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Chocolate consumption and prevalence of metabolic syndrome in the NHLBI Family Heart Study

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SUMMARY

Background & aims—Previous studies have suggested that cocoa products, which are rich sources of flavonoids, may lower blood pressure, serum cholesterol, fasting blood glucose and improve endothelial function. However, it is unclear whether consumption of cocoa products including chocolate influences the risk of metabolic syndrome (MetS).

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Conflict of interest

The authors declare no conflict of interest.

Statement of authorship

LD and OT conceived the study, carried out the statistical analyses and drafted the paper. CE, DA and SH participated in the design and coordination of this multicenter study and also helped review the manuscript. KN, JP and AK provided significant advice and insight with the analysis of the data and they helped review the draft. All authors have read and approved the final manuscript.

In a cross-sectional design, we sought to examine the association between chocolate consumption and the prevalence of MetS.

Methods—We studied 4098 participants from the National Heart, Lung, and Blood Institute (NHLBI) Family Heart Study aged 25–93 years. Chocolate consumption was assessed using a semi-quantitative food-frequency questionnaire. MetS was defined using the NCEP III criteria. Generalized estimating equations were used to estimate prevalence odds ratios of MetS according to frequency of chocolate intake.

Results—Of the 4098 participants (mean age 51.7 y) included in the analyses, 2206 (53.8%) were female. The prevalence of metabolic syndrome in our population was 30.2%. Compared with those who did not consume any chocolate, multivariate adjusted odds ratios (95% CI) for MetS were 1.26 (0.94, 1.69), 1.15 (0.85, 1.55), and 0.99 (0.66, 1.51) among women who reported chocolate consumption of 1–3 times/ month, 1–4 times/week, and 5+ times/week, respectively. Corresponding values for men were: 1.13 (0.82, 1.57), 1.02 (0.74, 1.39), and 1.21 (0.79, 1.85).

Conclusion—These data do not support an association between chocolate intake and the prevalence of MetS in US adult men and women.

Keywords

Chocolate; Metabolic syndrome; Cardiovascular disease risk

1. Introduction

National survey data from the NHANES (2003–2006) suggest that metabolic syndrome is very common, affecting about 34% of US adults who are 20–70 years of age.¹ The prevalence of the syndrome increases dramatically with age, from about 7% among people in their 20s to over 40% among people older than 60 years of age.² People with the syndrome are about twice as likely to develop cardiovascular disease (CVD) and over 4 times as likely to develop type 2 diabetes compared with subjects who do not have metabolic syndrome.^{2,3} Beyond the increased risk of developing CVD, metabolic syndrome has also been associated with an increased risk of CVD mortality.^{4–7} The persistent increase in the prevalence of obesity in the United States⁸ suggests that the current prevalence of the metabolic syndrome is now likely higher than that estimated from the NHANES (2003–2006) data. Even if the prevalence remained unchanged, the total number of people with MetS would have increased because of the US population growth.⁹ While MetS may have a genetic component, environmental factors are important modifiable risk factors for MetS.

Flavonoids are polyphenolic compounds found in many foods. Dark chocolate is an important source of flavonoids and consumption of dark chocolate is highly prevalent.¹⁰ Previous randomized trials and epidemiological studies have demonstrated that flavanols found in dark chocolate may reduce blood pressure, improve insulin sensitivity and reduce insulin resistance, reduce serum LDL and total cholesterol, and possibly increase serum HDL cholesterol concentration.^{11–15}

However, it is unclear whether consumption of chocolate is associated with MetS. Hence, this study aims to examine the association between self-reported chocolate consumption

(both dark and white) and the prevalence of MetS in an adult US population of men and women.

2. Methods and procedures

2.1. Subjects

The NHLBI Family Heart Study is a multicenter, population-based study designed to identify and evaluate genetic and non-genetic determinants of coronary heart disease (CHD), preclinical atherosclerosis, and cardiovascular disease risk factors. Families in the study had been chosen either at random (referred to as random group) or based on a higher than-expected risk of CHD (referred to as high-risk group) from previously established population-based parent studies: The Framingham Heart Study in Framingham, MA; the Atherosclerosis Risk in Communities Study cohorts in North Carolina and Minnesota; and the Utah Health Family Tree Study in Salt Lake City, UT. The high-risk group was defined based on a family risk score, which compares the family's age and sex-specific incidence of CHD to that expected in the general population.¹⁶ A detailed description of the methods and design of the study has been published.¹⁷ The evaluation included a detailed medical and lifestyle history, which was obtained through interview. All interviewers were trained centrally and required periodic certification. We obtained written consent from each participant, and the study protocol was reviewed and approved by the Institutional Review Board at each of the participating institutions.

