Genetic effects on weight change and food intake in Swedish adult twins[1–3]

Berit L Heitmann, Jennifer R Harris, Lauren Lissner, and Nancy L Pedersen

ABSTRACT

Background: Obesity is influenced by genetic and environmental factors. Additionally, synergistic effects of genes and environments may be important in the development of obesity.

Objective: The aim of this study was to test for genetic effects on food consumption frequency, food preferences, and their interaction with subsequent weight gain.

Design: Complete data on the frequencies of consumption of 11 foods typical of the Swedish diet were available for 98 monozygotic and 176 dizygotic twin pairs aged 25–59 y who are part of the Swedish Twin Registry. The data were collected in 1973 as part of a questionnaire study. Body mass index was measured in 1973 and again in 1984.

Results: There was some evidence that genetic effects influenced the frequency of intake of some foods. Similarity among monozygotic twins exceeded that among dizygotic twins for intake of flour and grain products and fruit in men and women, intake of milk in men, and intake of vegetables and rice in women, suggesting that genes influence preferences for these foods. Analyses conducted for twins reared together and apart also suggested greater monozygotic than dizygotic correlations, but cross-twin, cross-trait correlations were all insignificant, suggesting that the genes that affect consumption frequencies are not responsible for mediating the relation between the frequency of intake and weight change.

Conclusions: Genetic effects and the frequency of intake are independently related to change in body mass index. However, there was no suggestion of differential genetic effects on weight gain that were dependent on the consumption frequency of the foods studied. *Am J Clin Nutr* 1999;69:597–602.

KEY WORDS Weight change, monozygotic twins, dizygotic twins, gene-environment interactions, food consumption, obesity, food preferences, weight gain, Swedish Twin Registry

INTRODUCTION

Twin and adoption studies have clearly shown that body mass index (BMI) and other measures of fatness are under strong genetic control (1). In addition, there is no doubt that environmental factors, such as dietary intake and physical activity, influence the development of obesity (2). For example, twin data reveal significant genetic influences on overall intake of nutrients and intake pattern (size and frequency of meals) (3). A recent review of the literature concluded that most studies report evidence for genetic effects on overall and nutrient intakes, but evidence of heritability for food preferences is less strong (4). However, as pointed out by Pérusse and Bouchard (5), low estimates of heritability for food preferences do not imply that genes are unimportant for nutritional habits. In addition to genetic influences on diet intake and obesity, there may be synergistic effects between genes and lifestyle factors (such as diet) for obesity development. For example, both diet and physical activity have been found to be related to weight gain, particularly among those with a genetic disposition to obesity (6, 7). Results based on data from Finnish twins suggest a gene-environment interaction for physical activity whereby a sedentary lifestyle may have an obesity-promoting effect among men with a genetic predisposition for obesity (6). Similarly, a familial predisposition to obesity predicted sensitivity to a high-fat diet among Swedish women (7).

Although results of several animal studies suggest that a preference for fat intake is under partial genetic influence, only a few studies have examined whether genetic differences are important sources of variance for diet preferences in humans (4). In addition, almost nothing is known regarding interactions between genetic and dietary factors. Observational studies often measure diet with use of short food-frequency questionnaires, and most food-frequency questionnaires have been shown to perform well in ranking subjects by nutrient intake (8, 9).

[1] From the Danish Epidemiology Science Centre at the Institute of Preventive Medicine, Copenhagen Hospital Corporation, Municipal Hospital of Copenhagen; the Department of Population Health Sciences, Section of Epidemiology, National Institute of Public Health, Oslo; the Division of Genetic Epidemiology, Institute for Environmental Medicine, Section for Epidemiology, The Karolinska Institute, Stockholm; and the Departments of Internal Medicine and Primary Health Care, Gothenburg University, Gothenburg, Sweden.

[2] Supported by a grant from NOS-M (project no. 9501124) to assist the Inter-Nordic collaboration and by grants from the John D and Catherine T MacArthur Foundation, the Swedish Council for the Coordination of Planning and Research, the Danish Health Insurance Foundation, and the Wedell-Wedellsborgs Foundation. The Swedish Adoption Twin Study of Aging is supported by grants from the National Institute of Aging (AG 04563, AG 10175) and the MacArthur Foundation Research Network on Successful Aging.

