] typically over relatively short periods of time (6 months to 2 years) [14, 18, 19, 26••, 28, 29]. While highly informative in regard to the specific questions addressed, these studies are of more limited value in understanding the population-level determinants of weight gain and obesity [14].

Far fewer studies have addressed the food composition determinants of population weight gain and obesity [14, 30]. Most of these studies are non-randomised in design and require a more cautious interpretation because of the risk of bias and confounding inherent in the study designs [14, 26••]. Moreover, there is a lack of definitive data to describe the systems-level factors causing obesity at the population level. A key factor behind this is the difficulty of obtaining up-to-date, robust and reliable food consumption data [31–33]. At present food balance sheets, household budget and expenditure surveys, food consumption surveys, and supermarket sales data are the most commonly used approaches to source population dietary consumption data [31]. Each of these approaches has strengths and limitations [31], but the majority tends to fall short of providing a complete and accurate picture of the diet of populations [14, 26••, 33]. Mis-reporting of consumption patterns, incomplete food survey data and the absence of good data about real world *ad-libitum* food intake patterns represent significant challenges [14, 26••, 33]. Where population dietary consumption studies do exist, they often classify dietary patterns rather broadly, [26••] making both the combination and comparison of data across studies difficult.

Nevertheless, the totality of the available evidence shows that high-level characteristics of the food supply have a profound influence on population levels of overweight and obesity and their evolution over time [9, 26••, 33–35]. The challenge is identifying the characteristics of the food supply that are both important determinants of obesity [9, 13, 36, 37] and also amenable to change.

Developments in Food Processing

For most of the evolution of hominids the food supply contained no processed items and foodstuffs were consumed in a natural or almost natural state [9, 38]. Technological development commenced only a few thousand years ago with basic food storage techniques such as salting, curing and drying [7, 38]. In the last century, these technological advancements have developed quickly and have culminated in refrigeration, freezing and complex manufacturing methods that transform foods and deliver ultra-processed products on an unprecedented scale [36, 38, 39]. These changes to the food supply have increased safety, shelf-life and the availability of seasonal foods, and have also reduced food wastage due to less food spoilage [7–9, 38, 40]. The most rapid advancements were made in the mid- to late-twentieth century which saw a huge expansion in both the technologies available and the capacity for food processing [38]. For example, a series of novel extraction methods enabled the low cost preparation of oils from the seeds of plants and grains greatly increasing the quantities of cheap vegetable oils in the food supply [37, 38, 40]. In the 30 years from 1960, availability of vegetable oils tripled [9]. Production of high-fructose corn syrup (HFCS) and other sweeteners also rose [40] with data from the Food and Agriculture Organisation (FAO) recording increases of 30 % in per capita global supplies of sugar and sweeteners between 1961 and 2003 [37]. Currently HFCS represents 40 % of caloric sweetener used in the US [41]. In parallel, an increased focus on pre-prepared foods led to developments in refining, milling and other food transforming technologies. Unfortunately, increased processing was almost always associated with falls in fibre content [9] and the addition of large quantities of fat, simple sugars and salt [37, 40]. The increasing worldwide prevalence of these commodities in the food supply is a direct consequence of their profitability, with added salt, fat and sugar allowing for low production costs, long shelf-life, hyper-palatability and sustained high retail volumes [37, 39].

Accompanying changes in the extent of food processing have been profound advancements in the distribution systems supporting the food supply. Fresh or 'wet' markets stocked with unprocessed or minimally processed foods [7, 37, 39, 40] were once the primary source of foodstuffs for the population [13]. While perishable meats, dairy products and fresh produce can still be sourced in most areas of the world, the long shelf-life and stability of many processed foods has provided for their worldwide distribution through global transport networks [11, 39, 42]. Economies of scale, whereby ultra-processed foods can be produced in regions with a low cost base [42] and cheaply transferred to global markets has supported massive increases in the sales of these commodities and, in many cases, the replacements of local wet markets by large supermarkets [7, 39, 42]. In high-income countries, and increasingly in low-middle income countries, the food supply has become progressively more homogenous and comprised to an increasingly large extent of ultra-processed products [7, 13, 37].

Processed Food Products and Average Dietary Consumption

The increased availability of hyper-palatable ultra-processed foods has impacted adversely upon dietary consumption patterns in most countries in just a few decades [7, 37, 40, 43]. The extent to which ultra-processed foods have dominated markets is highlighted by a recent study from Canada which reported that 62 % of energy consumed by Canadian households is derived from ultra-processed food products [36], compared to 29 % in 1938 [44]. Large increases in the availability and consumption of ultra-processed food products have also been reported in low- and middle-income countries [11, 31, 45–47] with Brazil reporting that even by 2003 ultra-processed foods already constituted more than one-quarter of total energy intake [13].

