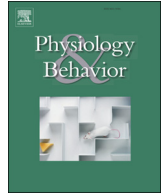




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Review

Are large portions responsible for the obesity epidemic?



C. Peter Herman^{a,*}, Janet Polivy^b, Lenny R. Vartanian^c, Patricia Pliner^b

^a Department of Psychology, University of Toronto, Canada

^b Department of Psychology, University of Toronto Mississauga, Canada

^c Department of Psychology, University of New South Wales, Australia

HIGHLIGHTS

- Large portions are frequently blamed for the obesity epidemic.
- Evidence that extended exposure to large portions causes weight gain is scanty.
- Some epidemiological data argue against large portions as the cause.
- Other possible causes have equal or great cogency.
- We should be more cautious about blaming large portions for the obesity epidemic.

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ABSTRACT

Large portion sizes are frequently blamed for the obesity epidemic. In this paper, we examine the culpability of large portion sizes. It is true that portion sizes have increased during the obesity epidemic, but there is as yet little evidence that exposure to large portions produces significant weight gain. Furthermore, some evidence argues against the role of large portions in the obesity epidemic. For instance, the epidemic is largely a matter of weight gain among the upper half of the population weight distribution, whereas large portions appear to have an indiscriminate effect on food intake. Factors other than the size of individual meals may well be responsible for a great deal of the observed population weight gain.

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* Corresponding author at: Department of Psychology, University of Toronto, Toronto, Ontario M5S 3G3, Canada.

E-mail addresses: herman@psych.utoronto.ca (C.P. Herman), polivy@psych.utoronto.ca (J. Polivy), l.vartanian@unsw.edu.au (L.R. Vartanian), patricia.pliner@utoronto.ca (P. Pliner).

1. Introduction

The portion-size effect (PSE) refers to the empirical observation that people tend to eat more when they are served larger portions. The PSE is not a mere artifact of people cleaning their plates and therefore necessarily eating more when there is initially more food on the plate. Reviews of the PSE have appeared in recent years [1–4], as have more speculative discussions of possible mechanisms underlying the PSE [5, 6]. With these overviews in hand, we may turn our attention to a question of greater interest to the public – namely, whether large portion sizes are responsible for the obesity epidemic.

1.1. Blaming large portions for the obesity epidemic

Excessive energy intake in response to increasing portion sizes has become a popular target to blame for the obesity epidemic. As Benton [1] notes, “it has become received wisdom that an increase in portion size has played a part in the raised incidence of obesity” (p. 988).

Certainly, large portion sizes are not the only target of anti-obesity crusaders; junk food, high-fructose corn syrup, increased sedentariness, and various other ills of modern society have all received their share of the blame [7]. Keith, Redden, Katzmarzyk et al. [8] and McAllister, Dhurandhar, Keith et al. [9] offer several other, more exotic candidate causes of or contributors to the epidemic, ranging from sleep deprivation to viruses, some of which might operate by increasing positive energy balance, but not necessarily by increasing intake. The most favored explanations involve what Keith et al. refer to as the “Big Two” (i.e., increased energy intake and decreased energy expenditure); and although there are several other plausible factors that might contribute to increased energy intake, large portion size is often identified as a prime culprit. Stroebe [10] asserts that “studies have demonstrated that...increased portion...size contribute[s] substantially to caloric intake and weight gain” (p. 91). Others are slightly more tentative, but statements such as “the proportion of people who are overweight has increased dramatically worldwide, partly due to increasing portion sizes of particularly energy dense foods” [11, p. 96] are ubiquitous in the literature.

Rolls often qualifies her indictment of large portion sizes, claiming that it is specifically energy-dense foods served in large portions that pose the problem: she refers to an “accumulating body of evidence that large portions of energy-dense foods are associated with increased energy intake and that this intake is contributing to the rising incidence of obesity” [12, p. 567; see also 3]. If people were to eat large portions of low-energy-dense food, the problem might be lessened or even reversed. The fact remains, however, that high-energy-dense food is popular and widely available. For some people it is a matter of economics, with people getting more caloric bang for the buck with high-energy-dense food, whereas for others it is simply a matter of palatability. Ultimately, for Rolls as for other commentators, the obesity epidemic is (almost certainly) a matter of people ingesting too many calories, with large portions promoting such overeating.

