Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment[1–3]

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ABSTRACT

Background: Type 2 diabetes is an epidemic that is affecting an ever-increasing proportion of the US population. Although consumption of refined carbohydrates has increased and is thought to be related to the increased risk of type 2 diabetes, the ecologic effect of changes in the quality of carbohydrates in the food supply on the risk of type 2 diabetes remains to be quantified.

Objective: The objective was to examine the correlation between consumption of refined carbohydrates and the prevalence of type 2 diabetes in the United States.

Methods: In this ecologic correlation study, the per capita nutrient consumption in the United States between 1909 and 1997 obtained from the US Department of Agriculture was compared with the prevalence of type 2 diabetes obtained from the Centers for Disease Control and Prevention.

Results: In a univariate analysis, a significant correlation with diabetes prevalence was observed for dietary fat ($r = 0.84$, $P < 0.001$), carbohydrate ($r = 0.55$, $P < 0.001$), protein ($r = 0.71$, $P < 0.001$), fiber ($r = 0.16$, $P = 0.03$), corn syrup ($r = 0.83$, $P < 0.001$), and total energy ($r = 0.75$, $P < 0.001$) intakes. In a multivariate nutrient-density model, in which total energy intake was accounted for, corn syrup was positively associated with the prevalence of type 2 diabetes ($\beta = 0.0132$, $P = 0.038$). Fiber ($\beta = -13.86$, $P < 0.01$) was negatively associated with the prevalence of type 2 diabetes. In contrast, protein ($P = 0.084$) and fat ($P = 0.79$) were not associated with the prevalence of type 2 diabetes when total energy was controlled for.

Conclusions: Increasing intakes of refined carbohydrate (corn syrup) concomitant with decreasing intakes of fiber paralleled the upward trend in the prevalence of type 2 diabetes observed in the United States during the 20th century. *Am J Clin Nutr* 2004;79:774–9.

KEY WORDS Type 2 diabetes, obesity, dietary fiber, refined carbohydrate, dietary carbohydrate, glycemic index

INTRODUCTION

Obesity and type 2 diabetes are occurring at epidemic rates in the United States (1–3). From 1935 to 1996, the prevalence of diagnosed type 2 diabetes climbed nearly 765% (4). Currently, > 16 million Americans have type 2 diabetes, one-third of whom do not even know that they have the disease (5). Recent data suggest that 47 million Americans have the metabolic syndrome—an insulin resistance syndrome that is associated with an increased risk of type 2 diabetes (6). The prevalence of adult obesity increased a staggering 57% in the brief period between 1991 and 1999, and currently > 60% of US adults are overweight (3, 7). These increases cannot be explained by the aging of the population alone, because similar increases are also being seen in US children (8).

The cause of type 2 diabetes is multifactorial. Factors such as changes in exercise patterns and the ethnic composition of the US population are likely contributors to the rising trends in diabetes, but there have been many debates in the scientific literature about the effects of specific dietary macronutrients on the risk of obesity and type 2 diabetes (9–12). Recent data suggest that a high intake of refined carbohydrates may increase the risk of insulin resistance (13–16). Although an increase in the intake of refined carbohydrates in the form of processed grains, soft drinks, sweeteners, and refined flours in the US food supply has been reported, scant quantitative data exist to determine whether such changes in dietary composition are related to the current epidemic of obesity and type 2 diabetes in the United States. To address this issue, we examined almost one century of dietary data and the history behind changes in the US diet. We conducted a multivariate analysis of the correlation between changes in the prevalence of type 2 diabetes and changes in dietary patterns in the United States.