2.2. Blood collection and assays

All participants were asked to fast for 12 h before arrival at the study center.

Serum insulin was measured by a radioimmunoassay method (Diagnostic Products Corporation, Los Angeles, CA). LDL cholesterol concentrations were measured by using the method of Friede-wald,¹⁸ except for participants with triacylglycerol concentrations >4.5 mmol/L, for whom concentrations were measured by ultracentrifugation.¹⁹ Triacylglycerol concentrations were measured by using a peroxidase-coupled method.²⁰ Total cholesterol concentrations were measured by using a commercial cholesterol oxidase method on a Roche Cobas Fara centrifugal analyzer (Boehringer Mannheim Diagnostics, Indianapolis).²¹ HDL cholesterol was measured after precipitation of the other lipoprotein fractions by using dextran sulfate.²²

2.3. Chocolate assessment

Dietary information was collected with the use of a semi-quantitative food-frequency questionnaire that was administered by the staff and developed by Dr. Willett and colleagues at the Harvard School of Public Health.^{23,24} The reproducibility and validity of the food-frequency questionnaire has been documented elsewhere.²⁴ Each subject was asked the following question: "In the past year, how often on average did you consume chocolate bars or pieces, such as Hershey's Plain, M & M, Snickers, Reeses'; 1 ounce?" (Item #39 in the questionnaire). Possible answers included > 6 per day, 4–6 per day, 2–3 per day, 1 per day, 5–6 per week, 2–4 per week, 1 per week, 1–3 per month, and almost never.

2.4. Metabolic syndrome definition

We used the National Cholesterol Education Program Adult Treatment Program III²⁵ to define MetS. Specifically, a subject was considered to have MetS if s/he had at least three of the following criteria: abdominal waist circumference (WC) >102 cm for men or >88 cm for women, fasting serum TGs of at least 150 mg/dl, serum HDL cholesterol <40 mg/dl for men or <50 mg/dl for women, average systolic blood pressure of at least 130 mmHg or diastolic blood pressure of at least 85 mmHg or current use of antihyper-tensive medication, and fasting serum glucose of at least 110 mg/dl or current use of hypoglycemic agents.

2.5. Other variables

Information on cigarette smoking was obtained by interview during the clinic visit. Oral contraceptive use and hormone replacement therapy were assessed using a reproductive history questionnaire and medication inventory. Frequency and duration of strenuous, moderate, and light physical activity during the previous year were estimated from a physical exercise questionnaire. Anthropometric data were collected with subjects wearing scrub suits. A balance scale was used to measure body weight, and height was measured using a wall-mounted vertical ruler.

Cardiovascular disease was assessed from the medical history and a 12-lead electrocardiogram. Prevalent CHD was defined as a self-reported history of myocardial infarction, percutaneous transluminal coronary angioplasty, or coronary artery bypass graft, or the presence of Q-waves on the resting 12-lead electrocardiogram. Detailed information on these covariates has been published.^{17,26,27}

2.6. Statistical analyses

Baseline characteristics were compared across categories of chocolate consumption. Within each sex, using the GENMOD procedure, we fitted generalized estimating equations to calculate odds ratios for MetS, comparing consecutive categories of chocolate consumption to the lowest category. We examined potential confounding by age, age-squared, current smoking status, educational status, prevalent coronary heart disease, physical activity, family risk score for coronary heart disease, total fat and calorie (kcal) consumption, and alcohol consumption (five categories). Secondary analyses were restricted to subjects that were randomly selected or those with prevalent CHD, diabetes, or hypertension. We used SAS for Windows version 9.3 for all analyses.

3. Results

Of the 4098 participants included in the detailed analyses, 53.8% were female, 96.4% were white and 3.6% were African-American. Of the whites, 51.6% were defined as being at high family risk for CHD and 48.4% were from the random group.