The developments in food processing that have driven the nutrition transition have mostly impacted upon dietary consumption in ways that conflict with World Health Organization (WHO) objectives [48] (Fig. 1). In general, nutrition transition has been characterised by increased consumption of sugars, fats and salt, and while average population intake levels appear to be plateauing in some higher-income countries [11], most lower- and middle-income regions for which data are available continue to report substantial increases in the consumption of these nutrients [10, 11]. Global estimates also indicate that average energy supply has increased from 2250 calories per person per day in 1961 to 2750 calories per person per day in 2007 [49]. In the United States, data from the National Health and Nutrition Examination Survey (NHANES) show parallel increases in the percentage of energy from carbohydrate sources over the last 30 years [43] — a change driven by increased consumption of ultra-processed foods which typically contain less than half the dietary fibre content and six times the amount of free sugars as un- or minimally-processed foods. Many also contain significant quantities of salt, saturated fat and *trans* fats [36]. That same

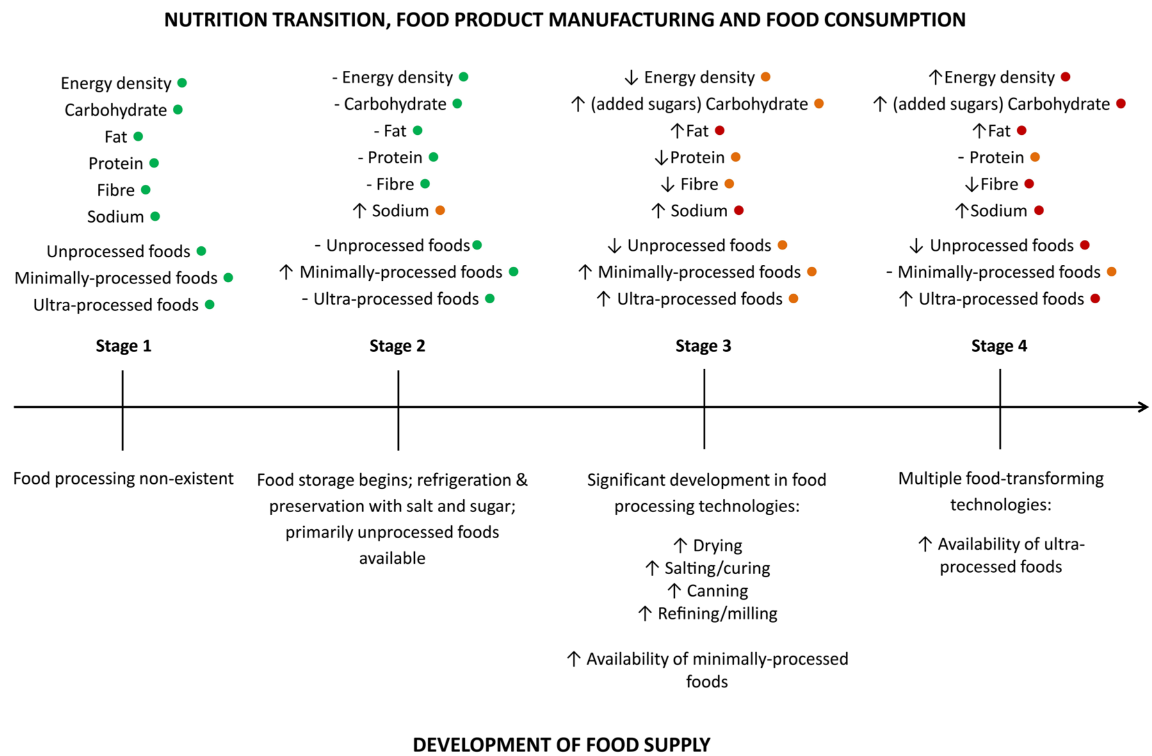

Fig. 1 Nutrition transition: trends in food consumption and the development of the food supply (● good, ● moderate, ● poor, consumption). Stage 1: Palaeolithic trend; stage 2: modern agriculture and famine trend; stage 3: receding famine trend; stage 4: degenerative diseases. For energy density, macronutrients and sodium, dietary consumption was classified as good if the reported consumption fell within the low- to mid-range of current WHO dietary intake targets. Consumption rates approaching mid- to high-range of current WHO dietary intake targets were classified as moderate consumption; classification of poor consumption was given when rates exceeded the WHO targets. For fibre consumption, good, moderate and poor consumption was classified as mid- to high-range, low- to mid-range and inadequate (below) WHO targets, respectively. An increase in consumption or processing technique is indicated by "↑", whilst a decrease is indicated by "↓". No change is indicated by "-". A rating of good, moderate or poor consumption of unprocessed, minimally-processed and ultra-processed foods was determined by the percentage of energy (calories/kilojoules) each food group contributed to total energy, with reference to WHO dietary intake targets. Content adapted from Austin et al. (2011), Kac and Perez-Escamilla (2013), Kearny (2010), Popkin (2006)

study also reported recent levels of energy intake in the United States derived from carbohydrate (49 %), fat (34 %) and protein (16 %) [43]. The 2011-2012 Australian Health Survey reported similar findings for carbohydrate (45 %), fat (31 %) and protein (18 %) with a large proportion of energy derived from total sugars (20 %) and saturated fats (12 %), and only low levels from fibre (2.2 %) [50]. WHO [48] recommends that daily energy intake is derived primarily from carbohydrates (55-75 %) with less than 10 % of energy from free sugars (with a current proposal to reduce this to 5 % [51]) and at least 11.1 g of fibre for every 4148 kJ of energy consumed. For fat, the WHO recommendation is 15-30 % of energy with <10 % from saturated fats, and 10-15 % of energy from protein [36, 48].