METHODS

We obtained estimates of the prevalence of type 2 diabetes in the United States from the National Health Interview Surveys maintained by the Centers for Disease Control and Prevention's Diabetes Surveillance System (4, 17). Such data are available for 1935 and then annually beginning in 1957 and are based on self-reports of having received a diagnosis of type 2 diabetes. In 1997, adoption of the type 2 diabetes diagnostic criteria of the World Health Organization effectively increased the prevalence of type 2 diabetes in the United States after that year (18). We

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therefore excluded data from later than 1997. The diagnostic criteria for type 2 diabetes also changed in 1979 (19). However, the rate of increase in the prevalence of type 2 diabetes actually decreased somewhat from the previous 2 decades after this change in 1979, which suggested that the increase in prevalence of diabetes beyond 1979 was not an artifact of that change. Thus, these data were included in our analysis.

The prevalence of obesity was based on the measured height and weight of a random sample of the civilian noninstitutionalized population aged ≥ 20 y, and is reported as a percentage of the US population with a body mass index (BMI; kg/m^2) of ≥ 30. The data came from the National Health Examination Survey (NHES 1960–1962); the first, second, and third National Health and Nutrition Examination Surveys (NHANES I, 1971–1974; NHANES II, 1976–1980; NHANES III, 1988–1994); and NHANES 1999.

Unless stated otherwise, the nutrient content of the US food supply and other nutrition data were obtained from the National Nutrient Data Bank, which is maintained by the Center for Nutrition Policy and Promotion and the Economic Research Service of the US Department of Agriculture (20). Nutrient data are based on food disappearance and were calculated with the use of food availability data from the Economic Research Service and on the basis of the nutrient content of the edible portion of the available food supply as calculated by the Nutrient Data Laboratory of the Agricultural Research Service. These food-composition data are the numerical foundation of essentially all public and private work in the field of human nutrition (20).

Regression analysis was performed to examine the correlation between macronutrient consumption and disease rates. A multivariate nutrient-density model was used to control for total energy intake (21). In particular, corn syrup was selected to represent refined carbohydrates in the model, because it is a highly refined substance that is consumed in vast quantities in the United States in the form of soft drinks, commercial baked goods, ready-to-eat breakfast cereals, and many other commercially processed food products. Similarly, dietary fiber was selected because it generally is removed during the refining process. All *P* values are two sided. The statistical analysis was performed with the use of EPI-INFO (2002; Centers for Disease Control and Prevention, Atlanta).

RESULTS

Dietary carbohydrate steadily decreased from 500 g/d in 1909 to 374 g/d in 1963, largely because of a decrease in the consumption of whole grains. Simultaneously, dietary fiber decreased at a greater rate—by nearly 40%. Since 1963, the consumption of carbohydrates steadily increased back to 500 g/d; however, fiber consumption did not increase proportionately. This finding reflects an increased consumption of refined carbohydrates over this time period (**Figure 1**). From 1963 to 1997, the consumption of total fat increased nearly 30%, protein consumption increased 8%, and total energy consumption increased 9%.

In a univariate analysis of the available data for the period between 1909 and 1997, a significant correlation with the prevalence of type 2 diabetes was observed for intakes of dietary fat ($r = 0.84$ $P < 0.001$), carbohydrate ($r = 0.55$, $P < 0.001$), protein ($r = 0.71$, $P < 0.001$), fiber ($r = 0.16$, $P = 0.027$), corn syrup ($r = 0.83$, $P < 0.001$), and total energy ($r = 0.75$, $P < 0.001$).
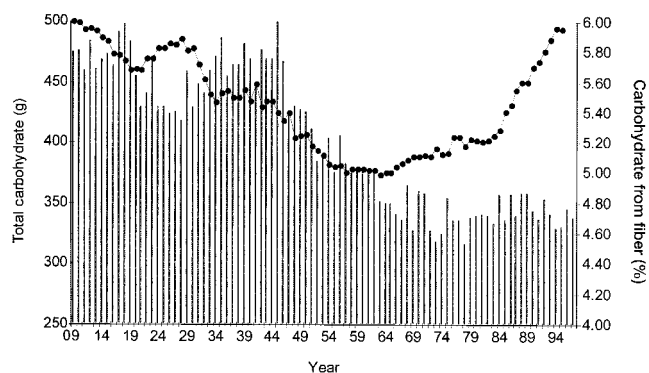

FIGURE 1. Change in total carbohydrate consumption (●) and the percentage of carbohydrate from fiber (vertical bars) in the United States between 1909 and 1997 (17).

