Familial Resemblance in Eating Behaviors in Men and Women from the Quebec Family Study

Véronique Provencher, Louis Pérusse,† Luigi Bouchard,† Vicky Drapeau,† Claude Bouchard,§ Treva Rice,¶ D.C. Rao,¶‖ Angelo Tremblay,† Jean-Pierre Després,†‡ and Simone Lemieux**

Abstract

PROVENCHER, VÉRONIQUE, LOUIS PÉRUSSE, LUIGI BOUCHARD, VICKY DRAPEAU, CLAUDE BOUCHARD, TREVA RICE, D.C. RAO, ANGELO TREMBLAY, JEAN-PIERRE DESPRÉS, AND SIMONE LEMIEUX. Familial resemblance in eating behaviors in men and women from the Quebec Family Study. *Obes Res.* 2005;13:1624–1629.

Objective: It is commonly recognized that genetic, environmental, behavioral, and social factors are involved in the development of obesity. The family environment may play a key role in shaping children's eating behaviors. The purpose of this study was to estimate the degree of familial resemblance in eating behavioral traits (cognitive dietary restraint, disinhibition, and susceptibility to hunger).

Research Methods and Procedures: Eating behavioral traits were assessed with the Three-Factor Eating Questionnaire in 282 men and 402 women (202 families) from the Quebec Family Study. Familial resemblance for each trait (adjusted for age, sex, and BMI) was investigated using a familial correlation model.

Results: The pattern of familial correlation showed significant spouse correlation for the three eating behavior phenotypes, as well as significant parent-offspring and sibling correlations for disinhibition and susceptibility to hunger. According to the most parsimonious model, generalized heritability estimates (including genetic and shared familial environmental effects) reached 6%, 18%, and 28% for cognitive dietary restraint, disinhibition, and susceptibility to hunger, respectively.

Discussion: These results suggest that there is a significant familial component to eating behavioral traits but that the additive genetic component appears to be small, with generalized heritability estimates ranging from 6% to 28%. Thus, non-familial environmental factors and gene-gene and gene-environmental interactions seem to be the major determinants of the eating/behavioral traits.

Keys words: heritability, behavioral genetics, Three-Factor Eating Questionnaire

Introduction

Obesity is a major public health problem in developed countries (1,2), and it is important to understand the factors that contribute to its development in order to improve prevention and treatment strategies. Behavioral factors influence food intake and energy balance regulation in children and adults (3,4). Data suggest that genetic factors are involved in the modulation of the response to a variety of lifestyle and environmental exposures (5). Because parents transmit genes and largely define the environmental conditions to which their children are exposed, families may be enhancing the children's susceptibility to obesity. Previous studies have suggested that parents' eating behaviors are related to food intake and to the development of obesity in their children, and this link may be particularly true between mothers and daughters (3,6–8). Despite the fact that there are many familial and twin studies on the genetic and environmental components of food intake phenotypes, few reports have addressed the same question for eating behavior traits, as assessed by the Three-Factor Eating Question-

Received for review November 16, 2004.
Accepted in final form June 20, 2005.
The costs of publication of this article were defrayed, in part, by the payment of page charges. This article must, therefore, be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

*Department of Food Sciences and Nutrition, †Division of Kinesiology, Department of Preventive and Social Medicine, and ‡Quebec Heart Institute, Hospital Laval Research Center, Laval University, Ste-Foy, Quebec, Canada; §Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, Louisiana; ¶Division of Biostatistics and ‖Department of Genetics and Department of Psychiatry, Washington University, School of Medicine, St. Louis, Missouri.
Address correspondence to Simone Lemieux, Institute of Nutraceuticals and Functional Foods, 2440 Hochelaga Boulevard, Laval University, Quebec, Canada, G1K 7P4.
E-mail: simone.lemieux@aln.ulaval.ca

Table 1. Descriptive statistics of the sample in each of the sex and generation groups