Macronutrients, Energy Density and Obesity

There is strong evidence describing the role of diet composition in weight change, but it is apparent from multiple intervention studies that the achievement of weight loss is dependent primarily upon energy reduction [19, 52, 53]. From the available data it appears that "a calorie is a calorie" regardless of its macronutrient source [19, 54] and there is a physiological support for this proposition [19]. However, while there are many studies defining the effects of short-to-medium-term dietary manipulation, rather little is known about effects beyond 2 years. And there are data to suggest that changing the macronutrient composition of diet could play a role in weight gain and obesity over the longer-term [19].

Carbohydrates — in the context of processed and packaged food composition, carbohydrates can be broadly separated into sugars and fibre. Multiple studies have identified a decrease in the average fibre content of the food supply with corresponding reports of reduced dietary fibre consumption in population studies [7, 9, 36]. It has been known for many years that diets high in fibre protect against weight gain, may induce weight loss [33, 34, 55, 56] and are associated with a decreased energy intake [33, 56]. These effects are, in part, attributed to the induction of the satiety response by which fibre reduces hunger [56]. Sugars have been particularly strongly implicated in weight gain [26••, 33, 57–61] with a recent meta-analysis of randomised control trials and cohort studies reporting adverse effects in both children and adults [62]. This finding is supported by data from randomised trials comparing the intake of sugar sweetened soda to sugar-free aspartame sweetened sodas [33, 63] which showed a reduced caloric intake and prevention of weight gain [63]. Observational studies of the association of artificial sweeteners with health outcomes have reported varied findings [55, 63, 64] as have studies examining the link between sugar intake and BMI [33, 59, 60]. In both cases, difficulties in measuring dietary consumption patterns [31] and changes in consumption patterns secondary to weight gain and poor health are likely to have caused serious and uncontrolled confounding of the true associations [14, 26••].

Fats — as for sugars, observational studies examining the influence of dietary fat on weight gain and obesity are mixed in their findings [33, 55, 65–67], and once again, this likely reflects the problem of confounding [26••, 33]. Several studies have suggested that diets with a high proportion of energy derived from fat promote weight gain and this is likely because of the high energy density of fat compared to carbohydrates and protein [26••, 33, 65]. Like sugars, fat also has a lower satiating effect and this may encourage over-consumption of foods with a high fat contribution [65]. However, while fat is more energy dense, there is no evidence to suggest that energy derived from fat is more fattening than the same amount of energy obtained from carbohydrate or protein sources [26••, 33, 34, 67].

Protein — the protein content of the diet has not been consistently associated with weight gain or weight loss and is not currently considered an important determinant of the prevalence of obesity in a population [26••, 33, 55]. However, protein does increase the sensation of satiety [68] and there is some evidence that individuals that consume a higher proportion of protein develop the sensation of fullness earlier and eat smaller portion sizes than those consuming a higher proportion of carbohydrates or fats [26••, 28, 34, 55, 69].

Alcohol — is recognised as a high energy-yielding macronutrient [26••, 70–74] and there is evidence that markedly elevated alcohol consumption has a role in weight gain [74–76]. Alcohol is not, however, typically considered to be a constituent of the food supply that importantly drives population weight change or obesity because it is rarely found in processed and packaged food products, which constitute the majority of people's diets [33, 36, 40].

Energy density — excessive caloric intake is the most easily understood determinant of the obesity epidemic, and is a product of the energy density of food and the quantity of food consumed [24, 69, 77]. The consumption of food with a higher average energy density is consistently associated with increased weight [24, 26••]. The propensity to over-consume calories because a food has a high energy density is compounded by the high levels of fats and sugars in many ultra-processed products which make them hyper-palatable and non-satiating, thereby encouraging consumption beyond energy needs [37, 40, 42, 77–82]. Most of the literature investigating the role of energy density in the diet had a focus on weight change at the individual level. These studies have found that diets with a higher energy density were not only associated with weight gain and obesity, but were also associated with unhealthy eating patterns including excess quantities of sugars and fat [83].

Portion size — like energy density, the portion size of processed foods is now a widely accepted determinant of weight gain and obesity. Several studies have shown that portion sizes, especially of energy dense foods, have increased enormously in recent decades [84–88]. Variation in weight and obesity has been observed to be directly associated with average portion size [87, 89–91]. While portion size and energy density are both correlated with energy intake [91], the association between portion size and energy density is weak [89, 91]. The reason for this is that the addition of water or air (neither of which have any caloric value) to a food product can profoundly change the mass, volume and portion size of the product [91]. Nevertheless, the combination of foods that are highly energy dense with large portion sizes provides for a powerful effect on weight gain and obesity [87, 89, 91]. The corollary is also true whereby the conjoint reduction of energy density and portion size is likely to deliver large reductions in total energy intake [89, 91, 92]. Recent interventions studies targeting portion size have demonstrated that changes in the food supply can lead to increased consumer awareness, and subsequent empowerment to make healthier choices [93, 94]. The most effective of these interventions included proportional pricing (standard price per unit, regardless of portion size of food) and the offering of a larger variety of portion sizes, especially in quick service restaurant and workplace settings [94].

Carbohydrates (4 kcal/g), fats (9 kcal/g) and protein (4 kcal/g) [26••, 28, 34] contain quite different quantities of energy but the effects on weight of manipulating macronutrients and energy density showed stronger effects for energy density than for any single macronutrient [26••, 27, 28]. While these findings are not entirely new [42, 65, 68, 82, 95, 96] the breadth of the evidence now supporting energy density as the primary determinant of weight gain identifies it as focus for amelioration efforts [26••, 27, 81]. Targeting energy density might also address some of the perversities that arise when weight control programs focus on one macronutrient or another. For example, NHANES data indicates that consensus efforts to lower dietary fat were observed to produce the planned decreases in the percentage of energy consumed from fat but were associated with increased total energy intake due to a compensatory over-consumption of energy from sugars [43].