In a multivariate nutrient-density model (**Table 1**)—in which total energy, corn syrup, fiber, fat, and protein intakes were simultaneously included—corn syrup was positively associated with the prevalence of type 2 diabetes ($\beta = 0.0132$, $P = 0.038$), whereas dietary fiber ($\beta = -13.86$, $P < 0.01$) was negatively associated with the prevalence of type 2 diabetes. In contrast, the percentages of energy from protein ($P = 0.083$) and fat ($P = 0.79$) were not associated with the prevalence of type 2 diabetes after adjustment for total energy intake and other dietary variables in the multivariate nutrient-density model.

Until 1980, the total energy intake remained relatively constant. Between 1980 and 1997, however, total energy intake increased by > 500 kcal/d. This increase was due primarily to increases in dietary carbohydrate. Specifically, 428 kcal (nearly 80% of the increase in total energy) came from carbohydrates, 64 kcal (12% of the increase in total energy) came from protein, and only 45 kcal (8% of the increase in total energy) came from fat. This represents a relative increase in consumption of dietary carbohydrates from 48% to 54% of total energy intake over a 20-y period and a relative decrease in dietary fat from 41% to 37% of total energy intake. During the same period, the prevalence of type 2 diabetes increased by 47% and the prevalence of obesity increased by 80%, indicating a significant positive correlation between the percentage of energy from refined carbohydrates and the prevalence of type 2 diabetes and obesity.

The total per capita use of caloric sweeteners increased by 86% between 1909 and 1997, and the type of sweeteners used also changed dramatically. Corn syrup sweeteners, which were almost nonexistent at the beginning of the century, now comprise

TABLE 1
Multivariate nutrient-density model for examining the associations between trends in nutrients and the prevalence of type 2 diabetes in the United States[1]

Nutrient contribution	β Coefficient	P
Dietary fiber (% of energy)	−13.86	0.0083
Corn syrup (% of energy)	0.0132	0.038
Protein (% of energy)	−3.58	0.084
Fat (% of energy)	0.00196	0.79
Total energy (kcal)	0.00011	0.28

[1] A positive β coefficient indicates an increased risk of type 2 diabetes, whereas a negative coefficient indicates a decreased risk of type 2 diabetes. All values are adjusted for each other. Energy value used for fiber = 4 kcal/g.
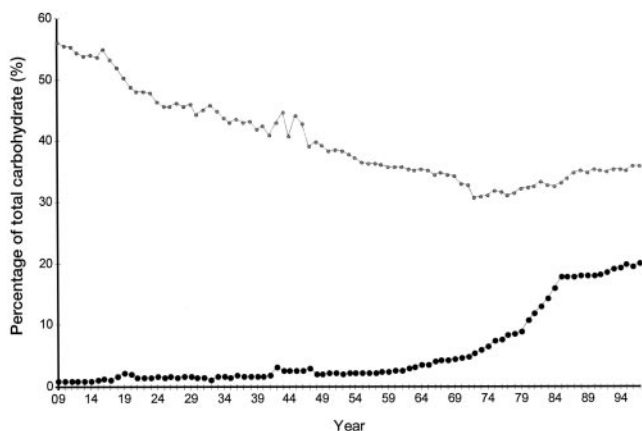
FIGURE 2. Change in total carbohydrate consumption in the United States between 1909 and 1997, reflected by the replacement of whole grains (smaller circles) with corn syrup (larger circles) (17).

> 20% of the total daily carbohydrate intake and 10% of the daily total energy intake, which represents an increase of > 2100%. These sweeteners have surpassed sucrose as the leading sweetener in the US food industry and account for much of the rebound increase in carbohydrate consumption after the mid-1960s, largely replacing the losses due to whole grains (**Figure 2**). There was a significant correlation between the percentage of carbohydrate from corn syrup and the prevalence of type 2 diabetes ($r = 0.85$, $P < 0.001$; **Figure 3**).

