Fast food consumption and increased caloric intake: a systematic review of a trajectory towards weight gain and obesity risk

R. Rosenheck

Harvard School of Public Health, 667 Huntington Avenue, Boston, MA 02115, USA

Received 12 December 2007; revised 12 February 2008; accepted 19 February 2008

Address for correspondence: R Rosenheck, Harvard School of Public Health, 667 Huntington Avenue, Boston, MA 02115, USA. E-mail: rrosenhe@hsph.harvard.edu

Summary

Consumption of fast food, which have high energy densities and glycemic loads, and expose customers to excessive portion sizes, may be greatly contributing to and escalating the rates of overweight and obesity in the USA. Whether an association exists between fast food consumption and weight gain is unclear. Sixteen studies (six cross sectional, seven prospective cohort, three experimental) meeting methodological and relevance criteria were selected for inclusion in this systematic review. While more research needs to be conducted specifically in regard to effects of fast food consumption among subpopulations such as children and adolescents, sufficient evidence exists for public health recommendations to limit fast food consumption and facilitate healthier menu selection. As the fast food industry continues to increase both domestically and abroad, the scientific findings and corresponding public health implications of the association between fast food consumption and weight are critical.

Keywords: Fast food, increased caloric intake, obesity, weight gain.

obesity reviews (2008)

Introduction

The rapid increase in obesity prevalence in the USA has baffled many public health professionals and led many to hypothesize that if trends continue, the USA will undoubtedly experience a decline in life expectancy (1,2), The 1999-2002 National Health and Nutrition Survey data estimate that 65% of US adults are either overweight or obese, which represents a 16% increase in prevalence from data obtained between 1988 and 1994 (3). Overweight is defined as a body mass index (BMI) of 25 kg m⁻² or over and obesity as a BMI of greater than 30 kg m⁻². Excessive body weight is associated with comorbidities such as hypertension, cardiovascular disease, diabetes, depression, infertility, and breast, endometrial, colon, and prostate cancers, unfortunately resulting in an estimated 300 000 excess deaths and \$100 billion per year in medical expenditures (4-8).

The acceleration of overweight and obesity prevalence has pointed to environmental factors at the root of the

epidemic rather than biological alterations. Fast food outlets have increased from about 30 000 in 1970 to more than 233 000 locations in the USA in 2004 and have been classified as the most rapidly expanding sector of the US food distribution centre (9,10). Correspondingly, money spent on out-of-home food expenditures represented 25% of total food spending in 1970 and subsequently increased to 47.5% of total food spending in 1999 and is projected to increase to 53% by 2010 (11,12). Fast food can be defined as convenience food purchased in self-service or carry-out eating venues without wait service (5). In addition to the documented increase in fast food expenditures, many aspects of fast food make it suspect to the associated increases in overweight and obesity. Specifically, fast food tend to be energy dense, poor in micronutrients, low in fibre, high in glycemic load and excessive in portion size, causing many to exceed daily energy requirements (5,13). Fast food expenditures are on the rise among the American population because of its convenience, but at what price to one's health must be investigated.

While it has been suggested that fast food consumption may be linked to weight gain and obesity, a causal relationship has been difficult to establish because of inconsistent study results. As the fast food industry and consumption continue to increase both domestically and abroad, many public health implications necessitate a scientific evaluation of the potential link between intake and weight gain. This review aims critically to examine the existing evidence investigating an association between fast food consumption and increased caloric intake leading to weight gain and obesity to facilitate future research and public health recommendations.

Materials and methods

The MEDLINE database was searched in order to identify reports published through February 2008 evaluating fast food consumption and weight gain or obesity. In order to explicate the potential link, scientific evidence generated from cross-sectional, prospective cohort and experimental studies were considered. MEDLINE search terms included 'fast food', 'increased energy intake', 'weight gain' and 'obesity' in the primary search strategy. Furthermore, MEDLINE search results were restricted to English publications and human study subjects. Additional articles were supplemented by searches of related manuscripts identified from cross-matching references on primary publications. Prospective cohort studies were also restricted to those with a duration of follow-up of at least 6 months to capture the relation between fast food consumption and weight change. Sixteen articles met the aforementioned criteria. More emphasis was placed on cross-sectional studies with more than 5000 participants, large prospective cohort studies with a long duration of follow-up and experimental intervention trials. Furthermore, metabolic studies were also considered in order to elucidate potential biological mechanisms of effect.

Exposure and outcome data were varied across numerous studies, and assessment of each was taken into consideration. Food frequency questionnaires (FFQs) and repeat measures of height and weight were considered the most advantageous measures of fast food consumption and weight gain or obesity respectively. Changes in caloric intake were also considered because such data could be extrapolated to hypothesize a change in one's weight status while holding other variables constant. No restrictions were placed on the age of study participants as many imminent public health implications are contingent on agespecific findings.

Results

The keyword search initially ascertained 470 potential publications. However, after screening each citation for the

aforementioned criteria, only 16 (six cross-sectional, seven prospective cohort, three experimental) articles remained under consideration. It is important to note that the majority of those citations that were excluded were commentaries or did not assess fast food consumption and calorie intake or weight change.

