HHS Public Access

Author manuscript

Obesity (Silver Spring). Author manuscript; available in PMC 2018 July 01.

Published in final edited form as:

Obesity (Silver Spring). 2018 January ; 26(1): 11–13. doi:10.1002/oby.22073.

Did the food environment cause the obesity epidemic?

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Abstract

Several putative explanations of the obesity epidemic relate to the changing food environment. Individual dietary macronutrients have each been theorized to be the prime culprit for population obesity, but these explanations are unlikely. Rather, obesity probably resulted from changes in caloric quantity and quality of the food supply in concert with an industrialized food system that produced and marketed convenient, highly-processed foods from cheap agricultural inputs. Such foods often contain high amounts of salt, sugar, fat, and flavor additives and are engineered to have supernormal appetitive properties driving increased consumption. Ubiquitous access to convenient and inexpensive food also changed normative eating behavior, with more people snacking, eating in restaurants, and spending less time preparing meals at home. While such changes in the food environment provide a likely explanation of the obesity epidemic, definitive scientific demonstration is hindered by the difficulty experimentally isolating and manipulating important variables at the population level.

Introduction

Body size is highly heritable and genetics plays a substantial role in determining individual obesity susceptibility. However, our genes have not changed appreciably over the past several decades implying that environmental changes must have caused the current obesity epidemic. Here, I describe putative explanations of obesity that focus on aspects of the food environment, including dietary macronutrient composition, energy content, and overall quality of the food supply.

Is it the protein?

The “protein leverage model” of obesity postulates that the body seeks to consume a target level of dietary protein and lowering the protein fraction of diet leads to compensatory increases in overall energy intake thereby resulting in obesity (1). A logical consequence of the protein leverage model is that providing experimental subjects with low protein diets should increase *ad libitum* energy intake to achieve the target protein level. One study, conducted by originators of the protein leverage model, found a significant short-term increase in *ad libitum* energy intake when a low protein diet was compared to a moderate protein diet (2). However, two larger and longer studies failed to reproduce this effect (3, 4). Further evidence against the protein leverage model is that the protein content of the food supply did not significantly decrease during the rise in US obesity prevalence (Figure 1A

and 1B). Thus, the protein leverage model of obesity does not seem to provide a satisfactory explanation of the obesity epidemic.

Is it the fat?

The dietary fat model of obesity postulates that increased fat consumption leads to increased overall energy intake since, compared to carbohydrate and protein that have less than half the calories per gram and are typically associated with water in food, fat has a high energy density, is less satiating, and results in a smaller post-prandial increment in energy expenditure (5, 6). Covert experimental increases in dietary fat result in higher *ad libitum* energy intake, positive energy balance, and accumulation of body fat (5, 6). Dietary fat does not substantially promote its own oxidation. Therefore, increasing fat intake results in efficient accumulation of body fat that is relatively unopposed until adipose tissue sufficiently expands such that daily lipolysis increases to elevate circulating fatty acids and balance fat oxidation with fat intake (7). Decreasing the fat content of meals leads to lower *ad libitum* energy intake and experimental overfeeding of dietary fat results in greater increases in body fat compared to isocaloric carbohydrate overfeeding (5, 6). Furthermore, dietary fat in the food supply has increased in absolute terms during the development of the obesity epidemic (Figure 1B).

While the above experimental observations favor the dietary fat model, ubiquitous advice to follow lower fat diets during the 1980s and 1990s did not appear to slow the progress of the US obesity epidemic. Furthermore, prescribing low-fat diets does not lead to greater weight loss when compared to other diets (8).

Is it the carbs?

The “carbohydrate-insulin model” of obesity posits that dietary carbohydrates are particularly fattening due to their propensity to elevate insulin secretion and thereby direct fat towards storage in adipose tissue and away from oxidation by metabolically active tissues. These changes in energy partitioning are postulated to decrease energy expenditure and increase hunger leading to the development of obesity (9). However, experimental tests of the carbohydrate-insulin model using isocaloric diets widely varying in carbohydrates and fat have failed to support key model predictions regarding changes in energy expenditure and body fat (9, 10). Nevertheless, it is possible that the increased carbohydrates in the food supply (Figure 1B), and particularly refined carbohydrates, contributed to the obesity epidemic by augmenting overall calorie intake.

Is it the calories?

