

The More Food Young Adults Are Served, the More They Overeat¹

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ABSTRACT Young and Nestle suggested that the increase in the portion size of food products evident in the United States during the past 20 years may be responsible for the epidemic of overweight and obesity. They based their conclusion on statistical correlations. The purpose of the present study was to provide experimental evidence to support their proposal. Cornell undergraduate students were given access to a buffet lunch on Monday, Wednesday, and Friday and were told this was a test of flavor enhancers. They were instructed to eat as much or as little as they wanted. On the same days of the following week, the subjects were divided into 3 groups. Each group was served either 100%, 125%, or 150% of the amount of food they had consumed the previous week. When larger amounts were served, significantly greater amounts of food were consumed. Each of the 4 foods that comprised the meal (soup, pasta, breadsticks, ice cream) increased significantly in proportion to the portion size. The data clearly support the hypothesis proposed by Young and Nestle and support the powerful role that environment plays in determining energy intake and potential increases in body weight. *J. Nutr.* 134: 2546–2549, 2004.

KEY WORDS: • portion size • food intake • overeating • humans

There is very little question that the adult population in the United States is gaining body weight (1,2). Along with the increase in body weight, there is an increase in chronic disease (3–8) and the cost of health care (9,10). The degree of increase has reached such proportions that several prominent researchers and public health officials have labeled the trend toward overweight as an epidemic (11–19).

To stop, or even reverse this trend toward increasing body weight, the root causes of the problem must be determined. An increase in body weight can be caused only by an increase in energy intake and/or a decrease in energy expenditure. Although the retrospective data on energy expenditure are difficult to obtain and reflect mainly leisure time activity, there is indication of a decline in energy expended in the U.S. population (20–22). The data demonstrating a trend toward increased daily energy intake are not definitive, but a number of studies suggested that a significant increase in energy consumption has occurred over the past 30 years (23,24). Perhaps the most compelling record showing that the increase in obesity was related to the increase in energy intake appeared in U.S. food disappearance data compiled by the USDA (25). A close examination of these data reveals that daily energy intake remained flat from 1960 until ~1983; then it increased every year in an almost linear fashion. A similar abrupt increase in incidence of obesity occurring in the early 1980s was revealed in data provided by the 4 National Health and

Nutrition Examination Surveys (NHANES) (26). The incidence of obesity remained constant from the first survey performed in the late 1960s until the late 1980s or early 1990s, but increased substantially by the mid-1990s when daily energy intake also began showing the yearly increase. Although food disappearance is not a precise indicator of food consumption, the concomitant rise in the incidence of obesity with the increase in daily energy consumption strongly suggests that yearly trends in food disappearance do reflect changes in food consumption in the population. If this is the case, then research into the cause of the rise in overweight and obesity should focus on the reasons why an increase in daily energy consumption is occurring in the population.

One reason for the increased consumption was recently suggested by Young and Nestle (27). They hypothesized that the trend toward increased obesity is closely related to the increase in the prevalence of large portion sizes being sold to the American public. The data displayed in Figure 1 are drawn from their analysis. The USDA food consumption data are also plotted in this figure. The bars indicate the number of larger-size portions introduced into the U.S. marketplace. The increase in the amount of food consumed appeared when the number of larger-size portions was introduced.

However, their argument does not establish a causal link between the increase in portion size and the amount of food consumed. Few papers have examined this relation and those that were published did not demonstrate a strong relation. Booth et al. (28) presented data showing that the larger the lunch that was served to subjects, the more they ate, but very few details of the study were provided. Edelman (29) did not find an increase in consumption when the amount of the entrée served increased from a small (225 g) to a medium

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portion (426 g), but did observe a small increase in the amount consumed when a large portion (1000 g) was served. Rolls et al. (30) found that the amount of food children >5 y old consumed increased as the amount they were served increased, but that younger children did not show such a relation. More recently, Rolls et al. (31), using a fairly large sample of adult subjects, noted an increase in intake with increasing serving size. However, the slope of the function relating increased intake with increased serving size was flat (0.19 g increase in amount consumed for every additional gram served) indicating a rather small effect. In the present study, we examined the effect of the amount of food served on the amount consumed by young adults.