The prevalence of the metabolic syndrome was 30.2% ($n = 1238$). Nine hundred and fifty seven (77.3%) of the cases of MetS were observed among participants who had coronary heart disease or were being treated for hypertension and/or diabetes. Characteristics of the study participants by frequency of chocolate consumption are shown in Table 1. Higher

intake of chocolate was associated with younger age, lower prevalence of coronary heart disease; higher levels of calorie intake, saturated, and poly-unsaturated fat intake; and lower plasma glucose concentrations.

Chocolate consumption was not associated with MetS in this study (Table 2). From the lowest to the highest level of chocolate consumption, the prevalence odds ratios were 1.0 (ref), 1.26 (0.94, 1.69), 1.15 (0.85, 1.55), and 0.99 (0.66, 1.51) among women; and 1.0 (ref), 1.13 (0.82, 1.57), 1.02 (0.74, 1.39), and 1.21 (0.79, 1.85) among men in a model adjusting for age, age-squared, current smoking status, education, physical activity, dietary cholesterol, family risk score for CHD risk, fat and calorie consumption, and alcohol consumption (5 categories). When analyses were restricted to subjects from families that were randomly selected or people diagnosed with coronary heart disease, treated diabetes, or hypertension, similar results were observed (Table 3).

When MetS components were individually examined, we observed no significant association between chocolate consumption and high serum TG, low serum HDL, or high blood pressure, (Table 4). However, a statistically significant lower prevalence of diabetes and a higher prevalence of adiposity (measured as increased waist circumference above 102 cm for men and 88 cm for women) was observed with chocolate consumption: adjusted odds ratios (95% CI) from the lowest to the highest category of chocolate intake were 1.0 (ref), 0.65 (0.50, 0.85), 0.61 (0.47, 0.80), and 0.44 (0.30, 0.65), respectively, for diabetes and 1.0 (ref), 1.34 (1.09, 1.66), 1.27 (1.02, 1.58), and 1.74 (1.31, 2.31), respectively, for obesity.

4. Discussion

In the present study, we demonstrated that chocolate consumption was not associated with prevalence odds of MetS among men or women. We also report a significant increase in the prevalence of adiposity (increase in WC) and a significant decrease in prevalence of diabetes with habitual consumption of chocolate. The prevalence of the metabolic syndrome in this study population was 30.2% which is lower than that reported in the NHANES 2003–2006 population (34%).

To the best of our knowledge, only one previous study²⁸ has attempted to examine the association between chocolate consumption and the prevalence odds of MetS. This study examined the association between candy consumption and body weight measures, CVD risk or metabolic syndrome, using the NHANES 1999–2004 data, and reported a 15% reduced risk of MetS in chocolate candy consumers when compared with non-consumers. Our study, though, reports a null association. Their study adjusted for sex, age and ethnicity alone in the final model, this study includes other covariates – current smoking status, educational status, coronary heart disease, physical activity, dietary cholesterol, risk group, fat and calorie consumption, and alcohol consumption – which were considered as potential confounders. Secondly, the present study quantified and categorized the frequency of chocolate consumption over three different levels (<1/wk., 1–4/wk. and 5+/wk.), their study analyzed chocolate candy consumption as a dichotomous variable (yes/no). Thirdly, both studies used different ways for ascertainment of chocolate consumption.

Previous clinical trials and prospective cohort studies have explored the relationship between chocolate consumption and some of the individual components of the metabolic syndrome.^{11–14,29} Chocolate consumption has been shown to reduce blood pressure and serum LDL cholesterol levels,^{11,12} increase HDL cholesterol levels,¹⁴ increase insulin sensitivity and reduce insulin resistance in both healthy and glucose intolerant individuals.¹¹

The MetS NCEP definition used in our study includes a threshold for central obesity and serum triglyceride concentration, which are the other two components. Most studies report a null association between chocolate consumption and increased serum TG concentration. Limited published data, however, report the relationship between habitual chocolate consumption and obesity.³⁰

From completed studies on the effect of chocolate consumption on the individual MetS components, we can speculate on the directionality of effect of chocolate consumption on MetS – a significant reduction in the prevalence odds of MetS with chocolate intake. Since this was not the case, it may be suggested that the obesity component is responsible for the non-association which we observed. This view is supported by the outcome we obtained after analysis of the association between chocolate consumption and the individual MetS components (Table 4). While this analysis showed no association between chocolate intake and high average blood pressure or low HDL, we observed a trend toward **lower** odds of ‘hypertension’ and ‘low HDL’ with increasing frequency of chocolate intake. Same can be said for the diabetes component (this association was found to be highly significant) and this finding is consistent with similar studies.^{11,12} We also observed, however, significant higher odds of obesity prevalence with chocolate consumption and this is possibly driving the overall results in the null direction.