[3] Address reprint requests to BL Heitmann, Copenhagen County Centre for Preventive Medicine, Glostrup University Hospital, DK-2600 Glostrup, Denmark. E-mail: bette@glostruphosp.kbhamt.dk.

Received January 13, 1998.

Accepted for publication August 17, 1998.

The aim of the present study was to explore the importance of genetic effects on change in BMI and to test for gene-environment interactions with respect to BMI changes among twins. The dietary data are based on the frequency of consumption of 11 different food groups characteristic of the Swedish diet. In this context, particular foods are considered to be proxy measures for a host of attributes that may show heritable variation, including fat intake or taste. To test for gene-environment interactions it is important to establish whether the putative environmental measure (food consumption frequency) is truly environmental. Therefore, we also examined whether variation in frequency of consumption was explained solely by environmental influences.

SUBJECTS AND METHODS

Subjects

The twins studied are part of the population-based Swedish Twin Registry (10, 11). The initial twin cohort, from which this sample was drawn, comprised 17 992 same-sex pairs who were born between 1926 and 1958 and who were sent a questionnaire in 1973. Altogether, responses were received from both members of 13 664 pairs aged 26–59 y. The questionnaire included items about lifestyle, height and weight, the frequency of consumption of selected food groups, smoking habits, and level of physical activity. Within the Swedish Twin Registry, a sample of twins who had been separated early in life have been identified and characterized (12). The most common reasons for separation were illness or death of the mother, the mother being single, or economic hardships. In 1984 a new study was initiated of 591 twin pairs reared apart and a matched group (by sex, age, and county of birth) of 627 twin pairs reared together (13, 14). Questionnaire responses, which included items about height and weight, were received from 346 twin pairs reared together and 404 twin pairs reared apart. A subsample of 1198 of these twins belonged to the cohort who completed the 1973 questionnaire. The present results are based on 548 of these individuals (98 monozygotic and 176 dizygotic twin pairs) for whom there was complete pair data at baseline and follow-up for weight and height in 1984, baseline information from 1973 on smoking habits and physical activity, and a zygosity classification. Among the monozygotic twins, 28 male and 30 female pairs had been reared together and 25 male and 15 female pairs had been reared apart. For dizygotic twins the corresponding numbers were 29 male and 38 female pairs reared together and 44 male and 70 female pairs reared apart. All studies were performed in accordance with the Helsinki Declaration of 1973 as revised in 1983.

Measures

Height and weight

In both questionnaires, weight was reported in kilograms and height in centimeters. BMI (in kg/m^2) was used as a measure of relative body weight. Because we wanted to study change in weight, adjusted for height, the baseline measure of height was used to calculate BMI at both baseline and follow-up. BMI changes were then calculated as BMI at follow-up minus BMI at baseline.

Diet

The baseline questionnaire included items about the frequency of consumption of 11 different food groups: *1*) meat, *2*) sausages, *3*) organ meats and other intestinal or blood products, *4*) fish, *5*) shellfish, *6*) rice and rice dishes, *7*) flour and other grain products (including porridge, cereals, and pancakes), *8*) eggs, *9*) vegetables and root fruit, *10*) fruit, and *11*) dairy products (milk, buttermilk, yogurt, or cheese). The response format was as follows: less than once a month, once a month, a few times a month or once a week, a few times a week, and more or less daily. For statistical analyses the item codes were converted into weekly frequencies. Exploratory factor analysis with oblique rotation was conducted to determine whether the items defined specific subscales representing dietary patterns. The factor structure did not reflect meaningful consumption patterns, however, and several items were split across factors. Therefore, we decided to analyze each food item separately.

Covariates

Other baseline measures analyzed as covariates included smoking habits (never, exsmoker, or current smoker) and frequency of exercise during leisure time. The exercise measure was coded by using a 7-point Likert-type scale as follows: 1 = none, 2 = hardly any, 3 = a little, 4 = some, 5 = fairly often, 6 = often, and 7 = very often.