Key Actions to Create a Healthier Food Supply

Population-level interventions to improve the nutrient composition of the food supply are an essential part of efforts to address the global burden of obesity and diet-related non-communicable diseases (NCDs) [4, 5, 48, 97, 98]. The global NCD action plan adopted by WHO member states in May 2013 includes a target to reduce salt intake of populations to less than 5 g a day, and an indicator to reduce *trans* and saturated fats in the food supply [98]. The WHO did recently review their recommendation for sugar intake [51, 98] but targets for population sugar consumption are not currently included in the NCD framework and there are few examples of system level efforts to modify the quantity of sugar in the food supply. Likewise, excessive serving sizes have been identified as an important determinant of obesity but there has been no systematic attempt to address this problem by any country to date.

Perhaps reflecting WHO priorities, the focus of efforts to change the food supply have been on sodium levels, with 22 countries reporting voluntary sodium reformulation targets and seven countries having mandatory sodium limits for selected food products [99]. These approaches have been highly successful in the UK and Finland [100–103] though less so in other countries [12, 104]. The UK salt reduction strategy is voluntary, but the overt threat of legislation if the program failed has driven reductions in the average sodium content of most processed foods. This, in turn, has reduced average population sodium consumption by 15 % over 7 years with thousands of premature deaths averted each year as a direct consequence [12, 103]. Reduction of *trans* fats has been the other area of activity targeting the food supply with several countries banning *trans* fats in food products [99]. For both salt and *trans* fat there has been strong uptake in a few settings but quality evidence describing the effectiveness of reformulation strategies is mostly absent and the optimal approaches to program implementation are substantially uncertain [12].

Beyond sodium and *trans* fats there are few food supply-based interventions. Whilst reductions in salt intake and *trans* fat consumption will improve population distributions of blood pressure and lipid levels, neither is likely to have a substantive impact on overweight or obesity. Australia, France, the Netherlands, New Zealand, Switzerland and the UK also have voluntary reformulation targets in place for saturated fats but impact is unknown [99] and effects on obesity are likely to be small. The UK, as a part of the Responsibility Deal, also has industry commitments to reduce calories and portion sizes [99, 105] but the programs are new and objective evaluation has not been completed. Objective data

Table 1 Examples of reformulation options and potential positives and negatives from a public health perspective based on Monteiro (2009) [38], Stubbs, Ferres (2000) [68], and National Heart Foundation of Australia (2012) [12]

Reformulation options	Potential positives	Potential negatives
Development of low-fat or reduced-fat products	Energy density remains the same or slightly decreased	Increased sugar content
Use of fat and carbohydrate substitutes	Decreases overall energy density	Technological difficulties for industry, namely stability and shelf-life of food Unknown long-term effects of substitutes on health
Substitution of sugar with artificial sweeteners	Decreased sugar content and overall energy density	Unknown long-term effects of consumption of artificial sweeteners on health Potential harm to dental health due to ongoing high levels of carbonated beverage consumption (high in acids)
Increased water/air content of food products	Increases weight/volume of food product without adding extra calories	Alters portion size of product, energy density perceived lower than it actually is
Increasing fibre content of food	Decreases overall energy density	Very high fibre consumption can interfere with absorption of certain nutrients
Changes in standard portion size	Changing the portion size of a product can both increase and decrease the energy density of a food product	Portion size of food products can be altered (primarily increased) to be perceived as healthier options to consumers
Reduction of sodium in foods	Reduced population salt consumption through slightly healthier food supply	Addition of fat or sugar to increase palatability of food Technological difficulties for industry, namely stability and shelf-life of food Unknown long-term effects of substitutes on health

describing the capacity of this program to effect change on average population energy intake and consequent impacts upon overweight and obesity will be of enormous interest.

Like the Responsibility Deal, the great majority of interventions targeting the food supply are voluntary in nature [99] despite evidence suggesting that legislative approaches are likely to produce better outcomes [12, 106–109]. For example, modelling of mandatory vs. voluntary sodium reformulation in Australia suggested health gains 20 times greater for the former [106]. The potential for mandatory interventions is highlighted by Mauritius where enforced replacement of palm oil with soybean oil [12] resulted in a 3.5 % decrease in energy intake from saturated fat over just a few years. Food supply reformulation programs do, however, need to be implemented with care because unintended negative effects can ensue (Table 1). The explosion of low-fat food products since the 1980s [40] has reduced levels of fat in the food supply [40] but only at the expense of greatly increased quantities of sugars [37]. The food industry is expert at using combinations of fat, sugar and sodium to make food products hyper-palatable and enticing to the consumer [110–112] but only with a comprehensive oversight of the food supply will health gains be assured.

It seems highly likely that regulatory actions will be required to address the obesity epidemic but actions on even voluntary approaches are still in their infancy. This may be because reformulation of the energy composition of foods is genuinely more complex, or a consequence of the focus of obesity intervention on interventions targeting personal responsibility rather than the food environment. Environmental approaches focused upon nutrient reformulation (specifically energy density), pricing by portion size, improved food labelling, taxes on junk foods/unhealthy food items and restricted marketing of such products are keys actions likely to discourage overconsumption of nutrient-poor, energy dense, ultra-processed food products and encourage consumers to make healthier choices.