1.2. A more cautious approach to assigning blame

We accept Keith et al.'s [8] argument that for a particular candidate factor to be a plausible contributor to the obesity epidemic, it must satisfy two criteria: first, the factor must be shown to increase weight when applied in an experimental (i.e., non-confounded) context; and second, the factor must be shown to have increased in prevalence or power during the years – let us say, since 1980 – when obesity began to reach epidemic proportions. It is important to note that satisfying these criteria is necessary but not sufficient for establishing a given factor as a cause of the epidemic. Indeed, it is probably impossible to declare with certainty that a given candidate cause has satisfied the criteria for sufficiency; in short, we can retain candidate causes but we cannot enshrine them as definite causes. If large portions do satisfy Keith et al.'s criteria, then large portions *may* be a plausible cause of

the obesity epidemic; but other factors may also satisfy the criteria, leaving large portions as only one of a set of competing (or complementary) explanatory factors. We may consider another epidemic, say lung cancer in the late 20th century, which would appear to satisfy Keith et al.-type criteria. We can possibly pinpoint cigarette smoking as *the* cause, even while acknowledging that some non-smokers develop lung cancer. In the case of lung cancer, it may be possible to identify which risk factors are most prevalent among those with the disease; there is no absolute certainty, but there is converging evidence. Do we have equally useful data for the obesity epidemic? Let us begin by asking whether large portions satisfy the criteria.

2. Do large portions lead to weight gain?

The first criterion requires that large portions demonstrably increase weight when applied in a systematic fashion. Such demonstrations, it turns out, are difficult to find. Most of the many studies on the portion-size effect demonstrate that large portions increase intake over the course of a single meal (see [1–4] for reviews). On reflection, we must concede that eating a single large or oversize meal is unlikely to produce a detectable increase in weight. What are needed are studies of extended exposure to large portions in which the participants' weight is tracked over time. Such studies are rare. Of course, studies of extended exposure are difficult to conduct and require resources not available to many researchers. The food industry does not have much incentive to sponsor such studies, unless such studies are expected to reveal that extended exposure to large portions does not lead to weight gain.

Some studies have measured the effects on food intake of extended exposure to large portions. Rolls, Roe, and Meengs [13 (2 days), 14 (11 days)] found that people maintain an increased intake when their exposure to large portions continues over days, but these studies did not measure changes in body weight, so they do not speak directly to our question. Jeffery, Rydell, Dunn et al. [15] exposed participants to a larger or smaller portion for four weeks (weekday lunches only, crossover design) and found increased intake along with differential weight gain in the larger (0.64 kg) versus smaller (0.06 kg) condition, although this weight-gain difference was not statistically significant ($p = 0.13$). Kelly, Wallace, Robson et al. [16] exposed experimental participants to standard or large portions during all meals for four consecutive days in a crossover design. Participants ate more in the large-portion condition and gained a significant amount of weight (on average, about 0.75 kg) over the course of the four days. In the most ambitious study, French, Mitchell, Wolfson et al. [17] assigned participants to eat free box lunches (400, 800, or 1600 kcal) on weekdays for 6 months. The 1600 kcal condition led to greater weight gain (1.1 kg) than did the 400 or 800 kcal conditions (both -0.1 kg); however, weight gain in the 1600 kcal condition was identical to the weight gain experienced by control participants who did not receive a free box lunch. Thus, French et al.'s data would appear to say more about the weight-control potential of moderate or small portions than about the weight-gain potential of large portions. Mattes [18] recently concluded that “there are no randomized controlled trials that would permit assignment of causality between long-term exposure to large portions and increments in body weight” (p. 112). Kelly et al., as we have seen, did find some evidence of weight gain in response to exposure to large portions over four days; but it is conceivable that with longer exposure to large portions, people would learn to compensate for the increased intake/weight and their intake/weight would subside. At this point, it would appear to be prudent to maintain some agnosticism regarding the effects of long-term exposure to large portions, given the paucity of hard data.

