Dietary Energy Density Is Associated With Obesity and the Metabolic Syndrome in U.S. Adults

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OBJECTIVE — Rising obesity rates have been linked to the consumption of energy-dense diets. We examined whether dietary energy density was associated with obesity and related disorders including insulin resistance and the metabolic syndrome.

RESEARCH DESIGN AND METHODS — We conducted a cross-sectional study using nationally representative data of U.S. adults ≥20 years of age from the 1999–2002 National Health and Nutrition Examination Survey (n = 9,688). Dietary energy density was calculated based on foods only. We used a series of multivariate linear regression models to determine the independent association between dietary energy density, obesity measures (BMI [in kilograms per meters squared] and waist circumference [in centimeters]), glycemia, or insulinemia. We used multivariate Poisson regression models to determine the independent association between dietary energy density and the metabolic syndrome as defined by the National Cholesterol and Education Program (Adult Treatment Panel III).

RESULTS — Dietary energy density was independently and significantly associated with higher BMI in women (β = 0.44 [95% CI 0.14–0.73]) and trended toward a significant association in men (β = 0.37 [−0.007 to 0.74], P = 0.054). Dietary energy density was associated with higher waist circumference in women (β = 1.11 [0.42–1.80]) and men (β = 1.33 [0.46–2.19]). Dietary energy density was also independently associated with elevated fasting insulin (β = 0.65 [0.18–1.12]) and the metabolic syndrome (prevalence ratio = 1.10 [95% CI 1.03–1.17]).

CONCLUSIONS — Dietary energy density is an independent predictor of obesity, elevated fasting insulin levels, and the metabolic syndrome in U.S. adults. Intervention studies to reduce dietary energy density are warranted.

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Rising rates of obesity and related disorders, such as type 2 diabetes and the metabolic syndrome, have become major public health problems in the U.S. (1–3). Studies have linked rising obesity rates to the consumption of an increasingly energy-dense diet (4,5). Energy density is the amount of energy (in kilocalories) per a given weight of food (in grams). Energy-dense foods, often high in refined grains, added sugars, and added fats (4), are palatable, inexpensive, and convenient. However, they have been associated with increased energy intakes and poor diet quality (6–8).

Decreasing dietary energy density is one strategy to stem the global obesity epidemic, and resources to decrease energy density are available for both the lay public (9) and health professionals (10). Several groups including the World Health Organization, the National Heart, Lung and Blood Institute, and the U.S. Department of Health and Human Services recommended consuming low–energy dense diets as an important strategy for obesity prevention (6,11,12). However, the links between dietary energy density and obesity rates are poorly understood. Whereas some studies have found associations between dietary energy density and BMI (4,5), others have not (13,14). A recent study that reported an association between dietary energy density and BMI used older data from 1994–1996 and relied upon self-reported rather than measured height and weight for the calculation of BMI (5). Another recent study was based on data from the 1988–1994 National Health and Nutrition Examination Survey (NHANES) III (4), which may no longer be representative of recent trends in BMI or the American diet. Also, the independent association between dietary energy density and type 2 diabetes or the metabolic syndrome has not been explored. If energy-dense diets are higher in starches and added sugars, they may contribute to the development of insulin resistance by virtue of their glycemic load (15). Additionally, energy-dense diets may contribute to insulin resistance by their higher levels of saturated fats, which have been shown to be related to impaired insulin sensitivity (16). Reducing dietary energy density might then become a viable therapeutic option not only for obesity, but also for type 2 diabetes and the metabolic syndrome.

This study tested two hypotheses using data from NHANES 1999–2002, a nationally representative survey used for disease and nutrition surveillance in the U.S. (17)—first, that energy-dense diets were independently associated with higher BMI and waist circumference on a population level and second, that energy-dense diets were independently associated with glycemia, insulinemia, and the metabolic syndrome.