Cross-sectional studies

Findings from cross-sectional studies (Table 1) suggest discrepant associations between frequent fast food consumption, increased energy and overweight or obesity in terms of BMI. Of the six studies included, two were conducted with children (14,15), one with both parents and children (16), and three with an adult population (10,17,18). Of the studies conducted among children, Shanthy et al. found a statistically significant association between fast food consumption and increased energy intake of 187 calories per day of those children who regularly consumed fast food compared with those who did not. Conversely, French et al. observed a significantly positive relationship between fast food consumption and energy and fat intake among 4344 US students. While there was no significant association between fast food consumption and BMI in females, males who consumed fast food three or more times per week had significantly lower BMI compared with males who frequented fast food restaurants two or fewer times per week.

The evaluation conducted by Boutelle *et al.* among 902 middle and high school adolescents and their parents assessed fast food intake for family meals and the participants' associated weight status. The authors determined that parents who reported purchasing fast food for family meals at least three times per week had higher mean BMIs (P < 0.01) and were more likely to be overweight (P < 0.01) than parents who reported less frequent fast food purchases. However, no significant association was found between frequency of fast food for family meals and weight status or BMI of children residing in those households.

Of the studies conducted among adults, Jeffery *et al.* reported a significant positive relationship between BMI and individuals who frequented fast food restaurants once a week or more. In a subsequent study, a positively significant association between fast food consumption and BMI was observed only among women. Lastly, a cross-sectional investigation among 1092 garage employees found no statistically significant relationship when assessing fast food consumption and BMI.

Prospective cohort studies

Results and descriptive characteristics from prospective cohort studies are shown in Table 2. Of the seven studies, four were conducted among children (19) and adolescents (20) (21,22), while young adults (5,23,24) were the study

Reference	Population	Age range	Outcome	Fast food	Results	Adjusted variables
Bowman <i>et al.</i> (14)	6212 children and adolescents; United States	-4 6	Total energy consumption	Two 24-h recall surveys on two non-consecutive days, 3–10 days apart. 30.3% of total sample reported fast food consumption on a typical day	Children who ate fast food, compared with those who did not, consumed more total energy (187 kcal; 95% CI: 109–265), more energy per gram of food (0.29 kcal g ⁻¹), more total fat (9 g), more added sugars (26 g), more added sugars (26 g), more sugar-sweetened beverages (228 g), less fibre (-1.1 g), less milk (-65 g), and fewer	Socioeconomic and demographic variables (i.e. income, race, ethnicity, age, and sex)
Jeffery <i>et al.</i> (10)	1033 Minnesota residents; United States	18-76	Self-report of height and weight used to calculate BMI	Self-report of frequency of eating at 'fast food' restaurants	Those who frequencies (~43 g) Those who frequented a fast food restaurant greater than or equal to one time per week had a BMI beta coefficient of 0.301 compared with those who never frequented fast food restaurants (<i>P</i> = 0.02)	Age, education, gender
French <i>et al.</i> (15)	4344 students; United States	5 1 1 8	Measured height and weight used to calculate BMI	Self-report of frequency of fast food restaurant use (FFFRU) with semiquantitative FFQ	Overweight and BMI status was not statistically associated with FFFRU. BMI was significantly lower among males who frequented fast food restaurants three or more times per week, compared with those reporting less frequent usage	Grade, race, school and socioeconomic status

© 2008 The	Author				
Journal con	pilation © 2008	The International	I Association for	the Study of C	besity. obesity reviews

Table 1 Cross-sectional studies

lable 1 Continued						
Reference	Population	Age range (years)	Outcome	Fast food	Results	Adjusted variables
Boutelle <i>et al.</i> (16)	902 middle and high school adolescents and their parents; United States	۲ Z	Parent height and weight self-reported over telephone. Student measured height and weight used to calculate BMI	Self-report of fast food purchases for family meals	Parents who reported purchasing fast food for family meals at least three times per week had higher mean BMI ($P < 0.01$) and were more likely to be overweight ($P < 0.01$) parents who reported less frequent fast food purchases. than No significant associations found between frequency of fast food for family meals and adolescent BMI or weight status	Race/ethnicity
Jeffery <i>et al.</i> (17)	198 men and 529 high-income women and 323 low-income women	20-45	Measured Height and Weight to calculate BMI	Self-report of fast food frequency consumption	In women, fast food eating was positively associated with BMI. $\beta = 0.39$ (0.15, 0.64) for high-income women, and $\beta = 0.85$ (0.43, 1.27) for low-income women. In men, no statistically significant association found [$\beta = -0.10$ (-0.43, 0.23)]	Age, education, smoking, total calories, percentage of fat and physical activity
French <i>et al.</i> (18)	1092 garage employees; United States	47.6 (10.2)	Measured height and weight used to calculate BMI	Self-report food frequency questionnaire	No relationship seen among fast food frequency consumption and BMI (not statistically significant)	Age, gender, education, income, marital status and race
BMI, body mass index.	×					