2.1. Have portions increased in size?

It is easier to adduce evidence for Keith et al.'s [8] second criterion – namely that the candidate cause has increased in prevalence or power over the course of the obesity epidemic. The inflation of portion sizes

over recent decades has been well documented. The 12-oz Coke that used to be “king-sized” in the 1950s¹ is now “child-sized,” with the 20-oz bottle now standard in vending machines [19], and at a contemporary steakhouse it is relatively easy to order (if not pay for) a 24-oz steak, which is 8–12 times the USDA recommended serving for meat [20]. Young and Nestle [20] found that portions of many foods are both large in an absolute sense and larger than in the past, in some cases doubling or tripling or more in size. It is not unusual for a single item (e.g., a muffin or a soda) nowadays to exceed by 100% or more the “recommended serving size” listed in small print on the packaging [20]. That portion sizes have increased dramatically over the past 30 years has become a truism. For instance, Hill and Peters [7] bemoan “a growing trend in the United States toward larger portions. This is especially evident in so-called fast-food restaurants, where ‘super sizing’ of menu items is commonplace” (p. 1371) and according to Young and Nestle [21], “for french fries, hamburgers, and soda ...current sizes are 2–5 times larger than the originals” (p. 246). A decade later, Young and Nestle [22] observed that “marketplace portions of many foods have continued to increase through the first decade of the 21st century” (p. 565), with burgers and sandwiches now approaching and exceeding 1000 kcal. (The burger entrée itself often exceeds 1000 kcal. A large order of fries can exceed 500 kcal and a soft drink can contain as many as 800 kcal [22].)

Although the finger is usually pointed at fast-food joints, it is worth noting that “fine dining” restaurants also tend to serve portions well in excess of USDA recommendations [23]; interestingly, younger chefs tend to serve larger portions [23], possibly a reflection of their not having been schooled in the smaller portions of yesteryear. The increase in portion sizes at restaurants, multiplied by the increasing proportion of meals eaten in restaurants, suggests a large increase in intake; Americans have appreciably increased the proportion of meals eaten away from home, and the proportion of meals eaten away from home is positively associated with energy intake [24]. French et al. [19] calculate that if people ate all their meals at home, they would eat about 200 fewer kcals a day.

It is easier to measure restaurant servings than what is prepared and eaten at home. Still, Wansink and Payne [25] found that portion sizes for home-cooked meals have increased dramatically over the past 70 years, if recipes (and serving-size recommendations) in *The Joy of Cooking* are any indication (see also [26]). The Eidner et al. study [26] serves as a reminder that increased portion sizes are not confined to the United States. American portions may be larger than those served elsewhere [27, 28], but portion size has certainly increased over the past few decades in Danish grocery stores, fast-food restaurants, and snack outlets [29]. A similar trend has been detected in the Netherlands [30]. Benton [1], on the other hand, argues that in the UK the trend toward larger portions is fitful at best.

We must remember that a portion served is not necessarily a portion consumed. The PSE, of course, demands that larger portions promote greater intake, but most of the studies on which the PSE is based are perhaps somewhat ecologically invalid in that they involve free food that is not easily put aside for later consumption. It is at least conceivable that larger portions at home could simply mean more leftovers. At restaurants, leftovers to take home are perhaps less feasible; but as restaurant portions reach gargantuan proportions, patrons may be more likely to split the portion and take half home, a strategy that makes financial sense to both the patron and the restaurant. Whether large portions are entirely consumed at one occasion or not, we may conclude with some confidence that portion sizes have indeed increased in the same time period as has the obesity epidemic.

Interestingly, Fisher et al. [31] note that “a recent survey by the American Institute for Cancer Research found that less than one-third

of 1003 adults surveyed believe that portion sizes at restaurants have increased over the past 30 years” (p. 1169); these people must be unaware of the newspapers, or even more scandalously, the scientific literature. Further, Seiders and Petty [32] cite studies indicating that people underestimate the role of large portion sizes in promoting a positive energy balance. Rolls [33] cites surveys in which the vast majority of respondents report that “the type of food they eat is more important for weight management than the amount of food” (p. 46). Perhaps one reason (beyond value-for-money) that portions have expanded with so little resistance from the public is that the public focuses more on what to eat than on how much to eat.

2.2. Interim conclusions

With respect to Keith et al.’s criteria, the evidence is fairly strong that portions have increased in size during the years of the obesity epidemic. It is equally clear, however, that evidence supporting the proposition that exposure to large portions increases weight is very weak, if not absent altogether. In the words of Livingstone and Pourshahidi, “despite pervasive commercial trends toward large portions, there is surprisingly little compelling evidence that these are causally linked to obesity” [4, p. 831]. Given that both of the criteria must be satisfied in order for a candidate factor to be considered a plausible contributor to the obesity epidemic, we are forced to the conclusion that large portions do not (yet) qualify. Admittedly, there have been so few studies of the effects on weight of long-term exposure to large portions that we cannot rule out the possibility that as more evidence accumulates, the case may become stronger. At present, however, the case cannot be made. The most that can be said is that the required evidence is lacking. As much as we would like to see the data, it is not clear that well-conducted studies of long-term exposure to large portions are in the offing. Also, it is worth remembering that even if long-term-exposure studies led to both criteria being satisfied, we could conclude only that large portions provide one plausible candidate explanation among many for the obesity epidemic. Fulfilling Keith et al.’s criteria is necessary but not sufficient.