Experimental studies in humans have demonstrated that increasing the overall amount of food available for consumption leads to increased *ad libitum* energy intake (11). Over the past several decades, per capita US food availability increased by about two to three-fold more than required to fully explain the observed population weight gain (Figure 1C), with the surplus food resulting in a progressive ~50% increase in per capita food waste (12). Globally, the increase in per capita food energy availability is positively correlated with

increased weight gain across nations and the magnitude of the increase is more than sufficient to account for the observed weight gain in 80% of nations (13). Thus, increased energy availability in the food supply is likely an important driver of the obesity epidemic.

Is it the food quality?

Focusing solely on the increased calories in the food supply masks the complexity of the simultaneous changes that occurred in the food system during the rise in population obesity prevalence (14). The increased food energy availability was driven by economic and policy influences to maximize agricultural production of cheap inputs (e.g., corn and soy) to an increasingly industrialized food system that produced and intensively marketed inexpensive, convenient, highly-processed “added value” foods. Such foods contain relatively high amounts of salt, sugar, fat, and flavor additives and are engineered to have supernormal appetitive properties thereby driving increased consumption (15). Ubiquitous and continuous access to food also influenced normative eating behaviors, such that more people snack between meals, eat in restaurants, and spend less time preparing meals at home (15). These factors likely increased calorie intake and thereby caused obesity.

Conclusion

It is difficult to imagine a definitive scientific demonstration of *the* cause of the obesity epidemic since population environmental changes are difficult to isolate and experimentally manipulate. It is easier to rule out simple explanations of obesity such as those based on individual dietary macronutrients. More plausible explanations invoke complex changes in the overall food environment and the associated alterations in normative eating behaviors. Furthermore, a confluence of multiple interrelated environmental changes apart from the food environment, such as decreased occupational physical activity, likely played important moderating roles in the development of the obesity epidemic. Disentangling the relative contributions of these environmental variables is a difficult problem, but it seems clear that the food environment is likely the primary driver of the obesity epidemic.

Acknowledgments

This research was supported by the Intramural Research Program of the NIH, National Institute of Diabetes & Digestive & Kidney Diseases.

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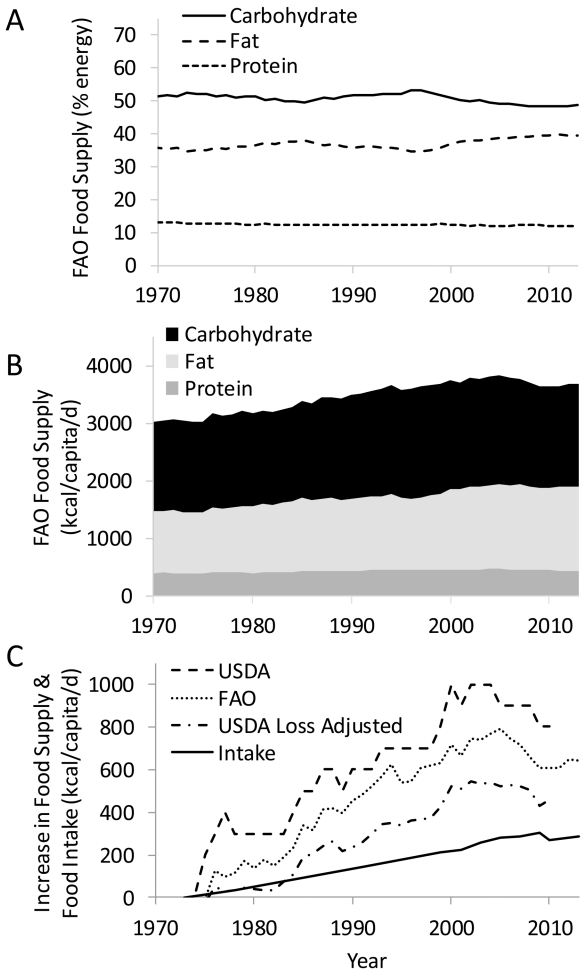

Figure 1.

A) Macronutrient proportions of the US food supply as determined by the United Nations Food and Agriculture (FAO) food balance sheets (http://www.fao.org/faostat/en/#data/FBS). B) Total per capita energy content and contributions of carbohydrate, fat, and protein in the US food supply as determined by the FAO. C) Incremental increases in food energy supply since 1973 calculated using data from the FAO, United States Department of Agriculture (USDA) food availability (https://www.ers.usda.gov/data-products/food-availability-per-capita-data-system/food-availability-documentation/), and USDA loss-adjusted food availability (https://www.ers.usda.gov/data-products/food-availability-per-capita-data-system/loss-adjusted-food-availability-documentation/), along with calculated energy intake changes corresponding to the US population weight gain (12).