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Abbreviations: NHANES, National Health and Nutrition Examination Survey.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

RESEARCH DESIGN AND METHODS

Data source and subjects

NHANES is now a continuous series of cross-sectional surveys conducted by the Centers for Disease Control and Prevention and serves as one of the key measures for Healthy People 2010 (17). We use NHANES 1999–2002, the latest fully released version of NHANES, to obtain a nationally representative sample of the U.S. noninstitutionalized civilian population through its complex, stratified, multistage, probability cluster sampling design. Dietary intake data for 1999–2001 were collected using the NHANES computer-assisted dietary interview, a multiple pass recall method (18). For 2002, dietary data were collected using the U.S. Department of Agriculture's fully computerized collection instrument, the Automated Multiple Pass Method (19). Most subjects were interviewed in person, although a small subsample was interviewed over the telephone. NHANES methods have been reported in detail elsewhere (20).

This study was reviewed and deemed exempt by the University of Washington Human Subjects Division.

For this analysis, we chose all subjects 20 years of age and older who were not pregnant.

Outcome variables

Height, weight, and waist circumference were obtained using standardized techniques and equipment (21). BMI was calculated as weight (in kilograms) divided by the square of height (in meters). Subjects were classified as normal weight (BMI $\geq$18.5 and $<$25 kg/m^2), overweight (BMI $\geq$25 and $<$30 kg/m^2), or obese (BMI $\geq$30 kg/m^2) (11).

GHb was determined by boronate-affinity high-performance liquid chromatography using Primus CLC330 and Primus CLC 3854 instruments. Fasting glucose was determined enzymatically, and fasting insulin was determined by radioimmunoassay. HDL was measured using a heparin-manganese precipitation method, and triglycerides were measured enzymatically. Blood pressure (systolic and diastolic) was measured by using a mercury sphygmomanometer following a standardized protocol based on recommendations from the American Heart Association (22).

The definition of the metabolic syndrome was based on the National Cholesterol Education Program (Adult Treatment Panel III) (23). The definition specifies three or more of the following abnormalities: *1*) abdominal obesity (waist circumference $>$102 cm in men or $>$88 cm in women); *2*) triglycerides $\geq$150 mg/dl; *3*) HDL cholesterol $<$40 mg/dl in men or $<$50 mg/dl in women; *4*) systolic blood pressure $\geq$130 mmHg, diastolic blood pressure $\geq$85 mmHg, or antihypertensive drug treatment; and *5*) fasting glucose $\geq$100 mg/dl or drug treatment for elevated glucose. The impaired fasting glucose level was modified from 110 to 100 mg/dl in order to be consistent with recently modified American Diabetes Association criteria for impaired fasting glucose as recommended by the joint American Heart Association/National Heart, Lung, and Blood Institute statement on the metabolic syndrome (24).

Main predictor

Dietary energy density was calculated by dividing each subject's daily energy intakes (in kilocalories) by the reported weight of all foods consumed (in grams). Caloric and noncaloric beverages were excluded from the calculation (25). This method of calculating dietary energy density has been shown to provide the best correlations with measures of obesity in previous analyses of the NHANES III dataset (4). Alternative methods of calculating dietary energy density, based on the inclusion of beverages, were associated with higher variance ratios, which may diminish associations when examining health outcomes (25). The inclusion of beverages, whether caloric or not, in energy density calculations may have a disproportionate influence on individual values, since beverages have a much lower energy density than foods (25).

For the regression analyses, dietary energy density values were standardized such that each unit of standardized dietary energy density represented a 1-SD change.

Covariates

The data were adjusted for several covariates that might confound the relationship between dietary energy density and outcomes of interest. Socioeconomic and demographic variables were reported as follows: *1*) sex; *2*) age, *3*) race/ethnicity categorized as non-Hispanic white, non-Hispanic black, Mexican-American, other Hispanic, and other race including multiracial; *4*) household income reported as the poverty-to-income ratio (the ratio of income to the family's appropriate poverty threshold as determined by the U.S. Census Bureau [26]), with values $<$1 below the poverty threshold, which is adjusted annually for inflation with the Consumer Price Index; poverty-to-income ratio was divided by NHANES into the following six categories: $<$1, $\geq$1$<$2, $\geq$2$<$3, $\geq$3$<$4, $\geq$4$<$5, and $\geq$5); and *5*) the highest education level achieved. We also included an age-squared term (age^2) to adjust for a nonlinear relationship between age and dietary energy density.