3. Contrary evidence

It is possible that further research will provide support for the inclusion of large portions among the candidate causes of the obesity epidemic according to the Keith et al. criteria. The jury is still out. More problematic for those who would indict large portions is evidence that runs directly contrary to the notion that large portions are making people fat. We will now consider two types of data that suggest that those who blame large portions are pointing the finger in the wrong direction.

3.1. The epidemiology of the obesity epidemic and the PSE

One of the most intriguing aspects of the obesity epidemic is that it has not manifested as a “main effect.” That is, some groups of people have become much fatter whereas others have barely become fatter at all. Flegal and Troiano’s [34] data, from consecutive National Health and Nutrition Examination Surveys (NHANES) administered repeatedly over time, indicate clearly that mean weight (adjusted for height) has increased much more than has median weight. In short, the preponderance of the gain in weight over time has been at the upper end of the distribution of initial weight. (Note: The NHANES surveys, administered at succeeding time intervals, are not within-subject measures, so it is not quite fair to say that the lighter people at earlier times gained less weight in succeeding measurement periods than did the heavier people. Still, it is fair to say that the proportion of the population that has become obese has not increased to the extent that most people assume. Rather, the heavier half of the population distribution has become much more overweight, whereas the lighter half has not.)

How is the epidemiological pattern of weight shifts relevant to the question of whether large portion sizes are responsible for the obesity

¹ A Pepsi jingle from the 1950s: “Pepsi Cola hits the spot, 12 full ounces, that’s a lot....” (Thanks to David Goldstein for this reminiscence.) In the film *From Here to Eternity* (1953), Montgomery Clift orders a large Coke for Donna Reed and she receives a glass of Coke containing perhaps 10 oz.

epidemic? Simply, if portion size is the culprit, then it should be the case that larger portion sizes have a stronger effect on overweight people than on normal-weight people. Such a differential effect would be consistent with the disproportionate increase in weight among people in the upper half of the weight distribution (i.e., the fat becoming fatter). The preponderance of experimental data, however, suggests that the portion-size effect (PSE) applies equally to normal-weight and overweight individuals. Most studies that bother to measure participants' weight or BMI find no differences in the magnitude of the PSE as a function of these participant characteristics, in adults or in children. Many of these studies have been conducted by Rolls and her colleagues, who make a point of measuring BMI [13, 14, 35–44] but other labs have found the same thing [45–47]. Inevitably, there are some conflicting data. Some studies (e.g., [48]) have found a stronger PSE for heavier participants, but often this differential effect is restricted to one sex or the other (e.g., 16) or for certain foods (e.g., [49]). In fact, a recent meta-analysis showed that overweight people are somewhat less responsive to increases in portion size than are normal-weight people [50]. On balance, these data do not support the notion that the PSE applies differentially to the already-overweight and thereby accounts for the fat-getting-fatter pattern documented by Flegal and Troiano [34].

The experimental studies examining the PSE typically expose both heavier and lighter participants to the same (larger and smaller) portions. If we think of inflated portions as being imposed uniformly on the entire population, then such studies may be appropriate for answering the question of whether large portions have different effects on heavier and lighter people. It remains possible, however, that heavier and lighter people (in the real world) do not experience the same portion sizes (as they would in any well-designed lab or field experiment). What if heavier people differentially *select* large portions, as was found, for example, by Dodd, Birky, and Stalling [51]? In that case, the PSE might well operate to increase weight differentially among the already overweight. There is some evidence suggesting that overweight people eat larger (or at least more calorically dense) portions than do normal-weight people (e.g. [52]), but that is not quite the same as evidence that overweight people select larger portions to begin with; and other research finds no difference between heavier and lighter participants in portions consumed of various foods (e.g., [53]). Brunstrom, Rogers, Pothos, Caltri, and Tapper [54] found no association between BMI and portions typically consumed. These studies, unfortunately, assess how much people actually eat, whereas what we are looking for are data addressing the question of whether overweight people are more likely to *order* or *serve themselves* a larger portion in the first place, perhaps because a larger portion represents greater value-for-money in terms of calories per dollar? This question has not been adequately addressed. Or perhaps, even if the PSE applies equally to overweight and normal-weight people, overweight people are not as effective at dealing with the caloric consequences. Even if large portions result in the same increase in intake, it may be that overweight people are more sluggish metabolically or poorer at implementing compensatory behaviors (eating less on subsequent occasions or making efforts to expend more energy). Accounting for the epidemiological data, then, probably requires not only large portions but also physiological or and/behavioral differences between the heavier and lighter segments of the population. In statistical terms, we are looking at an interaction, not a main effect.