SUBJECTS AND METHODS

Subjects. The 9 males and 4 female subjects were selected from a larger pool of undergraduate students and staff who were recruited by flyers and class announcements. They were screened for food allergies. Those allergic to any of the foods served in the study were excluded. In addition, the potential subjects were administered the Stunkard restraint scale (32). Only those exhibiting a restraint score <30 were accepted into the study.

The subjects were deceived into thinking that the study was about taste enhancers and the perception of certain foods. They received a debriefing session after the study. The study was approved by the Cornell Human Subjects Committee. Incentives for participation included free lunches, free dinner coupons, and extra credit for a Human Development class.

The weight of the subjects (mean ± SD) was 71.4 ± 16.4 kg, height was 174.3 ± 11.2 cm, and age was 23 ± 8.6 y. Their BMI was 23.2 ± 2.9 kg/m². Weights and heights were measured by staff using medical scales and height measures at the first test session.

Test meal and food intake measurement. On Monday, Wednesday, and Friday of wk 1 (baseline), the subjects were asked to record what they ate for breakfast, midmorning snack, and any beverages they consumed before lunch. They were also asked to record their activity before lunch. Subjects were asked to eat the same foods and maintain the same level of activity they exhibited in wk 1 throughout wk 2 of testing. In addition, subjects completed a 7-point hunger rating scale before and after eating.

Each lunch consisted of water and 4 foods: vegetable soup, rigatoni pasta and tomato sauce, breadsticks, and ice cream. All food was presented on a buffet table. Subjects took each food on separate plates or bowls. The experimental design is shown in Table 1. During wk 1 of testing, the subjects were instructed to take as much or as little as they wished from the buffet table. Each plate of food was weighed by one of the investigators. When subjects indicated that they had completed the meal, the plates were again weighed. The amount consumed was derived from the difference in plate weight. Although subjects could request additional food, they did not do so. They were also told that they were not required to eat everything on their plate. Water was consumed freely without measurement. The mean intake of each food for each subject during wk 1 was used as the baseline to determine the 100, 125, and the 150% condition administered in wk

TABLE 1

Experimental design¹

	Monday	Wednesday	Friday
Week 1	Buffet lunch	Buffet lunch	Buffet lunch
Week 2	Group A (100%)	Group A (150%)	Group A (125%)
	Group B (125%)	Group B (100%)	Group B (150%)
	Group C (150%)	Group C (125%)	Group C (100%)

¹ Percentages refer to portion size compared with the amount previously consumed ad libitum.

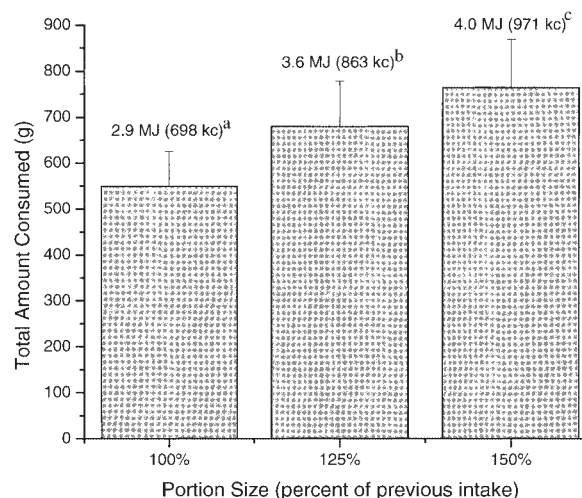


FIGURE 1 The amount of lunch consumed by young adults as a function of portion size. Portion size is the percentage of the amount previously consumed when food was offered in a buffet and consumed ad libitum. Values are means ± SEM, n = 13. Means without a common letter differ, P < 0.05. Energy intakes [mean MJ and kc (kcal)] are given above each bar.