Statistical methods

The analyses were conducted on a double-entry file. To determine the extent to which the food consumption variables reflected environmental measures, intraclass correlations were calculated for each food type for groups defined by zygosity and sex (15). Significant differences between the monozygotic and dizygotic correlations were tested by using *z* transformations and by testing the ratio of the differences between the *z* values over the SE. Evidence of genetic effects on food consumption frequencies would be indicated if the monozygotic correlations were significantly greater than the dizygotic correlations. Accordingly, the food consumption measure could not be regarded as a pure environmental measure. Furthermore, there may have been confounding because the genetic effects that influence intake of the foods that promote weight gain may be correlated with the genetic effects on weight gain itself. To test specifically for such confounding, cross-twin, cross-trait correlations were also inspected. These correlations measured the association between food intake in one twin with change in BMI in the other twin. Greater monozygotic than dizygotic correlations here would suggest some overlap in the genetic effects that contribute to variation in both phenotypes.

Hierarchical multiple regression was used to test sequentially for the significance of the main effects, the two-way interactions, and the three-way interactions (16, 17). The hierarchical models are listed below.

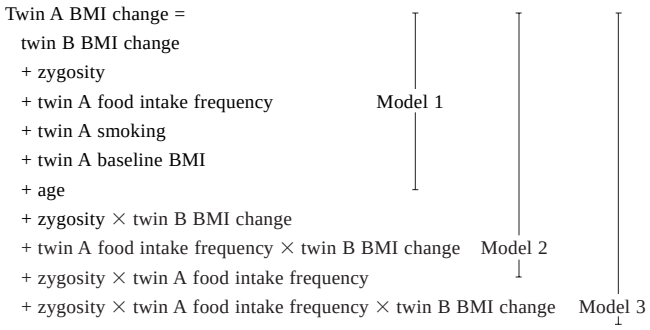

TABLE 1
Descriptive values for BMI and covariates by zygosity in twin men and women

	Men		Women	
	Monozygotic twin (n = 106)	Dizygotic twin (n = 146)	Monozygotic twin (n = 90)	Dizygotic twin (n = 206)
Age	45.2 ± 9.2[1]	45.2 ± 7.4	46.2 ± 9.1	47.1 ± 8.5
BMI in 1973	23.2 ± 3.3	23.6 ± 2.8	21.4 ± 2.5	22.3 ± 3.2
BMI in 1984	24.6 ± 3.6	24.9 ± 3.0	22.8 ± 3.1	23.4 ± 3.6
Change in BMI	1.4 ± 2.3[2]	1.3 ± 1.9[2]	1.4 ± 1.6[2]	1.2 ± 2.0[2]
Current smokers (%)	49 [52][3]	44 [64]	32 [29]	34 [70]
Physically active (%)	40 [42]	37 [54]	22 [20]	25 [51]

[1] $\bar{x}$ ± SD.
[2] Significantly different from zero, $P < 0.0001$.
[3] n in brackets.

The main effects model (model 1) analyzed the significance of food intake frequency on change in BMI after age, zygosity, and baseline measures of exercise, smoking, and BMI were adjusted for. The co-twin's change in BMI was also included and simply tested for twin similarity in weight change. Model 2 is an expansion of the main effects model and included the following two-way interactions: zygosity × co-twin's change in BMI, which represents genetic effects for weight change and is expected to be significant; diet × co-twin's change in BMI, which represents twin resemblance for weight change as a function of the food intake measure; and zygosity × diet, which merely reflects zygosity differences in food intake frequency and was not expected to be significant. This variable was included for statistical reasons because lower-order factors must be included in a regression model assessing higher-order interactions. Finally, the three-way interaction between zygosity, food intake frequency, and BMI change was added in model 3. If there was no evidence of genetic effects for the food intake measure, then this three-way interaction represented a gene-environment interaction for the effect of food intake on change in body weight. Statistical analyses were performed with SPSS PC (version 2.0; SPSS Inc, Chicago).

RESULTS

Descriptive results

Complete data were available for 126 male and 148 female twin pairs; the mean age of these pairs was 45 y in men and 47 y in women. Descriptive values for BMI at both measurement occasions, change in BMI, and baseline smoking and physical activity levels are listed by sex and zygosity in **Table 1**. Mean BMI values in men at baseline and follow-up were 23.4 ± 3.0 and 24.8 ± 3.3, respectively; the corresponding values for women were 22.0 ± 3.0 and 23.2 ± 3.5. The 11-y increases in BMI were significant in both sexes. There were no significant differences by zygosity for BMI or for change in BMI.