Table 2 Prospective cohort studies	hort studies					
Reference	Population	Age range (years)	Duration of follow-up	Fast food	Results as predictors of weight change	Adjusted variables
Schmidt <i>et al.</i> (19)	2379 White and African-American girls, United States	0 0	10 years	Self-report of fast food frequency consumption. Three-day food records were also utilized for nutrient assessment	Fast food intake was positively associated with intake of energy and total fat and saturated fat as percentage of calories. Fast food intake increased with increasing consumption of fast food, energy intake increased of 1837 kcal for the low fast food fast food for the highest fast food for the highest fast food for the highest fast food frequency group ($P < 0.05$)	Zone
Pereira <i>et al.</i> (5)	3031 African-American and White young adults; United States	18-30	15 years	Self-report of frequency of fast food consumption	After adjustment for lifestyle factors, baseline fast food frequency was directly associated with changes in body weight for both African Americans ($P = 0.005$) and Whites ($P = 0.0013$). Comparing the average 15 years weight gain in participants with frequent fast food consumption (more than twice per week) gained an extra 4.5 kg of body weight ($P = 0.0054$)	Age, sex, education, study centre, baseline body weight, height, smoking status, alcohol intake, television viewing physical activity, dietary intake
Duffey <i>et al.</i> (23)	3394 young adults; United States	18-30	3 years	Self-report of frequency of fast food consumption	 40% of the sample increased their weekly consumption of restaurant or fast food (<i>P</i> < 0.0001). Higher consumption of fast food at baseline was associated with a 0.16-unit increase in BMI at follow-up 	Race, sex, age, study centre, education, income, physical activity, total calories, smoking and marital status

© 2008 The Author		
Journal compilation © 200	8 The International Association for the Study of Obesi	ty. obesity reviews

Reference	Population	Age range (years)	Duration of follow-up	Fast food	Results as predictors of weight change	Adjusted variables
Nelson <i>et al.</i> (20)	4524 adolescent siblings sharing households for greater than 10 years were followed into adulthood	6 1- 8	10 years	Self-report of frequency of fast food consumption	Twins living apart had statistically significant changes in BMI gain. A non-significant correlation was seen among fast food consumption. Adolescent household environment accounted for 8–10% of variation in adolescent fast food intake and sedentary behaviours and 50% variation in adolescent overweight. Young adult fast food intake was significantly affected by young adult household environment, accounting for 12% variation	Age and sex
Niëmeiër <i>et al.</i> (21)	9919 adolescents; United States	18–27	5 years	Self-report of frequency of fast food consumption	Greater days of fast food consumption at Wave II (adolescence) predicted increased Zbmi scores at Wave III (young adulthood) For each additional day of fast food consumption at Wave II, ZBMI was non-significantly predicted to increase by 0.02 by Wave III	Race/ethnicity, month of interview, parental education, physical activity, sedentary behaviour, change in sedentary behaviour from Wave II to III
Thompson <i>et al.</i> (22)	101 healthy girls; United States	8-12	Varied (4-10 years)	Self-report of quick service food consumption	Those who ate quick service food twice a week or more at baseline experienced the highest increase in mean BMI z-score compared with those who ate quick service food once a week or not at all	Baseline BMI z-score
Bes-Rastrollo <i>et al.</i> (24)	7194 Mediterranean men and women; Spain	Mean age of 41	Median follow-up for 28.5 months	Self-report of consumption of hamburgers, pizza and sausages	Consumption of hamburgers, pizza, and sausages was independently associated with weight gain. Those who were in the fifth quintile of consumption compared with those in the first had an adjusted odds ratio of 1.2 (p for trend = 0.05) for weight gain	Age, sex, total energy intake from non-fast food sources, fibre, alcohol intake, leisure-time physical activity, smoking status, snacking, television watching, baseline weight and weight gain of ~3 kg in the past 5 years
BMI, body mass index.						

Table 2 Continued

participants in the remaining three studies. Six of the seven prospective cohort studies found a positive association between more frequent fast food consumption and an increase in total caloric intake or BMI.

In the studies conducted with youth, authors Schmidt and Niemeier found that fast food consumption increased with age and that those who consumed more fast food had a significantly higher caloric intake and BMI z-score, respectively, than those who did not consume fast food often. Schmidt et al. stratified fast food consumption by race and determined that, on average, African-American girls consumed more fast food and consequently more calories, total fat, saturated fat and sodium than White girls. In contrast, Nelson et al. performed a natural experiment in order to disaggregate the effects of genetics vs. environment by comparing siblings with shared households in childhood and adolescence and following them over 10 years. The authors demonstrated that compared with twin siblings living together, twins living apart exhibited greater discordance in BMI change, physical activity and fast food consumption from adolescence into adulthood. Furthermore, adolescent household environment accounted for 8% of fast food intake and 50% of variation in adolescent overweight, when combined with physical inactivity. Thus, this research did not indicate a clear association between fast food intake and change in weight status, but did highlight the influence of physical and household environments on BMI.

The three studies conducted among young adults all documented a direct link between fast food consumption and increases in BMI. Pereira et al. enrolled 3031 Black and White young adults aged 18-30 years and followed them over 15 years. After adjusting for lifestyle factors, the authors concluded that baseline fast food frequency was directly associated with changes in body weight for both African Americans (P = 0.0050) and Whites (P = 0.0013). Furthermore, compared with the average 15-year weight gain in participants with infrequent fast food restaurant use, defined as less than once per week, those with frequent fast food restaurant use or consumption of more than twice per week gained an extra 4.5 kg of body weight (P = 0.0083). In addition, those with frequent fast food restaurant use also had a twofold greater increase in insulin resistance (P = 0.0083) when compared with infrequent users. Similarly, Duffey et al. followed 3394 young adults over 3 years. During the study, 40% of the sample increased their weekly fast food restaurant use or consumption (P = 0.0001) and those who reported having a higher fast food consumption at baseline, on average, had a 0.16unit BMI increase at follow-up, holding all other variables constant. Furthermore, for every one unit increase in fast food consumption, defined as one fast food restaurant visit increase per week, an associated BMI increase of 0.0488 (P = 0.016) resulted in year 3. Bes-Rastrollo enrolled 7194 individuals, who were followed for a median of 28.5 months with mailed questionnaires, and used consumption of hamburgers, pizza and sausages as a proxy for fast food consumption. An odds ratio of 1.2 (p for trend = 0.05) for weight gain was observed when those in the fifth quintile were compared with those in the first quintile of fast food consumption.