Conclusions and Implications

The global disease burden caused by overweight and obesity continues to rise. Reformulation of food products to create a food supply that supports the control of weight at both an individual and population level provides a realistic opportunity to improve the health of many communities around the world. A series of actions focused on sodium and *trans* fat reduction have proved the effectiveness of this approach and governments need to broaden their target setting. For obesity control, preventive efforts that shift the focus from individual macronutrients to energy density and portion size appear to have most potential. Government leadership in setting reformulation targets, sector-wide food industry participation, objective independent monitoring of progress, and evaluations of sustainability, equity and cost–effectiveness will be required to achieve the required outcomes.

Acknowledgments Michelle Crino, Gary Sacks, Gary Swinburn and Bruce Neal are researchers within an NHMRC Centre for Research Excellence in Obesity Policy and Food Systems (APP1041020). Bruce Neal is supported by an Australian Research Council Future Fellowship (DP100100295) and a National Health and Medical Research Council of Australia Senior Research Fellowship (APP100311).

Compliance with Ethics Guidelines

Conflict of Interest Michelle Crino has received scholarship money from Sanitarium Honours Scholarship 2011, and was in the Student Placement Program 2009 at Campbell Arnott's.

Gary Sacks and Boyd Swinburn declare that they have no conflict of interest.

Stefanie Vandevijvere has received a Fellowship through the University of Auckland, and received travel/accommodations expenses covered or reimbursed for attending International conferences from Heart Foundation New Zealand, Maurice and Phyllis Paykel Trust New Zealand, and Auckland Medical Research Foundation.

Bruce Neal has received grant support from Australian Food and Grocery Council, and he interacts regularly on a non-financial basis with multiple large corporations in the Food Processing Industry and the Quick Service Restaurant industry in Australia and overseas in his efforts to improve the quality of the food supply.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

1.• Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2014. doi:10.1016/S0140-6736(14)60460-8. *This paper provides a comprehensive, up-to-date analysis of obesity trends over the past 30 years. Furthermore, this study highlights the significant burden of the obesity epidemic and the urgent need for public health intervention.*
2. Shiri R, Karppinen J, Leino-Arjas P, et al. The association between obesity and low back pain: a meta-analysis. Am J Epidemiol. 2010;171(2):135–54.
3. Dixon JB. The effect of obesity on health outcomes. Mol Cell Endocrinol. 2010;316(2):104–8.
4. World Health Organisation. Global status report on non-communicable diseases 2010. Geneva, Switzerland: 2011.
5. World Health Organisation. Assessing national capacity for the prevention and control of non-communicable diseases: report of the 2010 global survey. Geneva, Switzerland: 2012.
6. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990

and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2095–128.