2. The amount of each food in the meal was 100, 125, or 150% of the original amount consumed.

For wk 2, the subjects were randomly assigned to 1 of 3 groups, Group A, Group B and Group C. Each group differed only in the sequence with which they were served the 3 different portion sizes. On each test day, each group received either the 100%, the 125% or the 150% portion size as displayed in Table 1. Each group sat at a different table and received a different portion size. The amount of food was weighed and placed on a separate plate.

Statistical analyses. ANOVA with repeated measures was used to test for statistical significance with an α of 0.05.

RESULTS

Although subjects received the same food 6 times in 2 wk, intake did not decline during the period of testing. Similarly, the total food consumed did not differ among any of the 6 d of testing. No interactions between portion size and test day were observed.

The total grams of food consumed by subjects when they were offered the 3 portion sizes are illustrated in Figure 1. The greater the amount of food subjects were served, the more they consumed, with each portion size significantly different from the others (P < 0.05). The difference in energy intake between the control intake and 125% of the control portion was (mean ± SEM) 0.687 ± 0.131 MJ (165 ± 31 kcal) and the difference between the 125% portion and the 150% portion was 0.24 ± 0.212 MJ (105 ± 51 kcal). Both differences were significant.

The effect of serving large portion sizes was evident for all 4 components of the meal. Increasing portion size increased the amount of each of the 4 foods consumed (P < 0.01; Fig. 2).

The amount of food consumed was not associated with either the absolute hunger rating or the reduction in the hunger ratings. Males and females did not differ, but the sample contained only 4 females, severely limiting the statistical power to detect any effect of gender.

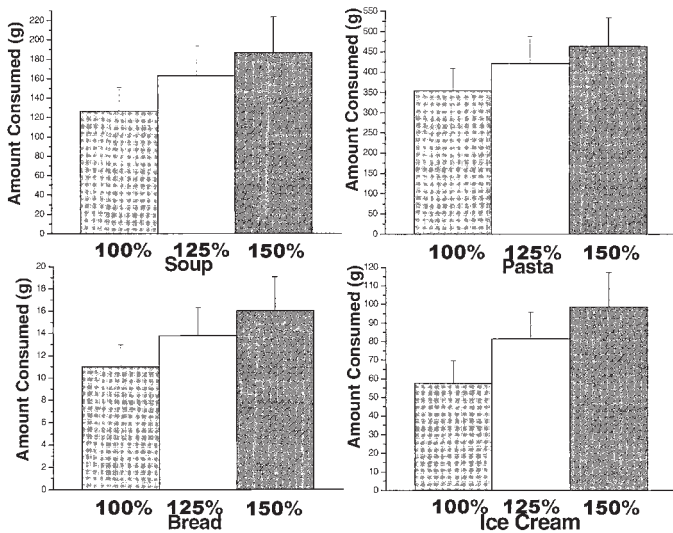


FIGURE 2 The amount of each food consumed by young adults as a function of portion size. Portion size is the percentage of the amount previously consumed when food was offered in a buffet and consumed ad libitum. Values are means \pm SEM, $n = 13$. For each food, the portion sizes differed, $P < 0.05$.

DISCUSSION

The data from the present study are consistent with the previous findings in adults (28,31,33,34) and in older children (30): serving larger portions results in eating more food. These data are also consistent with studies that have observed the consumption of foods is directly related to the size of the container in which they were served (35,36). These data, therefore, strongly support the argument made by Young and Nestle (27) that increased serving size could be a major factor responsible for the increase in overweight and obesity that is so evident today.