Physical activity was assessed by self-report during the in-person household interview using the following question, "Please tell me which of these four sentences best describes your usual daily activities? (Daily activities may include your work, housework if you are a homemaker, going to and attending classes if you are a student, and what you normally do throughout a typical day if you are a retiree or unemployed.) *1*) You sit during the day and do not walk about very much. *2*) You stand or walk about quite a lot during the day, but do not have to carry or lift things very often. *3*) You lift or carry light loads, or have to climb stairs or hills often. *4*) You do heavy work or carry heavy loads."

The mean daily total amount of food in grams was included as a covariate in the multivariate regression analyses, paralleling procedures used with nutrient density ratio variables in regression models (27).

Statistical analyses

We used bivariate analyses to assess the unadjusted relationship between dietary energy density tertiles and the outcomes. Because no standard definition for describing dietary energy density has been established, we constructed sex-specific tertile cutoffs to classify the diets of participants as having low, medium, or high dietary energy density, similar to Ledikwe et al. (5). A Wald test was used for pairwise comparison of mean values among tertiles of dietary energy density. We used a series of multivariate linear regression models, stratified by sex, to determine the independent association between standardized dietary energy density and our anthropometric measures of obesity (BMI and waist circumference as continuous variables), adjusting for food weight, age, race/ethnicity, education, income, and physical activity.

We used a similar series of multivariate linear regression models adjusting for

food weight, sex, age, race/ethnicity, education, income, physical activity, and waist circumference to determine the independent association between standardized dietary energy density and glycemia/insulinemia. Waist circumference was included as a covariate in these models because central obesity has already been shown to be an important predictor of type 2 diabetes (28,29) and is one of the major criteria for the metabolic syndrome as defined by several expert organizations such as the National Cholesterol Education Program (23).

To determine the independent association between standardized dietary energy density and the metabolic syndrome, we used a series of Poisson regression models, adjusting for food weight, sex, age, race/ethnicity, education, income, and mean physical activity. We used Poisson regression instead of logistic regression because the outcome of the metabolic syndrome is relatively common among U.S. adults, i.e., the age-adjusted prevalence was 27.0% in 1999–2000 (3).

Subjects with missing data were dropped from the multivariate regression models. Demographic differences between dropped subjects and those included in the regression models were tested by the Pearson χ^2 statistic.

We used Stata 9 for Windows (StataCorp, College Station, TX) and its survey estimation commands for complex survey data in the analyses. These commands take into account weighted observations and the probability of selection, nonresponse, and poststratification adjustments to obtain representative estimates of U.S. adults 20 years of age and older. A significance level of 0.05 was used for all analyses. We present data as means ± SE where indicated. Taylor series linearization was used to estimate SEs.

RESULTS — Table 1 shows characteristics of the participants and the distribution by sex, race/ethnicity, education, poverty-to-income ratio, and metabolic syndrome. The mean age of the sample was 47.6 ± 0.4 years for women and 45.2 ± 0.4 years for men. The unstandardized mean dietary energy density values were 1.79 ± 0.02 kcal/g for women and 1.91 ± 0.02 kcal/g for men, similar to values from NHANES III (4). The estimated percentages of overweight and obese participants were as follows: 14.5 ± 0.6% overweight woman, 16.9 ± 0.6% obese woman, 20.2 ± 0.5% overweight men, and 13.1 ± 0.4% obese men.

Table 1—*Characteristics of study subjects ≥20 years of age from NHANES 1999–2002* (n = 9,688)

	n	Estimated % ± SE
Sex		
Female	4,883	51.2 ± 0.5
Race/ethnicity		
Non-Hispanic white	4,811	71.8 ± 1.8
Non-Hispanic black	1,875	10.8 ± 1.2
Mexican-American	2,237	6.9 ± 0.8
Other race/ethnicity	765	10.4 ± 1.8
Education		
Less than high school	3,417	22.0 ± 0.8
High school	2,223	25.7 ± 1.0
More than high school	4,004	52.2 ± 1.4
Poverty-to-income ratio		
<1	1,593	14.4 ±0.8
≥1<2	2,277	21.0 ± 1.1
≥2<3	1,344	15.4 ± 0.7
≥3<4	999	13.4 ± 0.7
≥4<5	759	11.4 ± 0.5
≥5	1,586	24.4 ± 1.4
Metabolic syndrome	2,169	33.6 ± 0.9

The estimated means ± SE for the outcome measures were as follows: women's BMI 28.2 ± 0.2 kg/m^2, women's waist circumference 92.7 ± 0.5 cm, men's BMI 27.8 ± 0.1 kg/m^2, men's waist circumference 99.0 ± 0.3 cm, GHb 5.5 ± 0.02%, fasting glucose 102.1 ± 0.6 mg/dl, and fasting insulin 13.0 ± 0.4 μU/ml.