The multivariate nutrient-density model was modified to determine the "toxicity" of changing carbohydrate quality. This model included total carbohydrate, the percentage of carbohydrates from corn syrup, and the percentage of carbohydrates from fiber. In this model, corn syrup and fiber—potential indicators of carbohydrate quality—accounted for 18% of the variability in the prevalence of diabetes when the intake of total carbohydrate was controlled for.

DISCUSSION

In this ecologic analysis, in which national data from 1909 to 1997 were used, we found a strong association between an increased consumption of refined carbohydrates in the form of corn syrup, a decreased consumption of dietary fiber, and an increasing trend in the prevalence of type 2 diabetes in the United States during the 20th century. Furthermore, our data are consistent in that obesity and the prevalence of diabetes increased proportion-
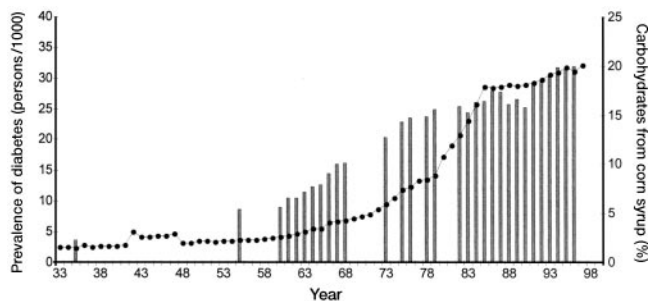

FIGURE 3. Increasing prevalence of type 2 diabetes (vertical bars) in the United States between 1933 and 1997 with increasing per capita percentage of carbohydrate intake from corn syrup (●) (14, 17).
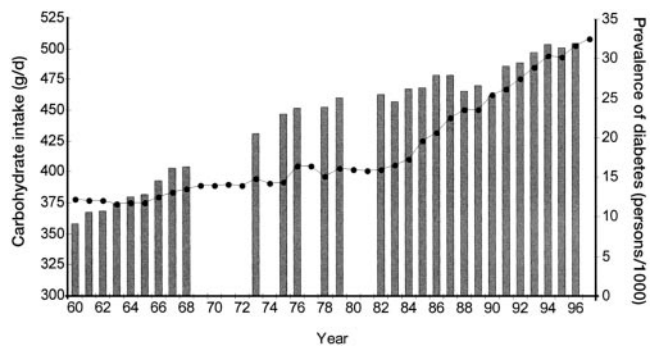

FIGURE 4. Increasing prevalence of type 2 diabetes (vertical bars) in the United States between 1960 and 1997 with increasing carbohydrate intake (●) (14, 17).

ately to the increase in consumption of refined carbohydrates in the United States (**Figures 4–6**) .

Our data also indicate that modern carbohydrates are considerably different from those consumed before the beginning of the 20th century and that the US food supply has become reliant on highly refined carbohydrates as significant sources of energy. The refining process has changed the composition and thus the quality of carbohydrates (22). For example, processing whole grains into white flour actually increases the caloric density by > 10%, reduces the amount of dietary fiber by 80%, and reduces the amount of dietary protein by almost 30% (23). Refining removes many of the main ingredients, leaving a dietary substance that is nearly pure starchy carbohydrate with fewer nutrients (24).