3.2. Eating frequency

Focusing on large portions as the culprit may distract us from other possible contributors to the obesity epidemic. As was mentioned earlier, Keith et al. [8] identified several other possible contributors to the epidemic (e.g., endocrine disruptors, smoking cessation, ambient temperature regulation). These factors have been shown to increase weight and to have increased in frequency or power during the time period of the epidemic, so they have at least as much potential explanatory merit as do large portion sizes. Another possibility is an increase in eating

frequency. In an interesting analysis, Duffey and Popkin [55] attempted, using large-scale food-intake data, to apportion changes in energy intake over time to three components: portion size (i.e., amount consumed on a given eating occasion); energy density (i.e., number of calories per unit of food consumed); and number of eating occasions. They concluded that portion size increased by 12% per eating occasion. It is important to note here that for Duffey and Popkin, “portion size” means amount consumed rather than amount served – we have been defining “portion size” as amount served or self-served – so we cannot determine from these data whether the serving sizes were larger. Energy density did not increase – in fact it declined by 2% – over time. Most surprising was the change in the number of eating occasions per day, which increased by 29%, from 3.8 to 4.9. It appears from this analysis that it is not so much that people are eating too much at a sitting as that they are eating too often! Eating more on a given occasion accounted for less than one-third of the increase in overall energy intake, whereas eating more often accounted for more than two-thirds of the effect. An earlier, similar analysis led Cutler, Glaeser, and Shapiro [56] to conclude that “the increase in caloric intake is because of greater frequency of eating, not eating more at any one sitting” (p. 101). Mattes [18], in his review, concludes that greater ingestive frequency (especially more frequent snacks and especially if the snacks are not planfully integrated into one's diet [57]) is a greater contributor to the obesity epidemic than are large meals stemming from large portion sizes. Benton [1], who concurs, even provides physiological evidence supporting the proposition that relatively small snacks are particularly unsuited to promoting caloric compensation [see also 58]. These analyses certainly point to at least one factor other than large portion sizes that could be responsible for the obesity epidemic. “The finding that increased caloric intake is from more snacks rules out [the notion] that obesity is a result of increased portion sizes” (56, p. 101). Although this conclusion may go too far, Cutler et al. go further, suggesting that the differential increase in BMI for people initially above the BMI median (i.e., the Flegal & Troiano [34] finding) reflects the particular susceptibility of heavier people, presumably deficient in self-control, to the lure of readily available, convenient, no-preparation-required snacks. So even if overweight people do in fact eat more than do normal-weight people, it may be more a matter of eating more frequently than of larger meals and/or larger portions. Of course, there remains the distinct possibility that snacks have become not only more frequent but also larger and more highly caloric.

Before we rush to indict eating frequency as the cause of the obesity epidemic, we must remember that, as with large portions, some skepticism is in order. Although it may well be the case that the number of eating episodes per day has increased over the course of the obesity epidemic, we do not have any better data on the effects of increased meal frequency on weight gain than we have on the effects of increased portion size on weight gain. And let us not forget that even if increased meal frequency were to qualify according to Keith et al.'s [8] criteria, that would not make increased meal frequency the leading candidate to explain the obesity epidemic. Increased meal frequency would simply be added to the lengthy list of viable candidates, which might still include large portions.

3.3. Further conclusions

The argument that portion size cannot be responsible for the obesity epidemic because both heavier and lighter people respond in the same way to larger portions but only heavier people have gained weight during the epidemic is admittedly convoluted. The related argument concerning whether heavier people select larger portions than do lighter people is short on conclusive data. Other data suggest that perhaps we should be pointing an accusing finger at eating frequency; the number of eating occasions per day has increased precipitously over the course of the obesity epidemic. Moreover, there may be reason to believe that this increase in eating frequency (and overall meal number) is differentially prevalent among the already-overweight insofar as

eating frequency is driven by the sensory allure of palatable foods [59]. Large portions are at best only one candidate cause among many for the obesity epidemic; and it would appear that large portions are by no means leading the pack of candidates.

