

Obesity in Canada

Angelo Tremblay, PhD
Division of Kinesiology, Laval University

The prevalence of obesity has increased dramatically over the past few decades despite its well documented determinants, consequences, and related interventions¹. This might reflect the complex nature of this problem but may also reveal that the obesity investigation paradigm may not take into account important aspects of this phenomenon. This article reviews some aspects of this issue to evaluate the likelihood of successfully preventing and treating obesity.

What is obesity?

Obesity is characterized by a large accumulation of body fat, which translates to a significant fat cell hypertrophy and possibly to fat cell hyperplasia as well. It results from the long term inability to match energy intake to energy expenditure as well as fat intake to fat oxidation^{2,3}. In this context, fat cells offer a preferential site of storage of excess calories and lipids. Obesity or fat content is often measured by body mass index (BMI), which is calculated from weight (kg) divided by height squared (m²). Currently, a BMI of 20-24.9 is considered to be a desirable weight, 25-29.9 is overweight, 30-40 is obese, and over 40 is morbid obesity [Table 1]. Understanding the determinants of obesity is vital because obesity is associated with several morbid events and increased mortality.

What are obesity determinants?

Since it is unlikely that the genetic pool of populations has changed significantly over the course of the 20th century, variations in environmental conditions thus constitute the main determinants of this phenomenon. This does not mean that genes are not involved in the problem since one's genetic background can provide an increased susceptibility to the impact of a "fattening" lifestyle. This so-called "gene-environment" interaction effect⁴ is being actively researched. Researchers are investigating the genetic variations to explain the most pronounced susceptibility of some individuals to an environment favoring excess energy intake.

One of the environmental factors that is thought to have greatly contributed to the increased prevalence of obesity is the change in dietary food intake. Indeed, a large body of scientific literature shows that a high-fat diet⁵, an increased energy density of foods⁶, a high glycemic index of carbohydrate-containing foods^{7,8}, and a low fiber intake^{9,10} are all factors that have, to differing degrees, the potential to

increase energy intake before reaching satiety, which obviously complicates the long term maintenance of a stable body weight.

Physical inactivity is another feature of a modern lifestyle that predicts weight gain over the years¹¹. In fact, the increasing number of sedentary individuals throughout the world is not surprising since humans have been looking for ways to reduce the energy demand of labor and to increase everyday efficiency and productivity over the course of their evolution.

As shown in Table 2, a low-fat diet and regular physical activity are important features of individuals who maintain a stable body weight after successful weight loss¹². A similar finding was reported by other investigators demonstrating that the combination of prolonged exercise of moderate intensity and a diet with a reduced fat content favors a substantial spontaneous daily energy deficit¹³.

Even if healthier dietary and physical activity habits affect energy balance, this does not mean that they are the sole factors whose variations can modify the matching between energy intake and expenditure. Preliminary evidence suggests that stress and pollution¹⁴ might also have the potential to disturb energy balance. In addition, variations in micronutrient intake might influence the equilibrium between energy intake and expenditure¹⁵.

What is the cost of obesity?

The financial burden of an increase in obesity prevalence has been a matter of preoccupation over the last decade¹⁶. In Canada, recent estimates suggest that the total direct cost of obesity in 1997 was above 1.8 billion dollars¹⁷. Several health conditions contribute to this figure. The three largest contributors are hypertension (\$656.6 million), type 2 diabetes mellitus (\$423.2 million) and coronary artery disease (\$346 million). In other words, a substantial proportion of health care spending in Canada is devoted to the treatment and management of obesity-related comorbidities¹⁷.

What is the optimal treatment of obesity?

Most health professionals agree that prevention is the best way to deal with obesity. In fact, from a physiological standpoint, prevention might be much better than gaining and then subsequently losing weight with success. Indeed, with our

current knowledge we cannot exclude the possibility that cycles of weight gain and weight loss negatively affect the equilibrium between energy intake and expenditure.

For those who are unsuccessful at preventing obesity, treatment is the alternative but it clearly deserves a closer look. A recent study utilized a weight-reducing program whose main aims were to induce body weight loss up to the achievement of a resistance to further fat loss and to reduce the risk of developing metabolic diseases such as diabetes and cardiovascular diseases¹⁸. The study was also imprinted by the paradigm according to which weight loss below a reference value of 30 kg/m² was a desirable intent and that its achievement would be largely beneficial to obese patients. This program included a first 15-week phase of drug therapy with fenfluramine which was combined to a dietary restriction. This was followed by a second phase during which subjects adhered to a healthy diet and exercise regime to maintain or even accentuate weight loss up to a resistance to further fat loss. As hypothesized, the regime succeeded in improving the metabolic profile, as reflected by a decrease in plasma glucose and insulin during an oral glucose tolerance test (OGTT) and by favorably improving the lipid-lipoprotein profile¹⁹. Moreover, subjects experienced a substantial weight loss, but this was not sufficient to confer the status of non-obese to the majority of the participants, i.e. with a BMI < 30 kg/m². Beyond this expected outcome, some undesirable effects were also noted, these included a greater than expected decrease in resting energy expenditure and fat oxidation^{20,21}, a slight increase in hypoglycemia at the end of an OGTT¹⁹ and an increase in hunger and desire to eat²². Subsequent analysis revealed that both a reduced availability of fat from the adipose tissue²³ and the amplification of hypoglycemia (Boulé et al. Unpublished results) both predicted weight regain during a follow-up of several years.