Experimental studies

Conclusions and descriptive characteristics of all three experimental trials are shown in Table 3. Two of three trials were conducted among adolescent participants (25,26), while one enrolled adult women 20–45 years of age (27). All three experimental studies consistently documented that fast food consumption was associated with an increase in total energy intake. Only one study had a sufficient follow-up time of 3 years in order to evaluate a change in weight status with increased fast food consumption.

The studies conducted among adolescents did not directly support the hypothesis that an increase in fast food consumption leads to an associated increase in weight. However, both studies did observe an increase in total energy intake from fast food consumption among overweight or obese study participants. Ebbeling et al. conducted a feeding trial with unlimited fast food among 26 overweight and 28 lean adolescents 13-17 years of age where mean energy intake among all study participants was 1652 kcal. When stratified by weight status, overweight adolescents statistically consumed more calories than lean participants, with 1860 and 1458 kcal respectively (P =0.02). In a corresponding study, the authors analysed the difference in caloric intake among overweight and lean participants on days when they consumed fast food vs. days when no fast food consumption occurred. Overweight subjects consumed 409 kcal more on fast food days than non-fast food days (P = 0.02) while no statistically significant difference in calorie intake was noticed among their lean counterparts (P = 0.76). Furthermore, no interaction between type of day and weight status was determined with regard to physical activity (P = 0.46). In a subsequent crossover study conducted among overweight and obese adolescents, altering portion sizes and eating rates was hypothesized to affect energy intake during an extra-large fast food meal. Energy intake was not significantly different depending on the condition. However, adolescents still consumed approximately 50% of their energy needs, suggesting that fast food consumption promotes excess energy intake. Thus, both studies consistently demonstrate that overweight or obese individuals consume more total energy during fast food consumption.

French *et al.* performed a randomized intervention trial on weight gain prevention on 891 women aged 20–45 years and measured fast food intake as a secondary

Reference	Population	Age range (years)	Design	Intervention and main outcome measures	Duration	Results
French <i>et al.</i> (27)	891 women, United States	20-45	RCT	Mail-based intervention; monthly mailed newsletters with return postcards and periodic opportunities to participate in eating and exercise programmes; fast food intake and weight status	3 years	Increase in frequency of fast food was associated with increase in total energy intake, percentage of energy from fat, and body weight. An increase of one fast food meal per week, on average, was associated with a weight gain of 0.72 kg over the 3-year period ($P = 0.01$)
Ebbeling <i>et al.</i> (25)	Overweight (<i>n</i> = 26) and lean (<i>n</i> = 28) adolescents; United States	13-17	Feeding trial	Study 1: fast food meal served to each participant; average consumption Study 2: energy intake assessed in free-living conditions during days of fast food consumption and no consumption	Study 1: 1 day Study 2: 4 days	Study 1: mean energy intake from fast food meal was 1652 kcal among all participants. Overweight subjects ate more than lean participants (1860 vs. 1458 kcal p = 0.02) Study 2: overweight subjects consumed significantly more total energy on fast food days vs. non-fast food days (2703 vs. 2295 <i>P</i> = 0.02), a trend not observed amond lean participants
Ebbeling <i>et al.</i> (26)	18 overweight/obese adolescents; United States	13–17	Crossover	Randomly assigned to sequence of three feeding conditions; fast food meal present as one large serving, fast food meal portioned into four smaller servings at 15-min intervals; fast food meals portioned into four servings served at once; energy intake	3 days	Energy intake during a fast food meal was not affected by portioning or eating rate. Adolescents consumed approximately 50% of energy needs

Table 3 Experimental studies

RCT, randomized controlled trial.

analysis. Women were randomized either to a no-contact control group or to a mail-based intervention group receiving monthly newsletters and periodic opportunities to participate in eating and exercise programmes. Intervention continued for 3 years with no significant differences between treatment and control groups on fast food restaurant use (P < 0.15). Increases in frequency of fast food restaurant use were associated with increases in total energy intake, percentage of energy from fat and body weight. An increase of one fast food meal per week was associated with, on average, an increase of 234.4 kJ day⁻¹ (P = 0.01), 0.6% in fat energy day⁻¹ (P = 0.01) and a weight gain of 0.72 kg (P = 0.01) over the 3-year study period.