One reason why the relation between amount served and amount consumed is much stronger in the present study than that observed by Rolls et al. (31) may be that the size of the portions offered were considerably smaller and were based on the amount of food the subjects actually served themselves. Moreover, a close examination of the plot of the amount consumed as a function of the amount served suggested that a maximum amount ingested may occur at ~ 450 – 500 g. Three of the 4 portions tested by Rolls et al. (31) were >500 g. It is interesting that Gelibter and Hashim (37) found that the maximum volume of the stomach of adults with no eating disorder is ~ 500 mL. Given that the density of most foods is ~ 1 kg/L, it is not unreasonable to suggest that a comfortable upper limit of food ingested may be ~ 500 g.

There are several factors concerning this study that limit the ability to generalize the results to the general population. First, “normal” intake may have been underestimated. The baseline amount of food consumed was determined during wk 1 of the study by measuring the amount of food subjects ate when they obtained their food from a buffet table. Most of the subjects were not familiar with other subjects in the study. Clendenen et al. (38) showed that college age students tend to eat less food when they eat in the presence of strangers than when they eat with friends. However, the mean amount the subjects consumed for lunch was well within the amounts normally consumed for lunch by subjects of this particular age, weight, and lifestyle.

The second limitation of the present study concerns the

population. The college age population used in this study displays considerably greater weight gain than the general population (39), frequently referred to as the “Freshman 15.” In addition, these subjects, Cornell undergraduate students, regularly eat in “all-you-can-eat” dining halls and may be more comfortable than the general population consuming as much food as is available.

A third limitation of the study is that the subjects ate in groups and may have been subjected to a “social facilitation” effect of eating in the presence of others. Although social facilitation increases the amount of food consumed (38,40–43), there is no indication from the literature that social facilitation interacts with portion size in determining an individual’s intake. However, the function that relates intake to portion size (Fig. 1) may not be generalized to situations in which individuals eat alone or in larger groups. In a similar vein, because the subjects ate together, watching others eat may have affected the intake/portion size function. Although food modeling by others has been demonstrated to significantly affect food choice (44–47), it has not been shown to affect the amount of food a person consumes.

The fourth, and perhaps most important limitation of the present study, is that the effect of increasing portion size was limited to the observation of a single meal. There are no published data examining the chronic effects of serving larger portion size on the amount consumed at the subsequent meal. It is possible that consuming a large meal may inhibit food consumption at later meals. However, there are data showing that the amount eaten at breakfast or at between-meal snacks does not affect the amount consumed at subsequent meals (48).

It should also be noted that the sample size (13 subjects) was relatively small. This limitation in sample size minimizes the power necessary to discern an effect of gender or other characteristics of the subjects that would have yielded more insight into the nature of the effect of portion size on intake.

Is it unrealistic to think that the effect of increasing portion size is sufficiently powerful to be a serious cause of the “Epidemic of Obesity?” Using USDA Consumption and Family Living data (49), we estimated that the rate of increase in daily energy intake during the last 20 years, when the rise in overweight and obesity occurred, was only 0.12 MJ (29.6 kcal)/d. Hill et al. (20), using other methods, estimated the median increase in intake at ~ 15 kcal/d. The present study indicated that increasing the portion size of a standard size lunch by 50% (Fig. 2) produced an increment in energy intake of 1.1 MJ (273 kcal). Therefore, the present data suggest that the magnitude of the portion size effect on energy consumption in adults is sufficient to account for the increase in population weight that occurred over the past 30 years.

At a theoretical level, these data provide strong support for the “Settling-Zone” theory of the regulation of body weight (48). This idea suggests that biological factors determine a “Settling-Zone” or a fairly wide range of body weights within which body weight is not regulated. Body weight within the “Settling-Zone” is solely a result of environmental factors that influence energy intake and energy expenditure. Portion size may be a good example of an environmental factor that may profoundly affect body weight within this “Settling-Zone.”