Since the completion of this study, a physiological assessment service has been developed and offered to physicians treating obesity in the Québec City area. Preliminary results show that such an evaluation allows health care professionals to identify problems in the control of energy intake and expenditure in a significant proportion of obese people²⁴. It is hoped that this service will help clinicians to treat obesity while seeking a reasonable compromise between

the reduction of the risk towards metabolic diseases such as diabetes and evaluating the risk of positive energy balance and weight regain.

Is micronutrient intake a relevant issue?

Even if many micronutrients are directly involved in the ATP-producing machinery, the consideration of their potential role in the control of energy intake and expenditure has not been traditionally part of the conceptual paradigm of health professionals. Recent research however emphasizes the potential relationship between variations in the intake of folic acid²⁵, magnesium²⁶, and calcium²⁷ and variations in body weight and/or adiposity. The potential contribution of vitamin supplementation on hunger, energy expenditure, and body composition in subjects of the Québec Family Study was investigated. These data revealed that vitamin supplement consumers are leaner than their non-consumer counterparts and that they are also characterized by a lower hunger level as well as an increased resting energy expenditure corrected for body weight¹⁵. Interestingly, our physiological assessment service to physicians revealed that a large percentage of the obese patients tested in this context reported calcium and folic acid intake levels that were below nutritional guidelines.

Are we losing against obesity?

The analysis presented in the previous sections suggests that the battle against obesity is extremely challenging. The fight against obesity is difficult due to three main factors: 1) the inability to recreate conditions related to the macronutrient composition of the diet and to a level of physical activity that spontaneously favors the equilibrium between energy intake and expenditure. After all, body fat gain still remains the best known strategy to permit the recovery of energy balance under sub-optimal environmental conditions; 2) the consideration of other environmental factors that might promote an energy imbalance, is difficult to account for; and 3) our limited knowledge of most of the genes which are currently responsible for a more pronounced susceptibility to obesity in some individuals.

In the meantime, the most realistic approach to manage obesity is to evaluate the overweight status while controlling the risk toward its related health complications. Any attempt to progress toward a healthy lifestyle is welcome even if the resulting outcome on body weight is

modest. In fact, many studies show that an improvement of dietary and activity habits, with or without drug therapy, may normalize the risk to develop metabolic diseases even if the obesity phenotype persists after the intervention²⁸⁻³⁰.

Table 1: Variation of weight with height for adults of similar fatness indicated by body mass index (BMI).

Height		BMI = 20 Weight		BMI = 25 Weight		BMI = 30 Weight		BMI = 40 Weight	
m	ft. in.	kg	lb	kg	lb	kg	lb	kg	lb
1.5	5'1"	48	106	60	132	72	158	96	211
1.60	5'3"	51	112	64	141	77	169	102	224
1.65	5'5"	54	119	68	150	83	183	108	238
1.70	5'7"	58	128	72	159	87	191	116	255
1.75	5'9"	61	134	77	169	92	202	122	268
1.80	5'11"	65	143	81	178	97	213	130	286
1.85	6'1"	68	150	86	189	103	226	146	321

Table 2: Characteristics of individuals maintaining a weight loss of at least 30 pounds (13.6 kg) for at least one year. Adapted from McGuire et al.¹⁶.

Body weight loss	30.1 kg
Duration of maintenance	5.7 years
Relative fat intake	25 % of total energy intake
Physical activity participation*	11847 kJ/week

* Including strenuous physical activities.

The Whitehall-Robins Report is a Whitehall-Robins publication that focuses on current issues on the role of vitamins and minerals in health promotion and disease prevention. Complimentary copies are distributed to Canadian health care professionals active or with a special interest in nutrition. Each issue is written and/or reviewed by independent health care professionals with expertise in the chosen topic.

Editor: Whitehall-Robins Inc.

If you have any comments about the Whitehall-Robins Report or would like to be added to the mailing list, please write to:

The Editor: The Whitehall-Robins Report,
5975 Whittle Road, Mississauga, ON L4Z 3M6



© 2001-August. May be reproduced without permission provided source is recognized.