Discussion

Sixteen studies were included in this systematic review. Specifically, six of the 13 were cross-sectional, seven prospective cohort and three experimental. Of the six crosssectional studies, four documented a positive association between fast food consumption and increased calorie consumption or BMI. The three cross-sectional studies that included children as study subjects demonstrated that while there was an increase in total energy intake, fast food consumption did not result in a corresponding increase in BMI. This could be due to the fact that children are still growing and as a result have increased energy demands. However, such behavioural patterns could put these youth at risk of obesity, when their energy needs are reduced during their early adulthood (15). Furthermore, three prospective cohort studies revealed that fast food consumption increased with increasing age (19,23,21). Powell et al. monitored trends in food advertisements that children, aged 2-11 years, were exposed to during popular television viewing times. While increasing trends in obesity did not parallel increasing food advertisements, the authors did note a recent (2000-05) upward trend in the amount of television food advertisements that were viewed by children in the USA (28). In addition, Robinson et al. conducted an experimental study in order to determine the effects of marketing on young children's taste preferences. Sixtythree children between 3 and 5 years of age tasted identical pairs of foods and beverages, with the only difference being the presence or absence of McDonald's packaging. There was a significant difference among preference if the children perceived the item was from McDonald's (29). Thus, advertising and branding did affect children's taste preferences and when considered in conjunction with trends in fast food consumption, cogent arguments for regulation of fast food advertisements to children result.

Six of the seven prospective cohort studies showed very clear associations with increased fast food consumption and associated caloric intake leading to weight gain. The sibling prospective cohort study conducted by Nelson *et al.* was the one prospective study with discordant results. It is important to emphasize that the aim of this study was not to document the relationship between fast food consumption and weight status, but to investigate the effect of genetics vs. environment on BMI. While this study is limited in scope, it does in fact strengthen the argument that biological factors are not exclusively responsible for the increased rates of obesity and that one's physical environment needs to be taken into consideration. Thus, as fast food restaurants continue to increase both domestically and abroad, recommendations concerning dietary patterns and usage need to be generated.

Findings from observational studies as yet are unable to demonstrate a causal link between fast food consumption and weight gain or obesity. Experimental studies conducted thus far have generated very interesting results and left much to be desired. The feeding trial conducted by Ebbeling et al. among 26 overweight and 28 lean adolescents documented that regardless of body type, individuals experienced increased caloric intake. While the effects of overconsumption were much more pronounced among overweight adolescents, these participants were additionally less likely to compensate for the excess calories by adjusting their caloric intake appropriately as was witnessed among the lean participants. It is difficult to ascertain the true relationship between fast food consumption and weight gain or obesity, as many confounding factors such as physical inactivity and less inhibited food consumption are independently associated with both fast food consumption and overweight or obesity. Thus, while many ethical considerations may impede a randomized controlled trial (RCT) with the duration and compliance necessary, residual confounding from immeasurable lifestyle choices will always distort results garnered from observational study designs.

Results from this systematic review have begun to elucidate a positive link between fast food consumption and risk of weight gain or obesity. Further research suggests that exposure to fast food outlets and the associated risks are not equally distributed within society. Baker et al. conducted a spatial clustering analysis in St. Louis, Missouri, in order to examine how food availability affects dietary choices. When taking fast food restaurants and supermarkets into consideration, the study determined that fast food restaurants and consequently an absence of supermarkets were most likely to be found in areas of mixed-race or with high White poverty and where African Americans dwelled (30). Furthermore, Kwate performed an ethnographic study that found consistent results of increased exposure to fast food outlets among African-American neighbourhoods (31). However, in a cross-sectional study of 15 358 subjects between 1998 and 2002, Lopez found divergent results. While environmental influences proved predictive of obesity risk, the presence of fast food establishments near one's residence was not associated with obesity (32). In order to explain such findings, the author posited that fast food establishments near one's residence may not be predictive because nearly every geographical area possessed at least one fast food restaurant. While patrons may be willing to drive to the restaurant, the proximity of the establishment to one's place of employment could be more predictive and other methodological limitations are more important to take into consideration. Primarily, all the aforementioned studies are cross-sectional by design and thus largely subject to reverse causation. In a study conducted among executives at major US restaurant franchises, growing sales and increasing profits were noted as the most important considerations when discussing menu items, and demands for healthier menu options were believed to be not ubiquitous among the general public (33). While results remain inconclusive and studies thus far do not exclude the possibility of an endogenous relationship, more research must be conducted in order to determine if exposure to fast food restaurants vary by race and socioeconomic status and contribute to increased rates of obesity as such differentials could further widen health disparities.

In summary, findings from this review suggest that while a causal relationship cannot be stated, an unequivocal association exists between increased fast food consumption and increased caloric intake making individuals much more susceptible to weight gain and obesity. Furthermore, in a recent publication by the World Cancer Research Fund and American Institute for Cancer Research, the expert panel found the current literature regarding fast food consumption as a cause of weight gain, overweight and obesity strong and consistent, resulting in recommendations for minimal fast food consumption (2). While a plausible biological mechanism is present, many findings are subject to interpretation bias as many methodological issues such as small sample size, short duration of follow-up, inconsistency and repeatability of measurement of exposure variables, in addition to inherent confounding by other lifestyle modifications, are present among the published manuscripts.

Biological mechanisms

In order for a causal link between fast food consumption and weight gain or obesity to be demonstrated, a plausible biological mechanism must exist, guided by criteria set forth by Hill (34). Many plausible biological mechanisms, such as large portion size (35,36), high glycemic load (37), excessive amounts of refined starch and added sugars (37), have been proposed linking fast food consumption with obesity. However, the most cogent and comprehensive hypothesis is the energy density of fast food, which leads to increased caloric intake and subsequently to weight gain or obesity (25,38,13). Energy density is defined as 'the energy content per unit weight of food' (13,38). Data collected on the average energy densities for three fast food outlets were 1167, 1087 and 1054 kJ 100 g⁻¹, with typical individual fast food items possessing energy densities in excess of 900 kJ 100 g⁻¹ (38). Thus, given such high energy densities per item, it is very difficult for individuals to not exceed the average recommended dietary intake.