Corn refining in the United States began around the time of the Civil War with the development of cornstarch (25). In 1866, it was discovered that cornstarch could be converted to glucose, and by 1882 the corn industry was manufacturing "refined corn sugar." The remainder of the corn plant (fiber, germ, and protein) that was removed in the refining process was sold for animal feed or for the conversion to corn oil. Corn syrup technology advanced significantly with the introduction of enzyme-hydrolyzed products. In 1921, crystalline dextrose hydrate was introduced. The purification and crystallization of glucose meant that, for the first time, corn-based sweeteners could compete in some markets that had been the sole domain of the sugar industry. In the mid-1950s, the technology for commercially preparing low-conversion products such as maltodextrin syrup was developed. The next
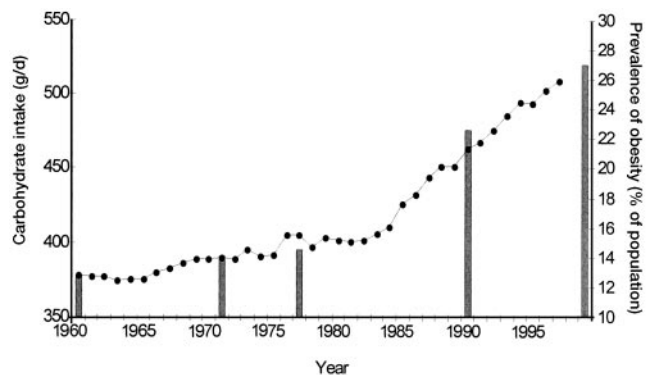

FIGURE 5. Increasing prevalence of obesity [BMI (in kg/m²) > 30; vertical bars] in the United States between 1960 and 1997 with increasing carbohydrate intake (●) (4, 17).
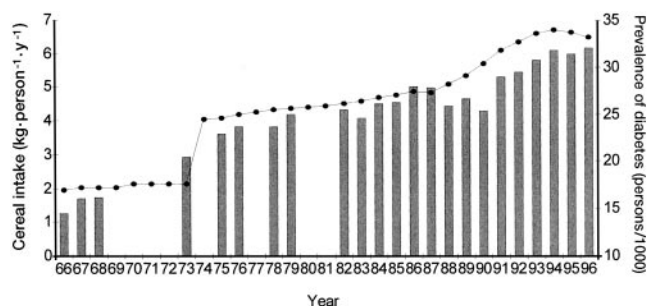
FIGURE 6. Increasing prevalence of type 2 diabetes (vertical bars) in the United States between 1966 and 1997 with increasing consumption of refined grains in the form of ready-to-eat cereals (●) (14, 17).

development involved the enzyme-catalyzed isomerization of glucose to fructose. The commercial production of high-fructose corn syrup (HFCS) began in 1967, at which time the fructose content of the syrup was ≈15%. Further research enabled the industry to develop a higher-conversion HFCS that had a fructose content of 42%. After a few more modifications, an HFCS with a fructose content of 55% became the sweetener of choice for the soft drink and ice cream industries, and an HFCS with a fructose content of 90% became a frequent choice for use in "natural" and "light" foods. By 2002, HFCS sweeteners represented > 56% of the US nutritive sweetener market.

The results of this ecologic study need to be interpreted in the context of the study's strengths and weaknesses. Ecologic studies, such as observational studies of individual persons, are susceptible to confounding. The so-called ecologic fallacy may occur when inferences are made about individual persons on the basis of solely population-level data. Our analysis may also have been limited by the use of food disappearance data at the population level that are indirectly related to intakes at individual levels. To address these issues, our analyses used only population-level variables (energy from macronutrients) to predict the only ecologic outcome (population-level prevalence of type 2 diabetes). Because we avoided inferences about individual persons from population data, no cross-level bias should occur (26, 27). Also, because we applied food disappearance data only from within the United States and did not compare regional, international, or ethnic group data, the bias, if any, would at least be uniform for the same population. Although food disappearance data are an indirect measure of individual consumption, these data have been calculated annually for more than a century, making them the only consistent data available for identifying secular trends (22).

To establish a causal diet-disease relation, however, one must examine evidence from a variety of sources and look for congruence between these sources (28). This is especially important when interpreting population-level analyses of macronutrient intakes because the specific effects of individual macronutrients and the generic effect of total energy intakes may not be evident in individual-based studies with small sample sizes (29, 30). To this end, such ecologic studies have advantages over population-based interventions for identifying potential diet-disease relations (24).