7. Kac G, Perez-Escamilla R. Nutrition transition and obesity prevention through the life-course. Int J Obes Suppl. 2013;3(S1):S6–8.
8. Popkin BM, Gordon-Larsen P. The nutrition transition: worldwide obesity dynamics and their determinants. Int J Obes Relat Metab Disord. 2004;28(S3):S2–9.
9. Popkin BM. The nutrition transition: an overview of world patterns of change. Nutr Rev. 2004;62:S140–3.
10. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. Nutr Rev. 2012;70(1):3–21.
11. Baker P, Friel S. Processed foods and the nutrition transition: evidence from Asia. Obes Rev. 2014;12(7):564–77.
12. National Heart Foundation of Australia. Rapid review of the evidence: effectiveness of food reformulation as a strategy to improve population health. Australia. 2012.
13. Monteiro C, Levy R, Claro R, et al. Increasing consumption of ultra-processed foods and likely impact on human health: evidence from Brazil. Public Health Nutr. 2010;14(1):5–13.
14. Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. Lancet. 2011;378(9793):804–14.
15. Clark SE, Hawkes C, Murphy SM, et al. Exporting obesity: US farm and trade policy and the transformation of the Mexican consumer food environment. Int J Occup Environ Health. 2012;18(1):53–64.
16. Thow AM, McGrady B. Protecting policy space for public health nutrition in an era of international investment agreements. Bull World Health Organ. 2014;92(2):139–45.
17.•• Dwyer-Lindgren L, Freedman G, Engell RE, et al. Prevalence of physical activity and obesity in US counties, 2001-2011: a road map for action. Popul Health Metrics. 2013;11(1):7. *This extensive report, with highly robust statistical analysis provides a turning point in the physical activity and obesity debate. Findings from this study help to pin-point key areas for the development of public health initiatives when addressing the obesity epidemic.*
18. Swinburn B, Sacks G, Ravussin E. Increased food energy supply is more than sufficient to explain the US epidemic of obesity. Am J Clin Nutr. 2009;90(6):1453–6.
19. Hall KD, Sacks G, Chandramohan D, et al. Quantification of the effect of energy imbalance on bodyweight. Lancet. 2011;378(9793):826–37.
20. Luke A, Cooper RS. Physical activity does not influence obesity risk: time to clarify the public health message. Int J Epidemiol. 2013;42(6):1831–6.
21. Hallal PC, Andersen LB, Bull FC, et al. Global physical activity levels: surveillance progress, pitfalls, and prospects. Lancet. 2012;380(9838):247–57.
22. Hill JO. Understanding and addressing the epidemic of obesity: an energy balance perspective. Endocr Rev. 2006;27(7):750–61.
23. Hill JO, Wyatt HR, Reed GW, et al. Obesity and the environment: where do we go from here? Science. 2003;299(5608):853–5.
24. Swinburn BA, Sacks G, Lo S, et al. Estimating the changes in energy flux that characterize the rise in obesity prevalence. Am J Clin Nutr. 2009;89:1723–8.
25. United States Department of Health and Human Services. Physical activity and health: a report of the Surgeon General. Atlanta GA: 1996.
26.•• Fogelholm M, Anderssen S, Gunnarsdottir I, et al. Dietary macronutrients and food consumption as determinants of long-term weight change in adult populations: a systematic literature review. Food and Nutrition Research. 2012; 56. *This comprehensive systematic review is of great importance to public health when asssessing the food environment (primarily composition) in relation to weight gain and obesity. The extensive summarised details provided in this review regarding macronutrient and overall food consumption highlights key areas and action points for public health interventions.*
27. Sacks FMMD, Bray GAMD, Carey VJP, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. N Engl J Med. 2009;360(9):859–73.
28. Roush K. Diets vs. combinations of fat, protein, and carbohydrates. AJN Am J Nurs. 2009;109(11):64.
29. Tsai AG, Wadden TA. Systematic review: an evaluation of major commercial weight loss programs in the United States. Ann Intern Med. 2005;142(1):56–66.
30. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. Int J Pediatr Obes. 2006;1:11–25.
31. Vandevijvere S, Monteiro C, Krebs-Smith S, et al. Monitoring and benchmarking population diet quality globally: a step-wise approach. Obes Rev. 2013;14(S1):135–49.
32. Swinburn B, Sacks G, Vandevijvere S, et al. INFORMAS (International Network for Food and Obesity/non-communicable diseases Research, Monitoring and Action Support): overview and key principles. Obes Rev. 2013;14:1–12.
33. Swinburn B, Caterson I, Seidell JC, et al. Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr. 2004;7(1a):123–46.
34. Schutz Y. Macronutrients and energy balance in obesity. Metabolism. 1995;44(S3):7–11.
35. Abete I, Astrup A, Martínez JA, et al. Obesity and the metabolic syndrome: role of different dietary macronutrient distribution patterns and specific nutritional components on weight loss and maintenance. Nutr Rev. 2010;68(4):214–31.
36. Moubarac J, Martins A, Claro R, et al. Consumption of ultra-processed foods and likely impact on human health. Evidence from Canada. Public Health Nutr. 2012;16(12):2240–8.
37. Monteiro C. Nutrition and health. The issue is not food, nor nutrients, so much as the processing (Invited commentary). Public Health Nutr. 2009;12(5):729–31.
38. Popkin B. Global nutrition dynamics: the world in shifting rapidly toward a diet linked with noncommunicable diseases. Am J Clin Nutr. 2006;84:289–98.
39. Stuckler D, McKee M, Ebrahim S, et al. Manufacturing epidemics: the role of global producers in increased consumption of unhealthy commodities including processed foods, alcohol, and tobacco. PLoS Med. 2012;9(6):e1001235.
40. Kearney J. Food consumption trends and drivers. Phil Trans R Soc B Biol Sci. 2010;365(1554):2793–807.
41. Kane G. What is the real cost of our food? Implications for the environment, society and public health nutrition. Public Health Nutr. 2011;15(2):268–76.
42. Drewnowski A. Obesity and the food environment: Dietary energy density and diet costs. Am J Prev Med. 2004;27(S3):154–62.
43. Austin GL, Ogden LG, Hill JO. Trends in carbohydrate, fat, and protein intakes and association with energy intake in normal-weight, overweight, and obese individuals: 1971–2006. Am J Clin Nutr. 2011;93(4):836–43.
44. Moubarac J-C, Batal M, Martins APB, et al. Processed and ultra-processed food products: consumption trends in Canada from 1938 to 2011. Can J Diet Pract Res. 2014;75(1):15–21.
45. Seiden A, Hawley NL, Schulz D, et al. Long-term trends in food availability, food prices, and obesity in Samoa. Am J Hum Biol. 2012;24(3):286–95.
46. Sheehy T, Sharma S. The nutrition transition in Barbados: trends in macronutrient supply from 1961 to 2003. Br J Nutr. 2010;104(08):1222–9.
47. Golzarand M, Mirmiran P, Jessri M, et al. Dietary trends in the Middle East and North Africa: an ecological study (1961 to 2007). Public Health Nutr. 2012;15(10):1835–44.