Furthermore, humans possess a weak innate ability to recognize foods that are energy dense and down-regulate the bulk of food eaten accordingly (36). In an experiment conducted by Rolls et al. when subjects were fed a diet where the energy density was tripled, total energy intake increased by 50%, indicating that while subjects did compensate for the increase in energy density, such compensation was insufficient as total caloric intake still increased substantially (39). Furthermore, the ability of energy dense food to interfere with appetite regulation may be exacerbated in obese individuals and children. As illustrated in the experimental study conducted by Ebbeling et al., obese individuals were unable to sufficiently adjust their caloric intake after a fast food meal in comparison with lean individuals (25). In addition, children are also suspected to be especially vulnerable to high energy density meals because they have not yet developed the necessary cognitive dietary restraint (38).

Sample size and duration of follow-up

Inconsistent findings among many cross-sectional, prospective cohort and experimental study designs may relate to the sample size and duration of follow-up. The crosssectional studies included in this review dramatically differed in sample size, with only the one conducted by Shanthy *et al.* enrolling more than 5000 study subjects. The fact that enrolment hovered around 1000 participants in four of the cross-sectional studies could impact the power necessary to effectively assess the association between fast food consumption and weight.

Duration of follow-up is important to consider because the outcome of weight gain or obesity is time dependent, and thus sufficient time must be allotted in order that changes in weight can be observed. Prospective cohort studies ranged in enrolment from 101 to 9919 study participants with follow-up times ranging from 2 to 15 years. Pereira *et al.* had the longest duration of follow-up of 15 years and concluded that those who consumed fast food frequently gained an extra 4.5 kg of body weight compared with those who reported infrequent consumption (5).

All three experimental studies possessed small sample sizes enrolling between 18 and 891 study subjects. While preoccupation with confounding is generally minimized in comparison with other study designs, external validity, however, is limited.

Exposure and outcome assessment methods

The dietary assessment method utilized in order to evaluate fast food consumption is also important to note because the discrepant results could be attributed to exposure assessment. The relationship between fast food consumption and weight is longitudinal and thus an FFO would be most appropriate in order to measure long-term patterns in intake (4). The methodology utilized in order to assess fast food consumption greatly varied across all studies. Four of the six cross-sectional studies assessed fast food intake with one survey question inquiring about fast food restaurant frequency. Prospective cohort studies utilized 3-day food records, FFQs, 3-day recall surveys and one survey question assessment.¹ While 24-h recalls are useful in estimating mean intakes of a population or validating FFQs, they are not a robust measurement strategy in order to estimate usual intakes of individuals because of large within-person variations in dietary intake. Studies that utilized food records do not rely on the subject's memory, and allow for direct and accurate quantification of food intake. However, investigators are limited in the sense that they require highly motivated and literate subjects. Furthermore, food records also tend to modify the eating habits of the study participant, with many subjects losing weight. While FFQs were deemed most appropriate for longitudinal analysis, it is important to note that they have inherent limitations such as lacking the detail and specificity of direct records or recalls, and potentially not providing accurate estimates of absolute nutrient intakes. The completion of FFQs involve memory, recall and cognitive estimation skills (40). In addition, studies that utilized one survey question in order to evaluate fast food consumption are limited in scope because they do not take into account the item consumed or the portion size, which could underestimate effect estimates and attenuate the results towards the null.

Outcome of increased calorie consumption, changes in BMI, or obesity were analysed by self-report or direct measure. Because ofmany under-report total calorie intake or weight, those studies that undertake direct measures were preferred. Furthermore, the most comprehensive evaluation of diet and weight change involves repeat measurements of both fast food consumption and weight in order to account for the inconsistency in fast food consumption over time and reduce between-person variability in weight change. All seven prospective cohort studies utilized repeat measurements of fast food consumption and five studies also calculated BMI with standardized, repeat measurements of weight. The study conducted by Schmidt *et al.* did not assess BMI as the outcome variable but intake of calories, fat and sodium calculated from 3-day food records and food-pattern questionnaires (19). While repeat measurements of dietary intake and weight change are preferred, it is important to emphasize that longitudinal analysis is still vulnerable to reverse causation.

Confounding

Issues of confounding present many limitations when interpreting study results and demonstrating a potential link between fast food consumption and weight. The largest methodological limitation present among cross-sectional studies is that they are greatly subject to confounding because fast food consumption and weight are only measured at one point in time and it is difficult to ascertain whether fast food consumption is contributing to an individual's BMI or other covariates are responsible. Nevertheless, cross-sectional studies are vital in facilitating hypothesis initiation to be further tested in observational or experimental studies.

Prospective cohort studies are better equipped than cross-sectional studies to adjust for issues of confounding but are still observational in nature and thus vulnerable to confounding as well. RCTs have been deemed the 'gold standard' when assessing issues of causality due to the fact that given sufficient compliance, all measured and unmeasured characteristics of study participants are equalized and potential for confounding is minimized. However, when attempting to assess the relationship between fast food consumption and weight, ethical considerations would impede the conduct of an RCT, forever preventing the demonstration of a causal relationship. Two studies conducted by Concato et al. (41), and Benson et al. (42) have questioned the assumption of RCTs as superior to observational study designs and demonstrated that results obtained from observational studies are highly consistent with those generated from RCTs.