The consumption frequencies of the various foods in men and women and the sex differences in food selection profiles are listed in **Table 2**. Women consumed eggs, vegetables, fruit, and dairy products more frequently and sausages less frequently than did men. The correlations between weight change and the frequency of consumption of each food were insignificant and ranged from −0.09 to 0.11 in men and from −0.07 to 0.08 in women.

Analytic results

To determine whether genetic factors influenced the frequency of consumption of the different foods, we inspected the pattern of intraclass correlations by sex and zygosity for each food type (**Table 3**). Monozygotic twins were more similar than dizygotic twins for consumption of flour and grain products and fruit in both men and women, for consumption of milk in men, and for consumption of vegetables and rice in women. To assess whether the greater monozygotic twin concordances in reported food consumption of these foods reflected genetic predisposition or learned food choices, further analyses were conducted by rearing status (**Table 4**). If the similarity in food preferences was greater among the monozygotic than the dizygotic twins and also greater among the twins reared together than among those reared apart, there would be evidence for genetic and shared environmental effects for these food preferences. Among the men who were reared apart, monozygotic correlations were significantly greater than dizygotic correlations for the frequency of consumption of shellfish (r = 0.48 compared with −0.09) and milk (r = 0.31 compared with −0.10). Also in men, the frequency of consumption of milk was more similar among monozygotic twins reared together than among those reared apart (r = 0.56 compared with 0.31), whereas dizygotic twins reared together were not more similar than dizygotic twins reared apart (r = −0.18 com-

TABLE 2
Percentage of twin men and women reporting that they consumed certain foods more than once a week to daily[1]

	Men (n = 252)	Women (n = 296)
	%	
Meat	53.2 [134]	58.1 [172]
Sausages	37.3 [94]	27.4 [81][2]
Organ meats	5.6 [14]	5.7 [17]
Fish	26.2 [66]	30.1 [89]
Shellfish	2.4 [6]	2.4 [7]
Rice	8.3 [21]	7.4 [22]
Flour and grain products	31.4 [79]	30.4 [90]
Eggs	41.7 [105]	54.4 [161][3]
Vegetables	68.3 [172]	83.5 [247][4]
Fruit	78.6 [198]	91.6 [271][4]
Dairy products	91.3 [230]	98.3 [291][4]

[1] n in brackets.
[2–4] Significantly different from men: [2] P = 0.01, [3] P = 0.003, [4] P = 0.0001.

TABLE 3
Intraclass correlation coefficients for food consumption by zygosity among twin men and women

	Men		Women	
	Monozygotic twin (n = 106)	Dizygotic twin (n = 146)	Monozygotic twin (n = 90)	Dizygotic twin (n = 206)
Meat	0.33[1]	0.18	0.05	0.32[1,2]
Sausages	−0.02	0.03	−0.05	0.11
Organ meats	0.16	0.04	0.13	0.19[3]
Fish	0.03	0.03	0.16	−0.07
Shellfish	0.28[3]	0.04	0.54[1]	0.36[1]
Rice	0.09	0.23[3]	0.80[1]	0.28[1,2]
Flour and grain products	0.54[1]	0.22[2,3]	0.49[1]	0.10[2]
Eggs	0.11	−0.10	0.03	−0.15
Vegetables	0.28[3]	0.10	0.41[1]	0.05[2]
Fruit	0.43[1]	0.09[2]	0.20	−0.08[2]
Dairy products	0.41[1]	−0.11[2]	−0.08	−0.06

[1] $P < 0.001$.
[2] Significantly different from monozygotic twins, $P < 0.05$.
[3] $P < 0.05$.

pared with −0.10). Only 30 monozygotic women were reared apart, compared with 130 dizygotic women, and none of the correlations in monozygotic women reared apart were significant.