Changes in diagnostic criteria and screening practices for type 2 diabetes may limit the ability to determine the extent of effect due solely to dietary changes. To reduce this bias, we excluded data beyond 1997. Although prevalence data from the National

Health Interview Survey are self-reported, many studies have indicated excellent agreement between self-reported data and data from medical records concerning a person's diabetes status (31–35). The issue of confounding with obesity, physical activity, or both is challenging. Unfortunately, there is no uniform source of consistent information about physical activity in the United States for the period studied. The Behavioral Risk Factor Surveillance Survey has only produced such data since the 1990s (36). Similarly, the first national obesity data were from the early 1960s, which provide only 5 usable data points from the National Health Examination Survey and NHANES studies since that time. Use of such scant data to control for obesity would lack sufficient power to be meaningful. However, because obesity is likely an intermediary for the development of type 2 diabetes, control for this variable would likely negate the contribution of any macronutrient. Thus, the control for obesity may be a case of overadjustment. These potential confounders will hopefully be teased out by future prospective studies.

Classifying foods according to the physiologic effects obtained directly from metabolic experiments is useful in understanding the health effects of diets (37). Indeed, the concept that carbohydrates vary in quality is not new (ie, carrots are not the same as cake) and appears to have important clinical significance. The glycemic index (GI) is a relative in vivo measure of the plasma glucose response to a standardized amount of carbohydrate. The glycemic load (GL) is a product of the GI and the total amount of carbohydrate consumed, incorporating the effects of both the quality and quantity of carbohydrate intake. A growing body of evidence suggests that a high dietary GL increases the risk of obesity, glucose intolerance, dyslipidemia, type 2 diabetes, and coronary heart disease (38–49).

In several small-scale metabolic trials, refined grains have been shown to cause a significant increase in insulin secretion and the postprandial glucose response (50–54). In general, substituting high-fiber, low-GI foods for high-GI foods significantly improves fasting insulin concentrations, the postprandial insulin response, glycemic control, and lipid profiles (55–65). Corn syrup largely consists of the monosaccharide fructose, in contrast with sucrose—which is a disaccharide of fructose and glucose. Fructose, unlike sucrose, has been directly linked to insulin resistance in small human and animal studies and has been implicated in every metabolic abnormality associated with the metabolic syndrome (66–69). Corn syrup is now endemic in the US food supply, which places an unprecedented biochemical evolutionary pressure for processing fructose.

Several prospective cohorts have incorporated the concept of GI in assessing the relations between dietary carbohydrate and the risk of type 2 diabetes. In the Nurses' Health Study, the multivariate-adjusted relative risk of type 2 diabetes during 6 y of follow-up was 1.37 (95% CI: 1.09, 1.71) for an increase in GI of 15 units and was 1.47 (95% CI: 1.16, 1.86) for extreme quintiles of dietary GL. Women with both a high dietary GL and a low cereal fiber intake were at an even higher risk of type 2 diabetes (relative risk: 2.43; 95% CI: 1.12, 5.27) (70). In the Health Professionals' Follow-up Study, the multivariate-adjusted relative risk was 1.37 (95% CI: 1.02, 1.83) in 6 y of follow-up for extreme quintiles of dietary GL and 2.17 (95% CI: 1.04, 4.54) for the combination of a high GL and a low intake of cereal fiber (71). In the Iowa Women's Health Study, however, neither the GI nor the GL were related to the risk of type 2 diabetes in 6 y of follow-up,

although dietary fructose and glucose were significantly associated with increased risk (72).

Our analysis confirmed that during the past century, especially the past 20 y, the American diet has undergone a dramatic change. Furthermore, our data indicate that, during the same period, type 2 diabetes has reached epidemic proportions, exerting a substantial health burden on society. These population-level data are consistent with findings from metabolic and prospective studies of individual persons, which suggest that the intake of refined carbohydrates increases the risk of obesity, glucose intolerance, dyslipidemia, and type 2 diabetes. The risk of type 2 diabetes may be reduced by replacing refined carbohydrates with low-GI carbohydrate sources and whole-grain, high-fiber products. Further prospective randomized trials are necessary to determine the absolute effect of such an intervention.

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