48. World Health Organisation. Diet, nutrition and the prevention of chronic diseases. Report of the Joint WHO/FAO Expert Consultation Geneva: 2003.
49. Alexandratos N, Bruinsma J. World agriculture towards 2030/2050: the 2012 revision. ESA Working paper Rome, FAO, 2012.
50. Australian Bureau of Statistics. Australian Health Survey: Nutrition First Results - Food and Nutrients, 2011-12. Canberra, Australia: 2014.
51. World Health Organisation. WHO Draft Guideline: sugars intake for adults and children. Geneva: World Health Organisation; 2014.
52. Bray GA, Champagne CM. Beyond energy balance: there is more to obesity than kilocalories. J Am Diet Assoc. 2005;105(S5):17–23.
53. Hall KD. Mechanisms of metabolic fuel selection: modeling human metabolism and body-weight change. Eng Med Biol Mag IEEE. 2010;29(1):36–41.
54. Buchholz AC, Schoeller DA. Is a calorie a calorie? Am J Clin Nutr. 2004;79(5):899S–906.
55. Astrup A. Macronutrient balances and obesity: the role of diet and physical activity. Public Health Nutr. 1999;2(Supplement 3a): 341–7.
56. Howarth NC, Saltzman E, Roberts S. Dietary fiber and weight regulation. Nutr Rev. 2001;59(5):129–39.
57. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ: Br Med J. 2013;346.
58. Grimes CA, Riddell LJ, Campbell KJ, et al. Dietary salt intake, sugar-sweetened beverage consumption, and obesity risk. Pediatrics. 2012.
59. Song WO, Wang Y, Chung CE, et al. Is obesity development associated with dietary sugar intake in the U.S.? Nutrition. 2012;28(11–12):1137–41.
60. Williams P. Viewpoint. Sugar: is there a need for a dietary guideline in Australia? Aust J Nutr Diet. 2000;58:26–31.
61. Bes-Rastrollo M, Schulze MB, Ruiz-Canela M, et al. Financial conflicts of interest and reporting bias regarding the association between sugar-sweetened beverages and weight gain: a systematic review of systematic reviews. PLoS Med. 2013;10(12):e1001578.
62. Malik VS, Pan A, Willett WC, et al. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. Am J Clin Nutr. 2013;98(4):1084–102.
63. de Ruyter JC, Olthof MR, Seidell JC, et al. A trial of sugar-free or sugar-sweetened beverages and body weight in children. N Engl J Med. 2012;367(15):1397–406.
64. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. Am J Clin Nutr. 2006;84(2):274–88.
65. Astrup A, Buemann B, Flint A, et al. Low-fat diets and energy balance: how does the evidence stand in 2002? Proc Nutr Soc. 2002;61(02):299–309.
66. Hill JO, Melanson EL, Wyatt HT. Dietary fat intake and regulation of energy balance: implications for obesity. J Nutr. 2000;130(2): 284.
67. Crowe T, La Fontaine H, Gibbons C, et al. Energy density of foods and beverages in the Australian food supply: influence of macronutrients and comparison to dietary intake. Eur J Clin Nutr. 2004;58:1485–91.
68. Stubbs J, Ferres S, Horgan G. Energy density of foods: effects on energy intake. Crit Rev Food Sci Nutr. 2000;40(6):481–515.
69. Thomas JG, Doshi S, Crosby RD, et al. Ecological momentary assessment of obesogenic eating behavior: combining person-specific and environmental predictors. Obesity. 2011;19(8):1574–9.
70. Murtaugh MA, Herrick JS, Sweeney C, et al. Diet composition and risk of overweight and obesity in women living in the southwestern United States. J Am Diet Assoc. 2007;107(8):1311–21.
71. Kantor ED, Lampe JW, Kratz M, et al. Lifestyle factors and inflammation: associations by body mass index. PLoS ONE. 2013;8(7):e67833.
72. Muñoz-Pareja M, Guallar-Castillón P, Mesas AE, et al. Obesity-related eating behaviors are associated with higher food energy density and higher consumption of sugary and alcoholic beverages: a cross-sectional study. PLoS ONE. 2013;8(10):e77137.
73. Wang L, Lee I, Manson JE, et al. Alcohol consumption, weight gain, and risk of becoming overweight in middle-aged and older women. Arch Intern Med. 2010;170(5):453–61.
74. Schröder H, Morales-Molina J, Bermejo S, et al. Relationship of abdominal obesity with alcohol consumption at population scale. Eur J Nutr. 2007;46(7):369–76.
75. Sayon-Orea C, Martinez-Gonzalez MA, Bes-Rastrollo M. Alcohol consumption and body weight: a systematic review. Nutr Rev. 2011;69(8):419–31.
76. Flores M, Macias N, Rivera M, et al. Dietary patterns in Mexican adults are associated with risk of being overweight or obese. J Nutr. 2010;140(10):1869–73.
77. Howarth NC, Murphy SP, Wilkens LR, et al. Dietary energy density is associated with overweight status among 5 ethnic groups in the multiethnic cohort study. J Nutr. 2006;136(8):2243–8.
78. Baldini M, Pasqui F, Bordoni A, et al. Is the Mediterranean lifestyle still a reality? Evaluation of food consumption and energy expenditure in Italian and Spanish university students. Public Health Nutr. 2009;12(02):148–55.
79. Davey RC. The obesity epidemic: too much food for thought? Br J Sports Med. 2004;38(3):360–3.
80. Drewnowski A, Darmon N. The economics of obesity: dietary energy density and energy cost. Am J Clin Nutr. 2005;82(1):265S–73.
81. Drewnowski A, Rolls B. How to modify the food environment. J Nutr. 2005;135:898–9.
82. Drewnowski A, Almiron-Roig E, Marmonier C, et al. Dietary energy density and body weight: is there a relationship? Nutr Rev. 2004;62(11):403–13.
83. Vernarelli JA, Mitchell DC, Hartman TJ, et al. Dietary energy density is associated with body weight status and vegetable intake in US children. J Nutr. 2011;141(12):2204–10.
84. Matthiessen J, Fagt S, Biltoft-Jensen A, et al. Size makes a difference. Public Health Nutr. 2003;6(01):65–72.
85. Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977-1998. JAMA. 2003;289(4):450–3.
86. Steenhuis IH, Leeuwis FH, Vermeer WM. Small, medium, large or supersize: trends in food portion sizes in The Netherlands. Public Health Nutr. 2010;13(06):852–7.
87. Young LR, Nestle M. Reducing portion sizes to prevent obesity: a call to action. Am J Prev Med. 2012;43(5):565–8.
88. Young L, Nestle M. Portion sizes and obesity: responses of fast-food companies. J Public Health Policy. 2007;28:238–48.
89. Steenhuis I, Vermeer W. Portion size: review and framework for interventions. Int J Behav Nutr Phys Act. 2009;6(1):1–10.
90. Ledikwe JH, Ello-Martin JA, Rolls BJ. Portion Sizes and the Obesity Epidemic. J Nutr. 2005;135(4):905–9.
91. Ello-Martin J, Ledikwe J, Rolls B. The influence of food portion size and energy density on energy intake: implications for weight management. Am J Clin Nutr. 2005;82:S236–41.
92. Rolls B, Roe L, Meengs J. Reductions in portion size and energy density of foods are additive and lead to sustained decreases in energy intake. Am J Clin Nutr. 2006;83:11–7.
93. Poelman MP, Steenhuis IH, de Vet E, et al. The development and evaluation of an internet-based intervention to increase awareness about food portion sizes: a randomized, controlled trial. J Nutr Educ Behav. 2013;45(6):701–7.
94. Vermeer W, Steenhuis I, Poelman M. Small, medium, large or supersize? the development and evaluation of interventions targeted at portion size. Int J Obes. 2014;38:S13–8.