	N	Fathers	*N*	Mothers	*N*	Sons	*N*	Daughters
Age (years)	143	55.5 ± 9.2[a]*	189	53.5 ± 9.4[a]	139	31.8 ± 9.4[b]	213	31.7 ± 10.3[b]
BMI (kg/m^2)	142	29.2 ± 6.2[ab]	188	30.1 ± 8.5[a]	139	28.0 ± 7.5[b]	213	28.2 ± 8.9[b]
Restraint	143	5.8 ± 3.7[a]	189	8.7 ± 4.7[b]	139	5.8 ± 3.5[a]	213	7.8 ± 4.8[b]
Disinhibition	143	4.4 ± 3.1[a]	189	5.6 ± 3.4[b]	139	4.7 ± 2.9[a]	213	6.2 ± 3.5[b]
Hunger	143	3.9 ± 3.4[a]	189	3.7 ± 3.1[a]	139	4.3 ± 3.6[a]	213	4.2 ± 3.3[a]

Values are means ± SD.
* Variables with different superscript letters are significantly different ($p < 0.05$).

naire (TFEQ).[1] In a family study conducted in an Old Order Amish sample, Steinle et al. (9) observed significant heritabilities for cognitive dietary restraint, disinhibition, and susceptibility to hunger. In twin studies, significant heritabilities for these behavioral traits were also reported (10–12). The main purpose of this study was to explore further the familial correlations and to estimate generalized heritability levels in eating behaviors (cognitive dietary restraint, disinhibition, and susceptibility to hunger) in a sample of men and women from the Quebec Family Study (QFS).

Research Methods and Procedures

Participants

The QFS was initiated in 1978 at Laval University to investigate the role of genetic factors in the etiology and health consequences of obesity (13). All adult participants (>18 years old), from French-Canadian families, signed an informed consent document (approved by the Medical Ethics Committee of Laval University).

Measurements

Body weight and height were measured according to standardized procedures (14), and BMI was calculated (kilograms per meter squared).

A French version of the TFEQ, also known as the Eating Inventory, was completed by 282 men and 402 women (202 nuclear families). The TFEQ is a 51-item validated questionnaire that assesses quantitatively three eating behavior traits: cognitive dietary restraint (conscious control over food intake with concerns about shape and weight), disinhibition (overconsumption of food in response to stimuli associated with a loss of control), and susceptibility to hunger (food intake in response to feelings and perceptions of hunger) (15).

[1] Nonstandard abbreviations: TFEQ, Three-Factor Eating Questionnaire; QFS, Quebec Family Study; f, father; m, mother; s, son; d, daughter.

Statistical Analysis

Before computing familial correlations, eating behavior traits were adjusted for potential confounders because age and BMI were related to these phenotypes (age, $-0.13 \leq r \leq -0.17$; BMI, $0.24 \leq r \leq 0.51$; data not shown). Adjustments were performed by step-wise multiple regression procedure separately on four age-by-sex generation groups: men < 40 years old, men ≥ 40 years old, women < 40 years old, and women ≥ 40 years old.

A familial correlation model based on four groups of individuals was implemented: fathers (f), mothers (m), sons (s), and daughters (d). These family members gave rise to eight correlations: one spouse correlation (fm), four parent-offspring correlations (fs, fd, ms, and md), and three sibling correlations (ss, dd, and sd). Detailed explanations have been given by Pérusse et al. (16). Maximal heritabilities were computed from the maximum likelihood estimates of the familial correlations obtained under the most parsimonious model (17):

$$h^2 = (r_{sibling} + r_{parent\text{-}offspring}) - (1 + r_{spouse})/$$
$$(1 + r_{spouse} + 2\, r_{spouse}\, r_{parent\text{-}offspring})$$

These heritability estimates, named generalized heritability, include the contribution of both genetic and shared familial environmental factors transmitted from parents to offspring and are adjusted for spouse resemblance. Heritability estimates from twin studies rather refer to genetic sources of variance and must be compared with caution with generalized heritability.

Results

Baseline characteristics of the sample for each of the sex-by-generation groups (fathers, mothers, sons, and daughters) are presented in Table 1. Sex differences were noted for cognitive dietary restraint and disinhibition.