4. Final conclusions

Large portions are likely to remain a popular target for anti-obesity crusaders. As we have seen, however, the actual evidence against large portions as the culprit is surprisingly weak. There is precious little evidence that extended exposure to large portions produces weight gain. This lack of evidence, of course, is not the same thing as the presence of evidence that extended exposure to large portions does *not* produce weight gain; it is simply a matter of assumptions (“extended exposure to large portions must produce weight gain”) outrunning the data, which are at this point remarkably scanty. Other candidates, including (but not confined to) more frequent eating occasions, would appear to have at least as much explanatory merit as do large portions. At the very least, we recommend a more skeptical stance toward cavalier assertions that large portions are responsible for the obesity epidemic.

Potential conflicts of interest

None.

References

- [1] D. Benton, Portion size: what we know and what we need to know, *Crit. Rev. Food Sci.* 55 (2015) 988–1004.
- [2] I.H.M. Steenhuis, W.M. Vermeer, Portion size: review and framework for interventions, *Int. J. Behav. Nutr.* 6 (2009) 58.
- [3] J.H. Ledikwe, J.A. Ello-Martin, B.J. Rolls, Portion sizes and the obesity epidemic, *J. Nutr.* 135 (2005) 905–909.
- [4] M.B.E. Livingstone, L.K. Pourshahidi, Portion size and obesity, *Adv. Nutr.* 5 (2014) 829–834.
- [5] L. English, M. Lasschuijt, K.L. Keller, Mechanisms of the portion size effect. What is known and where do we go from here? *Appetite* 88 (2015) 39–49.
- [6] C.P. Herman, J. Polivy, P. Pliner, L.R. Vartanian, Mechanisms underlying the portion-size effect, *Physiol. Behav.* 144 (2015) 129–136.
- [7] J.O. Hill, J.C. Peters, Environmental contributions to the obesity epidemic, *Science* 280 (1998) 1371–1374.
- [8] S.W. Keith, D.T. Redden, P.T. Katzmarzyk, et al., Putative contributors to the secular increase in obesity: exploring the roads less traveled, *Int. J. Obes.* 30 (2006) 1585–1594.
- [9] E.J. McAllister, N.V. Dhurandhar, S.W. Keith, et al., Ten putative contributors to the obesity epidemic, *Crit. Rev. Food Sci.* 49 (2009) 868–913.
- [10] W. Stroebe, Dieting, overweight, and obesity: self-regulation in a food-rich environment, American Psychological Association Press, Washington, DC, 2008.
- [11] E. Van Kleef, M. Shimuzu, B. Wansink, Just a bite: considerably smaller snack portions satisfy delayed hunger and craving, *Food Qual. Prefer.* 27 (2012) 96–100.
- [12] N. Diliberti, P.L. Bordi, M.T. Conklin, L.S. Roe, B. Rolls, Increased portion size leads to increased energy intake in a restaurant meal, *Obes. Res.* 12 (2004) 562–568.
- [13] B.J. Rolls, L.S. Roe, J.S. Meengs, Larger portion sizes lead to a sustained increase in energy intake over 2 days, *J. Am. Diet. Assoc.* 106 (2006) 543–549.
- [14] B.J. Rolls, L.S. Roe, J.S. Meengs, The effect of large portion sizes on energy intake is sustained for 11 days, *Obesity* 15 (2007) 1535–1543.
- [15] R.W. Jeffery, S. Rydell, C.L. Dunn, et al., Effects of portion size on chronic energy intake, *Int. J. Behav. Nutr.* 4 (2007) 27.
- [16] M.T. Kelly, J.M.W. Wallace, P.J. Robson, et al., Increased portion size leads to a sustained increase in energy intake over 4d in normal-weight and overweight men and women, *Br. J. Nutr.* 102 (2009) 470–477.
- [17] S.A. French, N.R. Mitchell, J. Wolfson, et al., Portion size effects on weight gain in a free living setting, *Obesity* 22 (2014) 1400–1405.