In one of the few published studies specifically exploring the topic of obesity and chocolate consumption, body weight and mesenteric white adipose tissue weight were significantly lower in rats fed with real cocoa diet than in those fed the mimetic cocoa diet,³⁰ suggesting that ingested cocoa may actually prevent high-fat diet-induced obesity by modulating lipid metabolism, especially by decreasing fatty acid synthesis and transport systems³⁰ – this, though, is at odds with our finding. This difference may be due to the wide variation in the way everyday chocolate products are manufactured and prepared before consumption – a variation that may influence the total calorie intake and ultimately, weight gain among people who consume chocolate, a thought also supported by the positive relationship between total calorie intake and chocolate consumption observed in Table 1. Unmeasured correlates of cocoa beverage ingestion (that increase calorie intake) may also exist that were not included in our analysis but could explain some of the effects we observed.

This study has some limitations. It is not possible to draw causal inference from the data due to the cross-sectional design. Because chocolate consumption was self-reported using a semi-quantitative food-frequency questionnaire, it is possible that some reporting bias may have biased the results toward the null. It might be possible that subjects already diagnosed with CHD, diabetes, or hypertension may selectively recall consuming higher quantities of chocolate. On the other hand, the inverse association observed between ‘elevated fasting plasma glucose’ and chocolate consumption as shown in Table 4 could be the result of

reverse association, because individuals who are diagnosed diabetics should and often do make alterations in their diet,³¹ including the avoidance of processed sugars as would be found in chocolate. We were also unable to assess the association specifically between dark chocolate and MetS in our study due to lack of detailed data on type of chocolate consumed. If dark but not white chocolate is effective in influencing MetS prevalence, then our inability to separate these two types of chocolate might have obscured a true association. Residual confounding by unmeasured correlates of chocolate consumption is also possible. Nevertheless, the large sample size, the multicenter design of the study (within the US), and the availability of data on numerous covariates are strengths of this paper.

In summary, this study does not provide evidence for an association between self-reported chocolate consumption and the prevalence of MetS among men and women.

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Table 1

Characteristics of 4098 subjects in the NHLBI FHS according to chocolate intake.

Characteristics	Frequency of chocolate consumption			
	0	1	2	3
		<1/wk.	1–4 times/wk.	5+ times/wk.
	<i>n</i> = 899	<i>n</i> = 970	<i>n</i> = 1632	<i>n</i> = 597
Age (y)	55.8 ± 13.1	51.9 ± 13.8	50.5 ± 13.7	48.4 ± 13.4
Females (%)	54.3	55.7	51.7	56.0
Current smokers (%)	14.6	12.7	13.0	16.6
Alcohol drinkers (%)	53.1	57.9	54.4	54.8
High-risk group	49.5	49.7	49.3	51.4
Coronary heart disease	15.0	12.8	9.4	5.4
Exercise (min/d)	36.2 ± 36.5	34.1 ± 39.5	31.1 ± 36.2	34.8 ± 41.2
Calories (kcal)	1561 ± 557	1646 ± 585	1778 ± 587	2120 ± 634
Southgate diet fiber (g/d)	19.1 ± 9.9	17.6 ± 8.2	17.4 ± 7.7	18.9 ± 8.0
Education (y)	16.0 ± 4.0	16.2 ± 3.8	16.2 ± 3.7	16.3 ± 3.6
Saturated fats (g/d)	16.6 ± 8.6	19.5 ± 9.7	22.9 ± 9.7	30.9 ± 11.3
Polyunsaturated fats g/d	7.4 ± 4.0	8.0 ± 3.8	9.2 ± 4.0	10.8 ± 4.5
LDL cholesterol (mg/dl)	123.4 ± 37.2	123.3 ± 33.7	126.0 ± 34.1	123.9 ± 35.0
HDL cholesterol (mg/dl)	52.4 ± 16.6	50.7 ± 15.4	49.8 ± 14.6	49.9 ± 14.5
Triglycerides (mg/dl)	149.2 ± 119.3	153.3 ± 123.4	144.4 ± 89.7	148.9 ± 102.0
Insulin (m/l)	13.9 ± 39.9	11.8 ± 16.2	10.9 ± 9.2	11.1 ± 8.2
Glucose (mg/dl)	105.7 ± 40.2	100.7 ± 32.9	97.5 ± 23.9	97.1 ± 31.3

Table 2

Prevalence odds ratio (95% CIs) for MetS according to chocolate intake.