Table 2 summarizes the model-fitting results for each eating behavioral trait. All hypotheses tested could not be

Table 2. Summary of results from fitting reduced models for cognitive dietary restraint, disinhibition, and susceptibility to hunger

Models	*df*	Cognitive dietary restraint		Disinhibition		Susceptibility to hunger	
		*p**	AIC†	*p*	AIC	*p*	AIC
1. No sex differences, offspring	4	0.379	12.21	0.535	11.14	0.059	17.08
2. No sex differences, offspring or parents	5	0.256	12.55	0.544	10.03	0.095	15.38
3. No sex or generation differences	6	**0.362**	**10.58**‡	0.506	9.30	0.145	13.55
4. Environmental model, equal	7	0.278	10.66	**0.606**	**7.45**	**0.207**	**11.70**
5. No sibling correlations	3	0.493	12.41	0.960	10.30	0.114	15.95
6. No parent-offspring correlations	4	0.432	11.81	0.108	15.59	0.001	25.84
7. No spouse correlation	1	0.051	17.80	0.800	14.06	0.147	16.11
8. No familial correlations	8	0.219	10.70	0.152	11.97	0.001	26.35

Values are adjusted for age, sex, and BMI.

* p values from the likelihood ratio χ^2 test; a significant value ($p < 0.05$) indicates rejection of the null hypothesis.

† AIC, Akaike's Information Criterion; the most parsimonious model is one with the smallest AIC.

‡ Values of the model in bold represent the most parsimonious model for this eating behavior.

rejected for cognitive dietary restraint and disinhibition. Regarding susceptibility to hunger, the hypotheses of no familial correlations and no parent-offspring correlations were rejected, whereas the hypothesis of no spouse correlation could not be rejected.

The familial correlations under the general model and under the most parsimonious model are presented in Table 3 for each eating behavioral trait. For disinhibition and susceptibility to hunger, familial correlations between mothers and daughters were higher compared with the other familial correlations in the general model. The spouse correlation was significant for each phenotype, but this correlation was higher for cognitive dietary restraint. The most parsimonious model was the no sex nor generation differences model (Model 3) for cognitive dietary restraint and the environmental model (Model 4) for both susceptibility to hunger and disinhibition (Table 3). Under the most parsimonious model, generalized heritability estimates were 5.5% for cognitive dietary restraint, 17.5% for disinhibition, and 28.4% for susceptibility to hunger.

Discussion

Results from this study suggest that eating behavioral traits are characterized by significant familial resemblance, particularly for susceptibility to hunger. Significant familial correlations were observed for susceptibility to hunger, with a higher generalized heritability estimate of 28.4% compared with cognitive dietary restraint (5.5%) and disinhibition (17.5%). Moreover, under the most parsimonious model, the spouse correlation was the only significant familial correlation observed for cognitive dietary restraint. Because the most parsimonious model was the environmental model for disinhibition and susceptibility to hunger, environmental factors seem to be a major determinant of these traits. Moreover, under the general model, correlations between mothers and daughters were the highest observed for disinhibition and susceptibility to hunger, suggesting that the maternal contribution may be particularly important in daughters, thus underscoring the possible importance of a gender effect on these behaviors.

Although using a different methodology, our results are supported by previous studies in which eating behaviors of parents correlated with those of children (3,8,18,19). For example, Birch et al. (18) reported that girls who had a mother with restrictive feeding practices were more likely to overeat as shown by a higher energy intake in the absence of hunger (8).

Even if there is evidence of familial transmission for cognitive dietary restraint, this resemblance is more predominant between spouses. Thus, an assortative mating effect cannot be ruled out, as previously suggested for other phenotypes related to obesity (20). Moreover, social and external factors, such as the high prevalence of dieting practices in women, may also be involved.