References 1. Tremblay A, Doucet E. Obesity: A disease or a biological adaptation? *Obesity Reviews* 2000; 1:27-35. 2. Schutz Y, Tremblay A, Weinsier RL, Nelson KM. Role of fat oxidation in the long-term stabilization of body weight in obese women. *Am J Clin Nutr* 1992;55:670-674. 3. Astrup A, Buemann B, Western P, Toubro S, Raben A, Christensen NJ. Obesity as an adaptation to a high-fat diet: evidence from a cross-sectional study. *Am J Clin Nutr* 1994;59:350-5. 4. Chagnon YC, Perusse L, Weisnagel SJ, Rankinen T, Bouchard C. The human obesity gene map: the 1999 update. *Obes Res* 2000;8:89-117. 5. Tremblay A, Plourde G, Després JP, Bouchard C. Impact of dietary fat content and fat oxidation on energy intake in humans. *Am J Clin Nutr* 1989;49:799-805. 6. Poppitt SD, Prentice AM. Energy density and its role in the control of food intake: Evidence from metabolic and community studies. *Appetite* 1996;26:153-174. 7. Holt SH, Miller JB. Particle size, satiety and the glycaemic response. *Eur J Clin Nutr* 1994;48:496-502. 8. Holt SH, Miller JB. Increased insulin responses to ingested foods are associated with lessened satiety. *Appetite* 1995;24:43-54. 9. Kimm SYS. The role of dietary fiber in the development and treatment of childhood obesity. *Pediatrics* 1995;96:1010-1014. 10. Van Italie TB. Dietary fiber and obesity. *Am J Clin Nutr* 1978;31:S43-S52. 11. Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? *Br Med J* 1995;311:437-439. 12. McGuire MT, Wing RR, Klem ML, Seagle HM, Hill JO. Long-term maintenance of weight loss: do people who lose weight through various weight loss methods use different behaviors to maintain their weight? *Int J Obes* 1998;22:572-7. 13. Tremblay A, Almeras N, Boer J, Kranenbarg EK, Després JP. Diet composition and postexercise energy balance. *Am J Clin Nutr* 1994;59:975-979. 14. Tremblay A, Imbeault P, Chevrier C, Richard D. Obesity as a response to environmental stress and pollution. *Obesity Matters* 1999;2:14-17. 15. Doucet E, Després J-P, Bouchard C, Tremblay A. Body weight and composition in consumers and non-consumers of vitamin supplements. *Int J Obes* 2000;24:S157. 16. Wolf AM, Colditz GA. Current estimates of the economic cost of obesity in the United States. *Obes Res* 1998;6:97-106. 17. Birmingham CL, Muller JL, Palepu A, Spinelli JJ, Anis AH. The cost of obesity in Canada. *Can Med Assoc J* 1999;160:483-8. 18. Doucet E, Imbeault P, Almeras N, Tremblay A. Physical activity and low-fat diet: is it enough to maintain weight stability in the reduced-obese individual following weight loss by drug therapy and energy restriction? *Obes Res* 1999;7:323-33. 19. Tremblay A, Doucet E, Imbeault P, Mauriege P, Despres JP, Richard D. Metabolic fitness in active reduced-obese individuals. *Obes Res* 1999;7:556-63. 20. Doucet E, St Pierre S, Almeras N, Mauriege P, Richard D, Tremblay A. Changes in energy expenditure and substrate oxidation resulting from weight loss in obese men and women: is there an important contribution of leptin? *J Clin Endocrinol Metab* 2000;85:1550-6. 21. Doucet E, St-Pierre S, Almeras N, Després J-P, Bouchard C, Tremblay A. Evidence for the existence of adaptive thermogenesis during weight loss. *Br J Nutr* In press. 22. Doucet E, Imbeault P, St-Pierre S, Almeras N, Mauriege P, Richard D, Tremblay A. Appetite after weight loss by energy restriction and a low-fat diet-exercise follow-up. *Int J Obes* 2000;24:906-914. 23. Mauriege P, Imbeault P, Doucet E, Lacaille M, Langin D, Almeras N, Tremblay A, Despres JP. Adipose tissue metabolic predictors of weight regain: existence of regional and gender differences. *Obes Res* 1999;7:52S. 24. Drapeau V, Berube S, Tremblay A. An evaluation service to support clinical intervention in obesity: description and preliminary data. *Obes Res* 2000;8:70S. 25. Kato I, Dnistrian AM, Schwartz M, Toniolo P, Koehnig K, Shore RE, Zeleniuch-Jacquotte A, Akhmedkhanov A, Riboli E. Epidemiologic correlates of serum folate and homocysteine levels among users and non-users of vitamin supplement. *Int J Vitam Nutr Res* 1999;69:322-9. 26. Singh RB, Beegom R, Rastogi SS, Gaoli Z, Shoumin Z. Association of low plasma concentrations of antioxidant vitamins, magnesium and zinc with high body fat per cent measured by bio-electrical impedance analysis in Indian men. *Magnes Res* 1998;11:3-10. 27. Zemel MB, Shi H, Greer B, Dirienzo D, Zemel PC. Regulation of adiposity by dietary calcium. *FASEB J* 2000;14:1132-8. 28. Wing RR, Jeffery RW. Effect of modest weight loss on changes in cardiovascular risk factors: are there differences between men and women or between weight loss and maintenance. *Int J Obes* 1995;19:67-73. 29. Goldstein DJ. Beneficial health effects of modest weight loss. *Int J Obes* 1992;16:397-415. 30. Tremblay A, Després J-P, Maheux J, Pouliot MC, Nadeau A, Moorjani PJ, Lupien PJ, Bouchard C. Normalization of the metabolic profile in obese women by exercise and a low fat diet. *Med Sci Sports Exerc* 1991;23:1326-1331.