Next, for those foods for which the pattern of intraclass correlations suggested that genetic effects may be important, the cross-twin, cross-trait correlations of food type in twin A with weight change in twin B were examined. These correlations tested whether the same genes that may have affected food intake also affected weight change. All correlations were insignificant and ranged between −0.08 and 0.14 for monozygotic twins and between −0.09 and 0.10 for dizygotic twins, with the exception of rice consumption for men (r = −0.19 in monozygotic twins and −0.03 in dizygotic twins), which may reflect multiple comparisons. These findings suggest that for those foods for which genetic effects may have explained some of the variance in the frequency of consumption, the same genes did not mediate the relation between frequency of consumption and weight change.

Hierarchical multiple regression model

Main effects of baseline BMI and co-twin change in BMI predicted twin A change in BMI in men. In women, age and baseline smoking status also predicted change in BMI. As expected based on the correlations described above, type of food intake did not predict change in BMI (model 1) for any of the food categories. After adjustment for the main effects of food type and the other covariates (baseline information on smoking, physical activity, age, and body mass index), the two-way interaction between zygosity and the co-twin's change in BMI remained significant in all of the models in both men and women. This interaction indicated that there were genetic influences on weight change. There was some evidence of sex differences for the foods that predicted weight change. In women, the two-way interactions between the co-twin's weight change and the frequency of consumption of flour and grain products, milk, and fruit were significant (all $P < 0.04$); in men, the two-way interactions including frequency of consumption of organ meats, rice, and fruit were significant (all < 0.03). As expected, none of the cross products between zygosity and food intake frequency were significant (model 2).

Next, the three-way interactions between zygosity, the co-twin's change in BMI, and intake frequency were analyzed to test for gene-environment interactions. Except for a significant relation between the frequency of consumption of shellfish, zygosity, and BMI increase in men (β = −0.14, P = 0.02), all other three-way interactions were nonsignificant (all $P > 0.12$). These results suggest that there was no differential genetic effect on weight gain with respect to the consumption frequency of the foods studied.

Closer examination of the three-way interaction between zygosity, change in BMI, and shellfish intake revealed greater similarity for weight change in monozygotic pairs with frequent shellfish intake (more than weekly; β = 0.83, $P < 0.0001$) than in monozygotic pairs with infrequent intakes (β = 0.41, $P < 0.0001$). There was no similarity for weight change in the dizygotic men with either high or low intake frequencies (both $P > 0.41$). In addition, we also examined three-way interactions between a summary measure of high consumption frequency (based on total weekly consumption of all 11 foods), zygosity, and change in BMI, and found no evidence of such interactions ($P > 0.56$).

DISCUSSION

The prevalence of obesity is increasing throughout most of the world. Twin and adoption studies clearly show that there are genetic influences on obesity. It is therefore important to begin to understand how the responsible genes are expressed. The present study examined whether some genetic effects may work through food intake; our results suggest that genes influence the preference for certain types of foods, including shellfish, flour and grain products, vegetables, fruit, dairy products, and rice. However, there was no evidence that the same genes mediated the relation between these foods and weight change. Hence, for the specific foods examined here, the present findings do not suggest synergistic effects between genes and intake frequency on subsequent weight change. Although the significant relation between the frequency of consumption of shellfish, zygosity, and BMI increase in men may be indicative of a specific eating pattern associated with weight change in men genetically predisposed to obesity, this finding may also simply have been the statistical consequence of performing multiple testing. Furthermore, despite the greater similarity in weight change among male monozygotic twins with more frequent intakes of shellfish than in those with less frequent intakes, a further analysis revealed that monozygotic men with both a high and a low intake frequency experienced similar weight gains (1.5 ± 2.4 compared with 1.4 ± 1.9 kg; P for difference = 0.66).