Familial environment seems to be important regarding the disinhibition trait because the most parsimonious model was the environmental model. However, the data were adjusted for the confounding effect of BMI, and this

Table 3. Maximum likelihood estimates of familial correlations (±SE) under the general and the most parsimonious inheritance models for cognitive dietary restraint, disinhibition, and susceptibility to hunger

Parameter	Cognitive dietary restraint	Disinhibition	Susceptibility to hunger
General model			
fm	0.174 ± 0.086	0.129 ± 0.092	0.127 ± 0.086
fs	0.055 ± 0.106	0.067 ± 0.103	0.171 ± 0.117
ms	0.121 ± 0.097	0.034 ± 0.104	0.003 ± 0.105
fd	−0.145 ± 0.104	0.103 ± 0.097	0.094 ± 0.093
md	0.030 ± 0.090	0.235 ± 0.072	0.307 ± 0.067
sd	−0.044 ± 0.097	0.047 ± 0.081	0.145 ± 0.093
ss	−0.019 ± 0.121	0.128 ± 0.121	0.238 ± 0.155
dd	0.134 ± 0.094	−0.057 ± 0.087	0.084 ± 0.080
Most parsimonious model			
fm	0.168 ± 0.086	0.089 ± 0.038	0.148 ± 0.040
fs	0.028 ± 0.041	(0.089)	(0.148)
ms	(0.028)*	(0.089)	(0.148)
fd	(0.028)	(0.089)	(0.148)
md	(0.028)	(0.089)	(0.148)
sd	(0.028)	(0.089)	(0.148)
ss	(0.028)	(0.089)	(0.148)
dd	(0.028)	(0.089)	(0.148)
Generalized heritability	5.5%	17.5%	28.4%

Values are adjusted for age, sex, and BMI.
* Values in parentheses are fixed or equal to preceding value.

adjustment may have had an impact on the results. When analyses were performed without adjustment for BMI, the most parsimonious model was the no sex differences in the offspring model (Model 1) (data not shown). This suggests that BMI and eating behavioral trait covary in humans. The finding that disinhibition was positively correlated with BMI is concordant with other studies (21–28). In this regard, it is not possible to establish whether a high BMI would lead to high disinhibition scores or whether a high disinhibition score is a risk factor for obesity. One could speculate that the familial correlations observed for the disinhibition trait may be related to the familial aggregation commonly observed for obesity (29–32). However, sequence variations in candidate genes for disinhibition, such as GAD2 (33) and neuromedin β (34), have been recently found, which suggest that genetic factors could be involved.

Significant familial correlations were observed for the susceptibility to hunger trait, and the maximum likelihood estimates of familial correlations suggest that familial environment may be the major determinant of this trait. Few studies have examined the development of eating behavioral traits in parents and adult offspring, which limits comparisons with the present study. Johnson and Birch (19) previously reported that, in order to sustain the ability for self-regulation, parents should not exert too much control over a child's feeding. In addition, accessibility and availability of food, which are part of both familial and social environment, could be factors that also contribute to the development of susceptibility to hunger (35). Candidate genes have also been found to be associated with the susceptibility to hunger (33,34), which suggests that a genetic contribution cannot be excluded.

As shown in the general model for disinhibition and susceptibility to hunger, the highest familial correlations were those between mother and daughter, which suggest that the maternal contribution may be particularly important for eating behavioral traits in daughters. In typical families, mothers purchase foods and plan and prepare meals. Previous studies have highlighted that maternal feeding practices are more related to daughters' than sons' eating behaviors (8,19,36,37). The father's contribution to offspring eating behavioral traits has not been studied to any extent.

Finally, few studies have examined adult eating behaviors in relation to their parents' behaviors. One could hypothesize that the importance of the immediate familial

environment on eating behaviors is greater in younger than in older offspring. In fact, as a child grows, other influences such as peers and other meaningful persons can be expected to exert a strong effect on behaviors, including those related to eating. In the present study, we found significant familial correlations even though the families included only adult offspring. This suggests that the development of eating behaviors during growth remains a critical determinant of eating behavioral traits during adult life.

In conclusion, results from this study suggest that eating behavioral traits aggregate in families. The familial environment seems to be the major determinant of the familial correlations observed, although a genetic contribution cannot be excluded. In tailoring prevention and treatment programs for obese and overweight people, the influence of family environment on eating behavioral traits should be considered.