Characteristics of participants excluded (due to missing data) from the multivariate linear regression models with BMI or waist circumference as the outcome variable did not differ by sex. However, excluded participants were more likely to be poor, less educated, older, and from a minority group ($P > 0.05$). Characteristics of participants excluded (due to missing data) from the multivariate linear regression model with fasting insulin as the outcome variable were similar according to age, sex, race/ethnicity, education, and poverty-to-income ratio. Characteristics of participants excluded (due to missing data) from the multivariate Poisson regression model with the metabolic syndrome as the outcome variable were more likely to be male, poor, older, and from a minority group ($P > 0.05$).

In bivariate analyses between dietary energy density and the main outcome variables, as expected, women in the highest tertile of dietary energy density had significantly higher BMI than women

Table 2—*Measures of obesity, glycemia, and insulinemia by dietary energy density* tertiles*

	Dietary energy density tertiles†‡		
	Low	Medium	High
Women's BMI (kg/m^2)	27.7 ± 0.2[a]	28.3 ± 0.3	28.6 ± 0.2[b]
Women's waist circumference (cm)	92.1 ± 0.6	92.8 ± 0.8	93.2 ± 0.6
Men's BMI (kg/m^2)	27.7 ± 0.2	27.9 ± 0.2	28.0 ± 0.3
Men's waist circumference (cm)	98.7 ± 0.5	99.5 ± 0.6	99.2 ± 0.7
GHb (%)	5.52 ± 0.03[a]	5.48 ± 0.03	5.42 ± 0.03[b]
Fasting glucose (mg/dl)	102.5 ± 1.3	101.8 ± 0.9	100.4 ± 0.9
Fasting insulin (μU/ml)	11.2 ± 0.3[a]	12.4 ± 0.5[b]	12.6 ± 0.4[b]

Data are means ± SE. *Dietary energy density was based on food only, no beverages. †Dietary energy density tertiles for women: low, <1.45 kcal/g; medium, 1.45–1.98 kcal/g; and high, >1.99 kcal/g; and for men: low, <1.53 kcal/g; medium, 1.53–2.08 kcal/g; and high, >2.09 kcal/g. ‡A Wald test was used for pair-wise comparison of mean values among tertiles of dietary energy density; mean values with different superscript letters were significantly different, $P < 0.05$

Table 3—*Standardized dietary energy density as an independent correlate of BMI, waist circumference, glycemia/insulinemia, or the metabolic syndrome, adjusting for physical activity,* age, race/ethnicity, education, and poverty-to-income ratio*