Categories of chocolate consumption (/week)				
	0	<1	1-4	5+
Model 1				
	1.00	1.20 (0.97, 1.49)	1.09 (0.88, 1.35)	1.14 (0.85, 1.51)
Model 2				
Women	1.00	1.31 (0.99, 1.71)	1.13 (0.88, 1.45)	1.09 (0.77, 1.54)
Men	1.00	1.14 (0.84, 1.54)	1.05 (0.81, 1.37)	1.29 (0.90, 1.81)
Model 3				
Women	1.00	1.26 (0.94, 1.69)	1.15 (0.85, 1.55)	0.99 (0.66, 1.51)
Men	1.00	1.13 (0.82, 1.57)	1.02 (0.74, 1.39)	1.21 (0.79, 1.85)

Model 1 – Main results in general population: Model was adjusted for the following variables: age, age-squared, current smoking status, educational status, coronary heart disease, physical activity, dietary cholesterol, risk group, fat and calorie consumption, and alcohol consumption (5 categories).

Model 2 – Sex stratified, age and age-squared adjusted-logistic regression model.

Model 3 – Adjusted for variables in model 2 in addition to the following variables: current smoking status, educational status, coronary heart disease, physical activity, dietary cholesterol, risk group, fat and calorie consumption, and alcohol consumption (5 categories).

Table 3

Subgroup analyses – prevalence odds ratio (95% CIs) for metabolic syndrome according to chocolate intake restricted to the random population group, AND subjects with diagnosed coronary heart disease or treated diabetes and/or hypertension.

Frequency of chocolate consumption (per week)				
	0	<1	1–4	5+
Model 4				
Women	1.00	1.16 (0.74, 1.83)	0.94 (0.59, 1.50)	1.24 (0.68, 2.28)
Men	1.00	1.25 (0.77, 2.01)	0.99 (0.63, 1.58)	1.18 (0.62, 2.23)
Model 5				
Women	1.00	1.28 (0.94, 1.76)	1.16 (0.85, 1.59)	1.06 (0.69, 1.64)
Men	1.00	1.30 (0.88, 1.93)	1.12 (0.76, 1.64)	1.27 (0.77, 2.08)

Model 4 – Similar to model 3 but restricted to the random population group.

Model 5 – Similar to model 3 but restricted to population with coronary heart disease or those being treated for hypertension and/or diabetes.

Table 4

Prevalence odds ratio (95% CIs) for individual metabolic syndrome components according to frequency of chocolate intake. Model was adjusted for: age, age-squared, current smoking status, educational status, coronary heart disease, physical activity, dietary cholesterol, risk group, fat and calorie consumption, and alcohol consumption (5 categories).

MetS individual components	Frequency of chocolate consumption			
	0	1	2	3
		<1/wk.	1–4 times/wk.	5+ times/wk.
	<i>n</i> = 899	<i>n</i> = 970	<i>n</i> = 1632	<i>n</i> = 597
Obesity WC >88 or 102	1.00	1.34 (1.09, 1.64)	1.25 (1.01, 1.53)	1.66 (1.26, 2.19)
Triglyceride 150 mg/dl	1.00	1.15 (0.94, 1.40)	1.03 (0.84, 1.26)	1.04 (0.79, 1.36)
Hypertension	1.00	0.94 (0.75, 1.17)	0.88 (0.70, 1.11)	0.84 (0.61, 1.15)
HDL<40 mg/dl in men or <50 mg/dl in women	1.00	1.10 (0.90, 1.36)	1.08 (0.88, 1.32)	1.01 (0.78, 1.32)
Fasting plasma glucose >110 mg/dl	1.00	0.65 (0.50, 0.85)	0.61 (0.47, 0.80)	0.44 (0.30, 0.65)