- [18] R.D. Mattes, Energy intake and obesity: ingestive frequency outweighs portion size, *Physiol. Behav.* 134 (2014) 110–118.
- [19] S.A. French, M. Story, R.W. Jeffery, Environmental influences on eating and physical activity, *Annu. Rev. Public Health* 22 (2001) 309–336.
- [20] L.R. Young, M. Nestle, Expanding portion sizes in the US marketplace: implications for nutrition counseling, *J. Am. Diet. Assoc.* 103 (2003) 231–234.
- [21] L.R. Young, M. Nestle, The contribution of expanding portion sizes to the US obesity epidemic, *Am. J. Public Health* 92 (2002) 246–249.
- [22] L.R. Young, M. Nestle, Reducing portion sizes to prevent obesity: a call to action, *Am. J. Prev. Med.* 43 (2012) 565–568.
- [23] M. Condraskey, J.H. Ledikwe, J. Flood, B.J. Rolls, Chefs' opinions of restaurant portion sizes, *Obesity* 15 (2007) 2086–2094.
- [24] A.K. Kant, B.I. Graubard, Eating out in America, 1987–2000: trends and nutritional correlates, *Prev. Med.* 38 (2004) 243–249.
- [25] B. Wansink, C.R. Payne, The joy of cooking too much: 70 years of calorie increases in classic recipes, *Ann. Intern. Med.* 150 (2009) 291–292.
- [26] M.B. Eidner, A.Q. Lund, B.S. Harboe, I.H. Clemmensen, Calories and portion sizes in recipes throughout 100 years: an overlooked factor in the development of over-weight and obesity? *Scand. J. Public Health* 41 (2013) 839–845.
- [27] P. Rozin, K. Kabnick, E. Pete, C. Fischler, C. Shields, The ecology of eating: smaller portion sizes in France than in the United States help explain the French paradox, *Psychol. Sci.* 14 (2003) 450–454.
- [28] L.R. Young, M. Nestle, Portion sizes and obesity: responses of fast-food companies, *J. Public Health Policy* 28 (2007) 238–248.
- [29] J. Matthiessen, S. Fagt, A. Biloft-Jensen, A.M. Beck, L. Ovesen, Size makes a difference, *Public Health Nutr.* 6 (2003) 65–72.
- [30] I.H.M. Steenhuis, F.H. Leeuwis, W.M. Vermeer, Small, medium, large or supersize: trends in food portion sizes in The Netherlands, *Public Health Nutr.* 13 (2009) 852–857.
- [31] J.O. Fisher, B.J. Rolls, L.L. Birch, Children's bite size and intake of an entree are greater with large portions than with age-appropriate or self-selected portions, *Am. J. Clin. Nutr.* 77 (2003) 1164–1170.
- [32] K. Seiders, R.D. Petty, Obesity and the role of food marketing: a policy analysis of issues and remedies, *J. Public Policy Mark.* 23 (2004) 153–169.
- [33] B.J. Rolls, The supersizing of America: portion size and the obesity epidemic, *Nutr. Today* 38 (2003) 42–53.
- [34] K.M. Flegal, R.P. Troiano, Changes in the distribution of body mass index of adults and children in the US population, *Int. J. Obes.* 24 (2000) 807–818.
- [35] J.O. Fisher, A. Arreola, L.L. Birch, B.J. Rolls, Portion size effects on daily intake in low-income Hispanic and African American children and their mothers, *Am. J. Clin. Nutr.* 86 (2007) 1709–1716.
- [36] J.O. Fisher, Y. Liu, L.L. Birch, B.J. Rolls, Effects of portion size and energy density on young children's intake at a meal, *Am. J. Clin. Nutr.* 86 (2007) 174–179.
- [37] T.V.E. Kral, L.S. Roe, B.J. Rolls, Combined effects of energy density and portion size on energy intake in women, *Am. J. Clin. Nutr.* 79 (2004) 962–968.
- [38] B.J. Rolls, E.L. Morris, L.S. Roe, Portion size of food affects energy intake in normal-weight and overweight men and women, *Am. J. Clin. Nutr.* 76 (2002) 1207–1213.
- [39] B.J. Rolls, L.S. Roe, T.V.E. Kral, J.S. Meengs, D.E. Wall, Increasing the portion size of a packaged snack increases energy intake in men and women, *Appetite* 42 (2004) 63–69.
