Extra Calories Cause Weight Gain But How Much?

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Extra Calories Cause Weight Gain—But How Much?

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How much weight would an individual gain by eating an extra chocolate chip cookie every day for life? One approach to answering this question, frequently used in textbooks[1] and scientific articles, is based on the assumption that a pound (454 g) of fat tissue has about 3500 kilocalories (kcal). Thus, a daily 60-kcal cookie would be expected to produce 0.2 kg (0.5 lb) weight gain in a month, 2.7 kg (6 lb) in a year, 27 kg (60 lb) in a decade, and many hundreds of pounds in a lifetime. This of course does not happen. In this article, the physiology of weight gain and loss is reviewed, and the amount of reduction of caloric intake necessary to avoid becoming overweight or obese is estimated.

Weight Change Is Self-limiting

Body weight remains stable as long as the number of calories consumed equals the number expended through physical activities and metabolic processes. When energy intake increases above expenditure, the excess is used to build new tissue, and weight gain results. However, weight gain does not continue indefinitely. Carefully controlled overfeeding experiments show that calorie expenditure increases progressively because of the energetic costs of maintaining the newly created tissue. A person who consumes an extra cookie every day will initially experience weight gain, but over time an increasing proportion of the cookie's calories will go into repairing, replacing, and carrying the extra body tissue. After a few years of daily cookie eating, weight gain will level off at approximately 2.7 kg (6 lb).[2] Thus, a one-time step-up in caloric intake will cause body weight to increase asymptotically to a new, stable level.

The converse occurs when an individual reduces food intake. As body size diminishes, so does the amount of fuel needed to maintain and move it, and weight settles at a new steady level. In addition, weight loss produces changes in hormones, the autonomic nervous system, and the intrinsic efficiency of muscle that serve to conserve energy.[3] Therefore, additional weight loss can only be achieved by a more severe diet or a more arduous physical activity routine. Most individuals do the opposite: after having achieved some weight loss, they resume their original diet and exercise habits. Consequently, weight gain recurs rapidly.

How Much Are Americans Overeating?

According to the first National Health and Nutrition Examination Survey (NHANES)—a nationally representative study of the US population—women aged 20 to 29 years had a mean body mass index (BMI) of 23 in the early 1970s.[4] The fourth NHANES, conducted in 1999 to 2002, found that women aged 50 to 59 years (who would have been in their 20s in the original study), had a mean BMI of 29,[4] representing a weight gain of approximately 16 kg (35 lb) in 28 years (Figure, A). How much overeating is needed to gain this amount of weight? Physiologists and physicists have developed mathematical models that accurately predict the effect of a discrete change in energy intake on body weight.[2,5,6] These equations suggest that a young adult woman must increase energy intake by 370 kcal per day to increase BMI from 23 to 29.[2] That increase probably occurs gradually. For example, adding 30 mL (1 oz) of sugar-sweetened beverage and walking 1 minute less per day creates a temporary energy surplus of about 13 kcal/d, leading to a weight gain of 0.6 kg (1.4 lb). Repeating changes in diet and physical activity of this magnitude on an annual basis for 28 years would produce the 370 kcal/d "energy gap" and 16-kg (35-lb) weight gain considered above.

To become obese, a much larger cumulative change in lifestyle would be required. The 90th percentile of BMI is 35 for men aged 50 to 59 years.[7] To reach this degree of adiposity from a BMI of 25 at age 25 years (Figure, B), an individual would need to increase energy intake, decrease physical activity, or both by 680 kcal per day.[2] For obese children, this energy gap is even greater. An adolescent at the 95th percentile of BMI at age 15 to 17 years is approximately 26 kg (58 lb) over ideal body weight.[6] Assuming normal weight at age 5 to 7 years, this individual must overconsume 700 to 1000 kcal every day during this period.[6] It is difficult to determine with certainty how energy intake has changed since the early 1970s, but some studies suggest a per capita increase of up to 500 kcal/d.[8]

Preventing Weight Gain

Obesity is difficult to reverse. But what would it take for a lean young adult to stay that way, instead of gaining about 1 or 2 lbs every year? If the effect of excess energy intake on body weight