Acknowledgments

V.P. and L.B. are recipients of a studentship from the Fonds de la Recherche en Santé du Quebec. S.L. is a research scholar from the Fonds de la Recherche en Santé du Quebec. A.T. is partly funded by the Canada Research Chair in Physical Activity, Nutrition, and Energy Balance. C.B. is partially supported by the George A. Bray Chair in Nutrition. We thank the subjects for excellent collaboration and the staff of the Physical Activity Sciences Laboratory for contribution to this study. We especially thank G. Thériault, L. Allard, G. Bouchard, S. Brulotte, L. Bargone, M. Chagnon, G. Fournier, H. Bessette, and C. Leblanc for help in the collection and analysis of the data. The QFS has been supported over the years by multiple grants from the Medical Research Council of Canada and other Canadian agencies.

References

1. **Flegal KM, Carroll MD, Ogden CL, Johnson CL.** Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*. 2002;288:1723–7.
2. **Ledoux M, Rivard M.** Poids corporel. In: Institut de la Statistique duq Uébec, ed. *Enquête Sociale et de Santé 1998.* 2nd ed. Sainte-Foy, Canada: Les Publications duq Uébec; 1998, pp. 187–99.
3. **Birch LL, Fisher JO.** Development of eating behaviors among children and adolescents. *Pediatrics*. 1998;101:539–49.
4. **Provencher V, Drapeau V, Tremblay A, Despres JP, Lemieux S.** Eating behaviors and indexes of body composition in men and women from the Québec Family Study. *Obes Res*. 2003;11:783–92.
5. **Perusse L, Bouchard C.** Role of genetic factors in childhood obesity and in susceptibility to dietary variations. *Ann Med.* 1999;31(Suppl 1):19–25.
6. **Cutting TM, Fisher JO, Grimm-Thomas K, Birch LL.** Like mother, like daughter: familial patterns of overweight are mediated by mothers' dietary disinhibition. *Am J Clin Nutr*. 1999;69:608–13.
7. **Hood MY, Moore LL, Sundarajan-Ramamurti A, Singer M, Cupples LA, Ellison RC.** Parental eating attitudes and the development of obesity in children: The Framingham Children's Study. *Int J Obes Relat Metab Disord*. 2000;24:1319–25.
8. **Birch LL, Fisher JO, Davison KK.** Learning to overeat: maternal use of restrictive feeding practices promotes girls' eating in the absence of hunger. *Am J Clin Nutr*. 2003;78:215–20.
9. **Steinle NI, Hsueh WC, Snitker S, et al.** Eating behavior in the Old Order Amish: heritability analysis and a genome-wide linkage analysis. *Am J Clin Nutr*. 2002;75:1098–106.
10. **Neale BM, Mazzeo SE, Bulik CM.** A twin study of dietary restraint, disinhibition and hunger: an examination of the eating inventory (three factor eating questionnaire). *Twin Res*. 2003;6:471–8.
11. **Tholin S, Rasmussen F, Tynelius P, Karlsson J.** Genetic and environmental influences on eating behavior: the Swedish Young Male Twins Study. *Am J Clin Nutr*. 2005;81:564–9.
12. **de Castro JM, Lilenfeld LR.** Influence of heredity on dietary restraint, disinhibition, and perceived hunger in humans. *Nutrition*. 2005;21:446–55.
13. **Bouchard C.** Genetic and body fat content. In: Angel A, Anderson H, Bouchard C, Lau D, Leither L, Mendelson R, eds. *Progress in Obesity Research*. London, UK: John Libbey; 1996, pp. 33–41.
14. **The Airlie (VA) Consensus Conference.** *Standardization of Anthropometric Measurements.* Champaign, IL: Human Kinetics Publishers; 1988.
15. **Stunkard AJ, Messick S.** The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res*. 1985;29:71–83.
16. **Perusse L, Rice T, Province MA, et al.** Familial aggregation of amount and distribution of subcutaneous fat and their responses to exercise training in the HERITAGE family study. *Obes Res*. 2000;8:140–50.
17. **Rice T, Despres JP, Daw EW, et al.** Familial resemblance for abdominal visceral fat: the HERITAGE family study. *Int J Obes Relat Metab Disord*. 1997;21:1024–31.
18. **Carper JL, Orlet FJ, Birch LL.** Young girls' emerging dietary restraint and disinhibition are related to parental control in child feeding. *Appetite*. 2000;35:121–9.
19. **Johnson SL, Birch LL.** Parents' and children's adiposity and eating style. *Pediatrics*. 1994;94:653–61.
20. **Hebebrand J, Wulftange H, Goerg T, et al.** Epidemic obesity: are genetic factors involved via increased rates of assortative mating? *Int J Obes Relat Metab Disord.* 2000;24:345–53.
21. **Westenhoefer J, Stunkard AJ, Pudel V.** Validation of the flexible and rigid control dimensions of dietary restraint. *Int J Eat Disord*. 1999;26:53–64.
22. **Carmody TP, Brunner RL, St Jeor ST.** Dietary helplessness and disinhibition in weight cyclers and maintainers. *Int J Eat Disord*. 1995;18:247–56.
23. **Lawson OJ, Williamson DA, Champagne CM, et al.** The