When considering the relation between fast food consumption and weight, it is important to consider if fast food consumption is causal to weight gain or merely a proxy for other unhealthy lifestyle factors as many studies suggest that such behaviours tend to cluster (4). Thus, variables such as physical activity, television viewing, neighbourhood socioeconomic status, and overall eating patterns or total caloric intake should be included in the analysis to help minimize residual confounding. While residual confounding or unobserved heterogeneity presents a large barrier when conducting observational studies, statistical methods have been proposed in order to minimize such potential shortcomings and replace confidence in the results obtained. Zohoori and Savitz propose inserting an instrument variable not associated with the error term nor cor-

¹Questions utilized to gauge fast food consumption from survey questionnaires were similar to this found in prospective cohort study conducted by Schmidt *et al.* (22): 'How often do you eat food from a place like McDonald's, Kentucky Fried Chicken, Pizza Hut, Burger King or some other fast food restaurant?'

related with the predictor of interest in order to minimize potential confounding (43), whereas Sturmer *et al.* offer another technique of propensity score calibration (44). It is important to note that while residual confounding remains an important consideration when assessing the relationship between fast food consumption and increased energy intake leading to weight gain and obesity, Psaty *et al.* comment on the fact that there are limits to effects produced from unmeasured confounding and that results from observational studies should not be discounted because of their susceptibility to bias (45).

Conclusion

The rapid increase in obesity prevalence coupled with the expanding fast food industry has caused many to posit a potential link between the two, in addition to causing great public health concern. While speculation abounds, few large epidemiological studies have been conducted in order to prove this potentially deleterious association. In this systematic review, findings from several large crosssectional evaluations, well-powered prospective cohort studies with long follow-up and repeated measures of diet and weight, and experimental studies have provided strong evidence for the independent role of fast food consumption contributing to increased caloric intake, hastening rates of weight gain or obesity. As prevalence of childhood obesity begins to increase in addition to patterns of fast food consumption among children and adolescents, further research must be conducted to elucidate the potential association among these subpopulations.

Conflict of Interest Statement

No conflict of interest was declared.

References

1. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, Hayflick L, Butler RN, Allison DB, Ludwig DS. A potential decline in life expectancy in the United States in the 21st century. *N E J Med* 2005; **352**: 1138–1145.

2. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition Physical Activity and the Prevention of Cancer: A Global Perspective. AICR: Washington DC, 2007.

3. National Center for Health Statistics. *Prevalence of overweight and obesity among adults: United States, 1992–2002.* Center for Disease Control [WWW document]. URL http://www.cdc. gov/nchs/products/pubs/pubd/hestats/obese/obse99.htm (accessed November 2007).

4. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr*, 2006; 84: 274–288.

5. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, Ludwig DS. Fast-Food Habits, Weight Gain,

and Insulin Resistance (the CARDIA Study): 15-year prospective analysis. *The Lancet* 2005; **365**: 36–42.

6. Thompson D, Edelsberg J, Colditz GA, Oster G. Lifetime health and economic consequences obesity. *Arch Intern Med* 1999; **159**: 2177–2183.

7. Allison DB, Fontaine KR, Manson JE, Stevens J, VanItallie TB. Annual deaths attributable to obesity in the United States. *JAMA* 1999; **282**: 1530–1538.

8. Sturm R. The effects of obesity, smoking, and drinking on medical problems and costs. *Health Aff* 2002; 21: 245-253.

9. National Restaurant Association. *Quick Service Restaurant Trends.* National Restaurant Association: Washington DC, 2005. 10. Jeffery RW, Baxter J, McGuire M, Linde J. Are fast food restaurants an environmental risk factor for obesity? *Int J Behav Nutr Phys Act* 2006; **3**: 2.

11. Clauson A. Spotlight on national food spending. *Food Rev* 2000; September: 15–17.

12. National Restaurant Association. *Restaurant industry pocket factbook*. [WWW document]. URL http://www.restaurant.org/ store/C1660.html (accessed November 2007).

13. Isganaitis E, Lustig RH. Fast food, central nervous system insulin resistance, and obesity. *Arterioscler Thromb Vasc Biol*, 2005; **25**: 2451–2462.

14. Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics* 2004; **113**: 112–118.

15. French SA, Story M, Neumark-Sztainer D, Fulkerson JA, Hannan P. Fast food restaurants use among adolescents: associations with nutrient intake, food choices, and behavioral and psychosocial variables. *Int J Obes* 2001; **25**: 1823–1833.

16. Boutelle KN, Fulkerson JA, Neumark-Sztainer D, Story M, French SA. Fast food for family meals: relationships with parent and adolescent food intake, home food availability and weight status. *Public Health Nutr* 2006; **10**: 16–23.

17. Jeffery RW, French SA. Epidemic obesity in the United States: are fast foods and television viewing contributing? *Am J Public Health* 1998; 88: 277–280.

18. French SA, Harnack LJ, Toomey TL, Hannan PJ. Association between body weight, physical activity and food choices among metropolitan transit workers. *Int J Behav Nutr Phys Act* 2007; 4: 52.

19. Schmidt M, Affenito SG, Striegel-Moore R, Khoury PR, Barton B, Crawford P, Kronsberg S, Schreiber G, Obarzanek E, Daniels S. Fast-food intake and diet quality in black and white girls. *Arch Pediatr Adolesc Med* 2005; **159**: 626–631.