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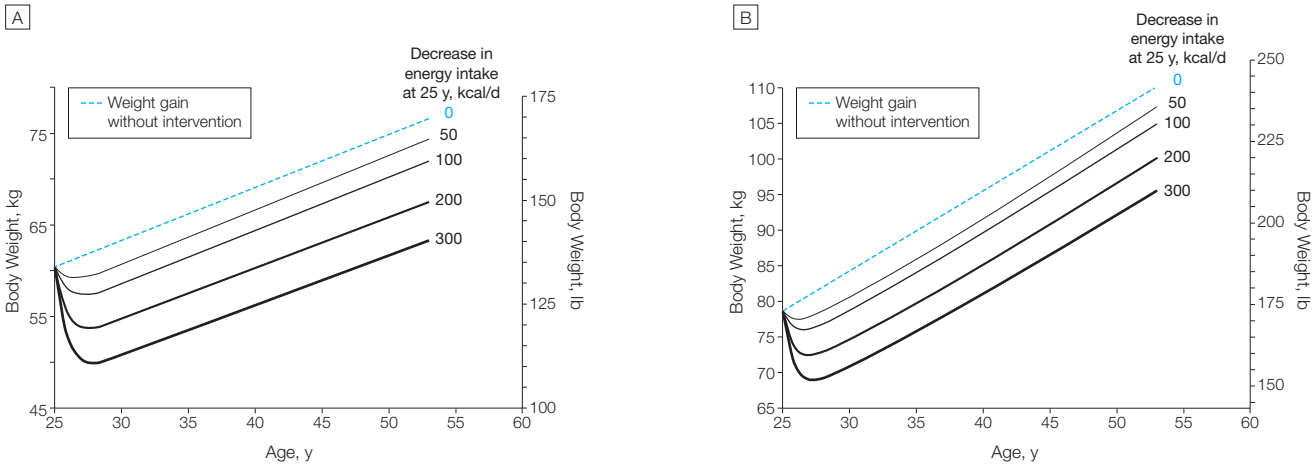
Figure. The Effects of Graded Reductions in Calorie Intake Beginning at Age 25 Years on Body Weight



Solid curves demonstrate the predicted effects of a decrease in energy intake initiated at age 25 years on the weight gain that results from progressive changes in diet and physical activity in 2 situations. Panel A represents deviations from the natural course of weight gain (the dashed line) for the average US women interpolated from National Health and Nutrition Examination Survey (NHANES) I to IV data covering a 28-year period.[4] Panel B represents the hypothetical case of a man aged 25 years whose body mass index increased from 25 to 35 over 28 years (dashed line). Mathematical models were based on Hall et al.[10]

were linear, a small, one-step change in energy balance initiated at age 25 years would be sufficient to prevent overweight by middle age for most individuals.[9] However, any single change in diet or physical activity, even if permanent, will elicit compensatory mechanisms that limit long-term effect on body weight. Since the weight gain experienced by a typical American must be caused by repeated changes in diet, physical activity, or both, a small decrease in food intake or increase in physical activity will halt this increase only temporarily (Figure).

Implications

These calculations suggest that small changes in lifestyle would have a minor effect on obesity prevention. Walking an extra mile a day expends, roughly, an additional 60 kcal compared with resting—equal to the energy in a small cookie. Physiological considerations suggest that the apparent energy imbalance for much of the US population is 5- to 10-fold greater, far beyond the ability of most individuals to address on a personal level. Rather, an effective public health approach to obesity prevention will require fundamental changes in the food supply and the social infrastructure. Changes of this nature depend on more stringent regulation of the food industry, agricultural policy informed by public health, and investments by government in the social environment to promote physical activity.

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REFERENCES

1. Shils ME, Shile M, Ross AC, Caballero B, Cousins RJ, eds. *Modern Nutrition in Health and Disease.* 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006: 982.
2. Hall KD, Jordan PN. Modeling weight-loss maintenance to help prevent body weight regain. *Am J Clin Nutr*. 2008;88(6):1495-1503.
3. Goldsmith RL, Joanisse DR, Gallagher D, et al. Effects of experimental weight perturbation on skeletal muscle work efficiency, fuel utilization, and biochemistry in human subjects [published online November 4, 2009]. *Am J Physiol Regul Integr Comp Physiol*. 2009. doi:10.1152/ajpregu.00053.2009.
4. Ogden CL, Fryar CD, Carroll MD, Flegal KM. Mean body weight, height, and body mass index, United States 1960-2002. *Adv Data*. 2004;347(347):1-17.
5. Weinsier RL, Bracco D, Schutz Y. Predicted effects of small decreases in energy expenditure on weight gain in adult women. *Int J Obes Relat Metab Disord*. 1993; 17(12):693-700.
6. Wang YC, Gortmaker SL, Sobol AM, Kuntz KM. Estimating the energy gap among US children: a counterfactual approach. *Pediatrics*. 2006;118(6):e1721-e1733.
7. McDowell MA, Fryar CD, Hirsch R, Ogden CL. Anthropometric reference data for children and adults: US population, 1999-2002. *Adv Data*. 2005;361(361): 1-5.
8. Swinburn B, Sacks G, Ravussin E. Increased food energy supply is more than sufficient to explain the US epidemic of obesity [published online October 14, 2009]. *Am J Clin Nutr*. 2009;90(6):1453-1456. doi:10.3945/ajcn.2009.28595.
9. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science*. 2003;299(5608):853-855.
10. Hall KD, Guo J, Dore M, Chow CC. The progressive increase of food waste in America and its environmental impact. *PLoS One*. 2009;4(11):e7940. doi:10.1371/journal.pone.0007940.