Note that additional analyses using co-twin obesity as a measure of familial predisposition also did not indicate that high or low intake frequencies of any of the foods were differentially associated with weight change in persons genetically disposed or nondisposed to obesity (data not shown). However, although the present analyses did not, in general, indicate gene-environment interactions, the findings do not preclude their existence. First, only a small proportion of the respondents may have been genetically predisposed to obesity. In this respect, previous studies suggested that only 1–2% of a population may be particularly sensitive to dietary fat (2, 7). Second, the statistical power to detect interactions is a function of the study design, the sample size, and the magnitude of the interaction. If the putative interactions account for a small portion of the variance, then larger

XTABLE 4
Intraclass correlation coefficients for food consumption by zygosity and rearing among twin men and women

	Men				Women			
	Reared apart		Reared together		Reared apart		Reared together	
	Monozygotic twin (n = 50)	Dizygotic twin (n = 88)	Monozygotic twin (n = 56)	Dizygotic twin (n = 58)	Monozygotic twin (n = 30)	Dizygotic twin (n = 130)	Monozygotic twin (n = 60)	Dizygotic twin (n = 76)
Meat	0.26[1]	—	0.38[1]	0.27[1]	—	0.29[2]	—	0.35[2]
Sausages	—	—	—	—	—	—	—	0.23[1]
Organ meats	—	—	—	—	—	0.18[1]	—	0.21[1]
Fish	—	—	—	—	—	—	0.48[3]	—
Shellfish	0.48[3]	—	—	—	—	—	0.62[2]	—
Rice	—	0.28[1]	0.45[2]	—	—	0.37[2]	0.89[2]	—
Flour and grain products	—	—	0.51[2]	0.50[2]	—	—	0.78[2]	—
Eggs	—	—	—	—	—	−0.17[1]	—	—
Vegetables	—	0.19[1]	0.49[2]	—	—	—	0.49[2]	—
Fruit	0.40[1]	—	0.44[2]	—	—	—	0.33[1]	—
Dairy products	0.31[1]	—	0.56[2]	—	—	—	—	—

[1] $P < 0.05$.
[2] $P < 0.0001$.
[3] $P < 0.001$.

samples than were used here would have been necessary to detect such an interaction. Finally, the relatively limited between-person variability in the frequency of consumption of the foods may have masked the potential to identify gene-environment interactions. Although dietary habits are typically considered to be environmental measures, both genetic and environmental effects may influence intake patterns for some foods. Accordingly, we cannot exclude the theory that food intake represents a behavioral phenotype and that it therefore may be more relevant to think in terms of phenotype-gene interactions.

The present study used a food-frequency questionnaire to assess intakes of individual food categories rather than general dietary patterns. This was done to examine whether a certain eating pattern would be influenced by genes and in addition would interact with genes in promoting weight gain. Whereas a cluster of foods, such as those revealed by factor analysis, may reflect a general dietary pattern, individual food categories may be more informative regarding specific eating patterns [or certain aspects of eating, such as nutrient intake, taste, and satiation (4)] that may be characterized by heritable differences. Genetic effects were found for only some of the 11 food groups examined. Certain foods, such as meats, fruit, and dairy products, are preferred by most people, and a limited variability in the consumption frequency of these foods may have obliterated the ability of the statistical test to assess genetic heritability (4). In addition, for both meat and milk consumption there was evidence of shared environmental effects, suggesting that food behaviors learned in childhood may have long-lasting effects on food consumption frequency because most of the twins were in their 40s when the dietary data were collected.

We reported earlier that a high fat intake (>40% of energy from fat) predicted weight gain, particularly in women with a familial predisposition to obesity, suggesting that the relation between diet and obesity is modified by genes (7). The present study could not examine how total fat intake modified the relation between genes and weight change because total fat intake could not be derived from the food consumption data available. The frequency of intake of foods other than those examined here could represent a specific eating pattern that may potentiate obesity development in individuals with a genetic predisposition to obesity. In this context, food-frequency questionnaires have been shown to perform well in ranking individuals by nutrient intake (8, 9). In addition, we showed earlier that there is good agreement between individual foods reported by food-frequency questionnaires and foods reported by more thorough methods, such as diet history interviews (18).

The present study investigated whether genetic effects for preference of specific foods were related to a certain eating pattern. The results showed that although learned food choices seemed to play a role in the frequency of consumption of most of the foods examined, genetics also influenced the preference for several foods. However, there was no evidence that the consumption frequency of any of the foods examined was differentially associated with the expression of genes responsible for weight gain. The present findings do not exclude the possibility that gene-environment interactions may be important for weight gain in relation to dietary macronutrients, energy, or other more specific food groups. Future studies of twins that include more complete dietary information on nutrient intake are needed to address this issue more thoroughly.

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