20. Nelson MC, Gordon-Larsen P, North KE, Adair LS. Body mass index gain, fast food, and physical activity: effects of shared environment over time. *Obesity* 2006; **14**: 701–709.

21. Niemeier HM, Raynor HA, Lloyd-Richardson EE, Rogers ML, Wing RR. Fast food consumption and breakfast skipping: predictors of weight gain from adolescence to adulthood in a nationally representative sample. *J Adolesc Health* 2006; **39**: 842–849.

22. Thompson OM, Ballew C, Resnicow K, Must A, Bandini LG, Cyr H, Dietz WH. Food purchased away from home as a predictor of change in BMI z-score among girls. *Int J Obes* 2006; 28: 282–289.

23. Duffey KJ, Gordon-Larsen P, Jacobs DR Jr, Williams OD, Popkin BM. Differential Associations of Fast Food and Restaurant Food Consumption with 3-y Change in Body Mass Index. The Coronary Artery Risk Development in Young Adults Study. *Am J Clin Nutr* 2007; **85**: 201–208.

24. Bes-Rastrollo M, Sanchez-Villegas A, Gomez-Gracia E, Martinez JA, Pajares RM, Martinez-Gonzales MA. Predictors of Weight Gain in a Mediterranean Cohort: the Seguimiento Universidad Navarra Study. *Am J Clin Nutr* 2006; **83**: 362–370.

25. Ebbeling CB, Sinclair KB, Pereira MA, Garcia-Lago E, Feldman HA, Ludwig DS. Compensation for energy intake from last fast food among overweight and lean adolescents. *JAMA* 2004; **291**: 2828–2833.

26. Ebbeling CB, Garcia-Lago E, Leidig MM, Seger-Shippee LG, Feldman HA, Ludwig DS. Altering portion sizes and eating rate to attenuate gorging during a fast food meal: effects on energy intake. *Pediatrics* 2007; **119**: 869–875.

 French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the pound of prevention study: dietary, behavioral, and demographic correlates. *Int J Obes* 2000; 24: 1353–1359.
 Powell LM, Szcypka G, Chaloupka FJ. Exposure to food advertising on television among US children. *Arch Pediatr Adolesc Med* 2007; 161: 553–560.

29. Robinson TN, Borzekowski DL, Matheson DM, Kraemer HC. Effects of fast food branding on young children's taste preferences. *Arch Pediatr Adolesc Med* 2007; **161**: 792–797.

30. Baker EA, Schootman M, Barnidge E, Kelly C. The role of race and poverty in access to foods that enable individuals to adhere to dietary guidelines. *Prev Chronic Dis Public Health Res, Pract, Policy* 2006; **3**: A76.

31. Kwate NO. Fried chicken and fresh apples: racial segregation as a fundamental cause of fast food density in black neighborhoods. *Health and Place* 2007.

32. Lopez RP. Neighborhood risk factors for obesity. *Obesity* 2007; 15: 2111–2119.

33. Glanz K, Resnicow K, Seymour J, Hoy K, Stewart H, Lyons M, Goldberg J. How major restaurant chains plan their menus: the role of profit, demand, and health. *Am J Prev Med* 2007; 32: 383–388.
34. Morabia A. On the origin of Hill's causal criteria. *Epidemiology* 1991; 2: 367–369.

35. Fisher JO, Rolls BJ, Birch LL. Children's bite size and intake of an entrée greater with large portions than with age-appropriate or self-selected portions. *Am J Clin Nutr* 2003; 77: 1164–1170.

36. Stender S, Dyerberg J, Astrup A. Fast food: unfriendly and unhealthy. Int J Obes 2007; 31: 887–890.

37. Ludwig DS. The gylcemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 2002; **287**: 2414–2423.

38. Prentice AM, Jebb SA. Fast foods, energy density and obesity: a possible mechanistic link. *Obes Rev* 2003; **4**: 187–194.

39. Rolls BJ, Bell EA, Castellanos VH, Chow M, Pelkman CL, Thorwart ML. Energy density but not fat content of foods affected energy intake in lean and obese women. *Am J Clin Nutr* 1999; 69: 863–871.

40. Hu FB. Obesity Epidemiology: Methods and Applications. Chapter 6: Assessment of Energy and Dietary Intake. Oxford University Press: New York, 2008 (Forthcoming 2008).

41. Concato J, Shah N, Horwitz RI. Randomized, controlled trials, observational studies, and the hierarchy of research designs. *N E J Med* 2000; **342**: 1887–1892.

42. Benson K, Hartz AJ. A comparison of observational studies and randomized, controlled trials. *N E J Med* 2000; **342**: 1878–1886.

43. Zohoori N, Savitz DA. Econometric approaches to epidemiologic data: relating endogeneity and unobserved heterogeneity to confounding. *Ann Epidemiol* 1997; 7: 251–257.

44. Sturmer T, Schneeweiss S, Avorn J *et al.* Adjusting effect estimates for unmeasured confounding with validation data using propensity score calibration. *Am J Epidemiol* 2005; **162**: 279– 289.

45. Psaty BM, Koepsell TD, Lin D, Weiss NS, Siscovick DS, Rosendaal FR, Pahor M, Furberg CD. Assessment and control for confounding by indication in observational studies. *J Am Geriatr Soc* 1999; 47: 749–754.