95. Murakami K, Miyake Y, Sasaki S, et al. An energy-dense diet is cross-sectionally associated with an increased risk of overweight in male children, but not in female children, male adolescents, or female adolescents in Japan: the Ryukyus Child Health Study. Nutr Res. 2012;32(7):486–94.
96. Steyn NP, Nel JH, Parker W-A, et al. Dietary, social, and environmental determinants of obesity in Kenyan women. Scand J Public Health. 2011;39(1):88–97.
97. Hawkes C, Jewell J, Allen K. A food policy package for healthy diets and the prevention of obesity and diet-related non-communicable diseases: the NOURISHING framework. Obes Rev. 2013;14(S2):159–68.
98. World health Organisation. Global action plan for the prevention and control of non-communicable diseases 2013-2020. Geneva: World Health Organisation; 2013.
99. World Cancer Research Fund. WCRF international food policy framework for healthy diets: NOURISHING - improve nutritional quality of the whole food supply London 2014. Available from: http://www.wcrf.org/policy_public_affairs/nourishing_framework/food_supply_composition_reformulation. Accessed June 2014.
100. Pietinen P, Valsta LM, Hirvonen T, et al. Labelling the salt content in foods: a useful tool in reducing sodium intake in Finland. Public Health Nutr. 2008;11(04):335–40.
101. Laatikainen T, Pietinen P, Valsta L, et al. Sodium in the Finnish diet: 20-year trends in urinary sodium excretion among the adult population. Eur J Clin Nutr. 2006;60(8):965–70.
102. Puska P, Ståhl T. Health in all policies—the Finnish initiative: background, principles, and current issues. Annu Rev Public Health. 2010;31(1):315–28.
103. Wyness LA, Butriss JL, Stanner SA. Reducing the population's sodium intake: the UK Food Standards Agency's salt reduction programme. Public Health Nutr. 2012;15(02):254–61.
104. Webster J, Dunford E, Hawkes C, et al. Salt reduction initiatives around the world. J Hypertens. 2011;29:1043–50.
105. Department of Health. Public health responsibility deal: calorie reduction pledge United Kingdom: Department of Health, Government of the United Kingdom; 2014. Accessed: available from: https://responsibilitydeal.dh.gov.uk/pledges/pledge/?pl=23.
106. Cobiac LJ, Vos T, Veerman JL. Cost-effectiveness of interventions to reduce dietary salt intake. Heart. 2010;96(23):1920–5.
107. Bibbins-Domingo K, Chertow GM, Coxson PG, et al. Projected effect of dietary salt reductions on future cardiovascular disease. N Engl J Med. 2010;362(7):590–9.
108. Cappuccio FP, Capewell S, Lincoln P, et al. Policy options to reduce population salt intake. BMJ. 2011;343:4995.
109. Palar K, Sturm R. Potential societal savings from reduced sodium consumption in the US adult population. Am J Health Promot. 2009;24(1):49–57.
110. Slining MM, Ng SW, Popkin BM. Food companies' calorie-reduction pledges to improve U.S. diet. Am J Prev Med. 2013;44(2):174–84.
111. Department of Health. Industry engagement Australia: Australian Government Department of Health; 2009. Available from: http://www.foodhealthdialogue.gov.au/internet/foodandhealth/publishing.nsf/Content/industry-engagement. Accessed Feb 2014.
112. Moodie R, Stuckler D, Monteiro C, et al. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. Lancet. 2013;381:670–9.