association of body weight, dietary intake, and energy expenditure with dietary restraint and disinhibition. *Obes Res*. 1995; 3:153–61.

24. **Hays NP, Bathalon GP, McCrory MA, Roubenoff R, Lipman R, Roberts SB.** Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *Am J Clin Nutr*. 2002;75:476–83.
25. **Lindroos AK, Lissner L, Mathiassen ME, et al.** Dietary intake in relation to restrained eating, disinhibition, and hunger in obese and nonobese Swedish women. *Obes Res*. 1997;5:175–82.
26. **Karlsson J, Hallgren P, Kral J, Lindroos AK, Sjöström L, Sullivan M.** Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. *Appetite*. 1994;23:15–26.
27. **Boschi V, Iorio D, Margiotta N, D'Orsi P, Falconi C.** The three-factor eating questionnaire in the evaluation of eating behaviour in subjects seeking participation in a dietotherapy programme. *Ann Nutr Metab*. 2001;45:72–7.
28. **Gendall KA, Joyce PR, Sullivan PF, Bulik CM.** Personality and dimensions of dietary restraint. *Int J Eat Disord*. 1998; 24:371–9.
29. **Maes HH, Neale MC, Eaves LJ.** Genetic and environmental factors in relative body weight and human adiposity. *Behav Genet*. 1997;27:325–51.
30. **Katzmarzyk PT, Malina RM, Perusse L, et al.** Familial resemblance in fatness and fat distribution. *Am J Human Biol*. 2000;12:395–404.
31. **Bouchard C, Perusse L.** Genetics of obesity: family study. In: *The Genetics of Obesity.* Boca Raton, FL: CRC Press; 1994, pp. 79–92.
32. **Rice T, Perusse L, Bouchard C, Rao DC.** Familial aggregation of body mass index and subcutaneous fat measures in the longitudinal Quebec family study. *Genet Epidemiol*. 1999; 16:316–34.
33. **Boutin P, Dina C, Vasseur F, et al.** GAD2 on chromosome 10p12 is a candidate gene for human obesity. *PLoS Biol*. 2003;1:E68.
34. **Bouchard L, Drapeau V, Provencher V, et al.** Neuromedin B: a strong candidate gene linking eating behaviors and susceptibility to obesity. *Am J Clin Nutr*. 2004;80:1478–86.
35. **Hill AJ.** Developmental issues in attitudes to food and diet. *Proc Nutr Soc*. 2002;61:259–66.
36. **Birch LL, Fisher JO.** Mothers' child-feeding practices influence daughters' eating and weight. *Am J Clin Nutr*. 2000;71: 1054–61.
37. **Fisher JO, Birch LL.** Restricting access to foods and children's eating. *Appetite*. 1999;32:405–19.