	β	95% CI	*P*
Women's BMI (kg/m^2)†			
Standardized energy density	0.44	0.14–0.73	0.005
Physical activity 1	Ref.		
Physical activity 2	−1.58	−2.00 to −1.15	<0.001
Physical activity 3	−2.54	−3.39 to −1.68	<0.001
Physical activity 4	−2.16	−3.98 to −0.34	0.022
Men's BMI (kg/m^2)‡			
Standardized energy density	0.37	−0.007 to 0.74	0.054
Physical activity 1	Ref.		
Physical activity 2	−1.59	−2.20 to −0.99	<0.001
Physical activity 3	−2.36	−3.13 to −1.59	<0.001
Physical activity 4	−1.82	−2.63 to −1.00	<0.001
Women's waist circumference (cm)§			
Standardized energy density	1.11	0.42–1.80	0.003
Physical activity 1	Ref.		
Physical activity 2	−3.32	−4.22 to −2.42	<0.001
Physical activity 3	−5.03	−6.91 to −3.15	<0.001
Physical activity 4	−5.00	−9.61 to −0.40	0.034
Men's waist circumference (cm)‖			
Standardized energy density	1.33	0.46–2.19	0.004
Physical activity 1	Ref.		
Physical activity 2	−4.83	−6.48 to −3.19	<0.001
Physical activity 3	−6.85	−8.62 to −5.07	<0.001
Physical activity 4	−5.93	−8.17 to −3.70	<0.001
GHb (%)¶#			
Standardized energy density	0.01	−0.02 to 0.05	0.419
Waist circumference (cm)	0.013	0.012–0.014	<0.001
Physical activity 1	Ref.		
Physical activity 2	−0.01	−0.07 to 0.06	0.817
Physical activity 3	−0.02	−0.09 to 0.05	0.514
Physical activity 4	−0.06	−0.17 to 0.04	0.239
Fasting glucose (mg/dl)¶#			
Standardized energy density	0.31	−1.52 to 2.13	0.733
Waist circumference (cm)	0.36	0.29–0.43	<0.001
Physical activity 1	Ref.		
Physical activity 2	−0.64	−4.23 to 2.96	0.720
Physical activity 3	−0.09	−3.48 to 3.31	0.958
Physical activity 4	−0.85	−5.63 to 3.92	0.717
Fasting insulin (μU/ml)¶#**			
Standardized energy density	0.65	0.18–1.12	0.009
Waist circumference (cm)	0.33	0.29–0.36	<0.001
Physical activity 1	Ref.		
Physical activity 2	−1.07	−1.89 to −0.242	0.013
Physical activity 3	−1.30	−2.26 to −0.34	0.010
Physical activity 4	−2.43	−4.03 to −0.82	0.004
	Prevalence ratio	95% CI	*P*
Metabolic syndrome#††			
Standardized energy density	1.10	1.03–1.17	0.004
Physical activity 1	Ref.		
Physical activity 2	0.73	0.67–0.80	<0.001
Physical activity 3	0.67	0.57–0.79	<0.001
Physical activity 4	0.73	0.55–0.96	0.026

*For the physical activity covariate: 1 = "You sit during the day and do not walk about very much." 2 = "You stand or walk about quite a lot during the day but do not have to carry or lift things very often." 3 = "You lift or carry light loads or have to climb stairs or hills often." 4 = "You do heavy work or carry heavy loads." †n = 3,605, r^2 = 0.093 (P < 0.0001) for the women's BMI multivariate linear regression model. ‡n = 3,689, r^2 = 0.054 (P = 0.001) for the men's BMI multivariate linear regression model. §n = 3,608, r^2 = 0.1259 (P < 0.0001) for the women's waist circumference multivariate linear regression model. ‖n = 3,689, r^2 = 0.1312 (P < 0.0001) for the men's waist circumference multivariate linear regression model. ¶Waist circumference was a covariate in the regression models for GHb, fasting glucose, and fasting insulin. #Sex was a covariate in the regression models for GHb, fasting glucose, fasting insulin, and the metabolic syndrome. **n = 3,470, r^2 =0.3266 (P < 0.0001) for the fasting insulin multivariate linear regression model. ††n = 4,832, F statistic = 29.94 (P < 0.0001) for the metabolic syndrome multivariate Poisson regression model.

in the lowest tertile (Table 2; 28.6 vs. 27.7 kg/m^2, $P = 0.009$). Also as expected, participants in the medium and high tertiles of dietary energy density had significantly higher fasting insulin levels than participants in the lowest tertile (Table 2; high = 12.4, medium = 12.6, and low = 11.2 μU/ml, $P = 0.011$ and $P = 0.010$, respectively). Participants in the lowest tertile of dietary energy density had significantly higher GHb levels than participants in the highest tertile, respectively (Table 2; 5.52 vs. 5.42%, $P < 0.008$). No differences among outcome variables by tertiles of dietary energy density were noted for women's waist circumference, men's BMI or waist circumference, and fasting glucose (Table 2; $P > 0.05$).

In the multivariate linear regression models adjusting for food amount, age, race/ethnicity, education, income, and mean physical activity (Table 3), standardized dietary energy density was independently associated with higher BMI in women (β = 0.44 [95% CI 0.14–0.73]) and marginally associated with higher BMI in men (β = 0.37 [−0.007 to 0.74], $P = 0.054$). Increased standardized dietary energy density was also associated with greater waist circumference in both women (β = 1.11 [0.42–1.80]) and men (β = 1.33 [0.46–2.19]). In contrast, higher physical activity levels were associated with lower BMI and waist circumference in women and men (Table 3; $P < 0.05$).