- [40] B.J. Rolls, L.S. Roe, J.S. Meengs, Reductions in portion size and energy density of foods are additive and lead to sustained decreases in energy intake, *Am. J. Clin. Nutr.* 83 (2006) 11–17.
- [41] B.J. Rolls, L.S. Roe, J.S. Meengs, Portion size can be used strategically to increase vegetable consumption in adults, *Am. J. Clin. Nutr.* 91 (2010) 913–922.
- [42] B.J. Rolls, L.S. Roe, J.S. Meengs, D. Wall, Increasing the portion size of a sandwich increases energy intake, *J. Am. Diet. Assoc.* 104 (2004) 367–372.
- [43] M.K. Spill, L.L. Birch, L.S. Roe, B.J. Rolls, Eating vegetables first: the use of portion size to increase vegetable intake in preschool children, *Am. J. Clin. Nutr.* 91 (2010) 1237–1243.
- [44] M.K. Spill, L.L. Birch, L.S. Roe, B.J. Rolls, Serving large portions of vegetable soup at the start of a meal affected children's energy and vegetable intake, *Appetite* 57 (2011) 213–219.
- [45] D. Marchiori, L. Waroquier, O. Klein, Smaller food item sizes of snack foods influence reduced portions and caloric intake in young adults, *J. Am. Diet. Assoc.* 111 (2011) 727–731.
- [46] L. Smith, K. Conroy, H. Wen, L. Rui, D. Humphries, Portion size variably affects food intake of 6-year-old and 4-year-old children in Kunming, China, *Appetite* 69 (2013) 31–38.
- [47] B. Wansink, M.M. Cheney, Super bowls, serving bowl size and food consumption, *J. Am. Med. Assoc.* 293 (2005) 1727–1728.
- [48] K.S. Burger, J.O. Fisher, S.L. Johnson, Mechanisms behind the portion size effect: visibility and bite size, *Obesity* 19 (2011) 546–551.
- [49] T.V.E. Kral, A.C. Kabay, L.S. Roe, B.J. Rolls, Effects of doubling the portion size of fruit and vegetable side dishes on children's intake at a meal, *Obesity* 18 (2010) 521–527.
- [50] N. Zlatevska, C. Dubelaar, S.S. Holden, Sizing up the effect of portion size on consumption: a meta-analytic review, *J. Mark.* 78 (2014) 140–154.
- [51] D.K. Dodd, H.J. Birky, R.B. Stalling, Eating behavior of obese and normal weight females in a natural setting, *Addict. Behav.* 1 (1976) 321–325.
- [52] M.S. Westerterp-Plantenga, W.J. Pasman, M.J.W. Yedema, N.E.G. Wijkman-Duijns, Energy intake adaptation of food intake to extreme energy densities of food by obese and non-obese women, *Eur. J. Clin. Nutr.* 50 (1996) 401–407.
- [53] M.T. Kelly, K.L. Rennie, J.M.W. Wallace, et al., Associations between the portion sizes of food groups consumed and measures of adiposity in the British National Diet and Nutrition Survey, *Br. J. Nutr.* 101 (2009) 1413–1420.
- [54] J.M. Brunstrom, P.J. Rogers, E.M. Pothos, R. Calitri, K. Tapper, Estimating everyday portion size using a 'method of constant stimuli': in a student sample, portion size is predicted by gender, dietary behaviour, and hunger, but not BMI, *Appetite* 51 (2008) 296–301.
- [55] K.J. Duffey, B.M. Popkin, Energy density, portion size, and eating occasions: contributions to increased energy intake in the USA, 1977–2006, *PLoS Med.* 8 (2011), e1001050.
- [56] D.M. Cutler, E.L. Glaeser, J.M. Shapiro, Why have Americans become more obese? *J. Econ. Perspect.* 17 (2003) 93–118.
- [57] R. Mattes, S.-Y. Tan, Snacking and energy balance in humans, in: A. Coulston, C.J. Boushey (Eds.), *Nutrition in the Prevention and Treatment of Disease*, third ed. Elsevier 2013, pp. 501–515.
- [58] M.M. Perrigie, A. Drewnowski, Wang C-Y, M.L. Neuhouser, Higher eating frequency does not decrease appetite in healthy adults, *J. Nutr.* (2015), <http://dx.doi.org/10.3945/jn.115.216978>.
- [59] C.P. Herman, J. Polivy, External cues in the control of food intake in humans: the sensory–normative distinction, *Physiol. Behav.* 94 (2008) 722–728.