Increased standardized dietary energy density was independently associated with elevated fasting insulin (Table 3; β = 0.65 [95% CI 0.18–1.12]) but not with GHb or fasting glucose ($P > 0.05$), adjusting for food amount, sex, age, race/ethnicity, education, income, mean physical activity, and waist circumference. Higher waist circumferences were significantly associated with elevated GHb and fasting glucose and insulin ($P < 0.001$) (Table 3). Increased physical activity was associated with lower fasting insulin ($P < 0.05$) (Table 3) but not with either measure of glycemia ($P > 0.05$) (Table 3).

Increased standardized dietary energy density was independently associated with the metabolic syndrome (prevalence ratio = 1.10 [95% CI 1.03–1.17]) (Table 3), adjusting for food weight, sex, age, race/ethnicity, education, income, and mean physical activity. In other words, with a 1-SD increase in dietary energy density, participants were 10% more likely to have the metabolic syndrome.

CONCLUSIONS — In a large, population-based survey of U.S. adults, we report that increased dietary energy density was independently associated with higher anthropometric measures of obesity, elevated fasting insulin, and metabolic syndrome. These results extend the previously observed links between energy density and measured BMI values of U.S. adults in the NHANES III dataset (4). To our knowledge, ours is the first population-based study to examine and identify dietary energy density as an independent predictor of the metabolic syndrome and insulinemia. Altogether, our results support recommendations by the World Health Organization and other authorities to decrease dietary energy density as a means to address rising rates of obesity and its related complications.

It is notable that dietary energy density was independently associated with elevated fasting insulin, independent of waist circumference and physical activity. An elevated waist circumference is strongly related to insulin resistance and is a criterion for the definition of the metabolic syndrome (23). This result, taken together with the association between increased dietary energy density and the metabolic syndrome, suggests that consumption of energy-dense diets may influence insulin resistance, independent of obesity. Several physiologic mechanisms may help to explain this finding. Higher tertiles of dietary energy density, calculated based on solid foods only, are positively linked to the content of added sugars (4). As a result, energy-dense diets with high-carbohydrate and added sugar content may pose a considerable glycemic load, which promotes insulin resistance and increases the risk of type 2 diabetes (15). Besides added sugars, dietary energy density is also positively associated with higher added fats and saturated fatty acids (4). Previous studies have positively linked higher saturated fat intake with insulin resistance (30), including a relatively large dietary intervention study (16). Altogether, these studies provide support for both potential mechanisms linking higher dietary energy density with insulin resistance, independent of central obesity, and with the metabolic syndrome.

This study has several limitations. Although the assessment of dietary intake was based on one-day only, this method's validity for assessing intakes of energy, protein, carbohydrate, and fat has been confirmed by previous research (31). Second, overweight adults tend to underestimate their food intake more than normal-weight adults (32), which would attenuate the association between dietary energy density and BMI. A third and important limitation is the cross-sectional nature of this study, which precludes drawing causal inferences regarding diet and the development of obesity, diabetes, or the metabolic syndrome. Fourth, the measure of physical activity relied on self-report and did not include leisure-time activities, which may limit its validity.

Despite these limitations, this report *1*) confirms that increased dietary energy density is associated with obesity; *2*) links increased dietary energy density to elevated fasting insulin, independent of central obesity; and *3*) is the first study to identify associations between higher dietary energy density and the metabolic syndrome in a nationally representative population of U.S. adults. These findings represent an important preliminary examination of the role of dietary energy density in obesity and related disorders. More research, including longitudinal and intervention studies, is necessary to examine the causal relationship and mechanisms linking dietary energy density and obesity. Investigators should also study the sociodemographic predictors of increased dietary energy density so that current programs and policy recommendations can be tailored to specific populations. Further studies are necessary to explore and characterize the potential role of dietary energy density in the development of the metabolic syndrome and type 2 diabetes. In the meantime, health care providers should counsel their patients to increase their intake of fruits and vegetables and decrease their intake of fat, both of which decrease dietary energy density (10) and are generally recommended by national health authorities (6,11,12).

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