The results of the present study, as well as others, support the hypothesis proposed by Young and Nestle (27) that a major reason for the increased incidence of overweight and obesity observed in the United States is the increased food consumption caused by serving larger portion sizes. The greater the quantity of food served to our subjects, the more they ate. From a public health perspective, the results of this

study are extremely encouraging. If Young and Nestle (27) are correct that the increase in portion size is a major cause of the "Epidemic of Obesity," then it should be possible to stop and possibly reverse this trend toward increased body weight by controlling the size of portions served to the American people.

LITERATURE CITED

- Galuska, D. A., Serdula, M., Pamuk, E., Siegel, P. Z. & Byers, T. (1996) Trends in overweight among US adults from 1987 to 1993: a multistate telephone survey. *Am. J. Public Health* 86: 1729-1735.
- Flegal, K. M., Carroll, M. D., Kuczmarski, R. J. & Johnson, C. L. (1998) Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int. J. Obes. Relat. Metab. Disord.* 22: 39-47.
- Colditz, G. A. & Coakley, E. (1997) Weight, weight gain, activity, and major illnesses: the Nurses' Health Study. *Int. J. Sports Med.* 18 (suppl. 3): S162-S170.
- Hu, F. B., Salmeron, J., Manson, J. E., Stampfer, M. J., Colditz, G. A., Rimm, E. B. & Willett, W. C. (1999) Dietary fat intake and risk of type 2 diabetes in women. *Am. J. Epidemiol.* 149: 3.
- Wolk, A., Manson, J. E., Stampfer, M. J., Colditz, G. A., Hu, F. B., Speizer, F. E., Hennekens, C. H. & Willett, W. C. (1999) Long-term intake of dietary fiber and decreased risk of coronary heart disease among women. *J. Am. Med. Assoc.* 281: 1998-2004.
- Everson, S. A., Goldberg, D. E., Helmrich, S. P., Lakka, T. A., Lynch, J. W., Kaplan, G. A. & Salonen, J. T. (1998) Weight gain and the risk of developing insulin resistance syndrome. *Diabetes Care* 21: 1637-1643.
- Diaz, M. E. (2002) Hypertension and obesity. *J. Hum. Hypertens.* 16: S18-S22.
- Adelman, R. D. (2002) Obesity and renal disease. *Curr. Opin. Nephrol. Hypertens.* 11: 331-335.
- Eisenstein, E. L., Shaw, L. K., Nelson, C. L., Anstrom, K. J., Hakim, Z. & Mark, D. B. (2002) Obesity and long-term clinical and economic outcomes in coronary artery disease patients. *Obes. Res.* 10: 83-91.
- Colditz, G. A. (1999) Economic costs of obesity and inactivity. *Med. Sci. Sports Exerc.* 31: S663-S667.
- Katzmarzyk, P. T. (2002) The Canadian obesity epidemic: an historical perspective. *Obes. Res.* 10: 666-674.
- Kumanyika, S. (2002) The minority factor in the obesity epidemic. *Ethn. Dis.* 12: 316-319.
- Korman, L. (2002) The growing epidemic of obesity. *N. J. Med.* 99: 30-39; quiz 39, 48.
- Mokdad, A. H., Bowman, B. A., Ford, E. S., Vinicor, F., Marks, J. S. & Koplan, J. P. (2001) The continuing epidemics of obesity and diabetes in the United States. *J. Am. Med. Assoc.* 286: 1195-1200.
- Mokdad, A. H., Serdula, M. K., Dietz, W. H., Bowman, B. A., Marks, J. S. & Koplan, J. P. (1999) The spread of the obesity epidemic in the United States, 1991-1998. *J. Am. Med. Assoc.* 282: 1519-1522.
- Dietz, W. H. (2001) The obesity epidemic in young children. Reduce television viewing and promote playing. *Br. Med. J.* 322: 313-314.
- Jeffery, R. W. & French, S. A. (1998) Epidemic obesity in the United States: are fast foods and television viewing contributing? *Am. J. Public Health* 88: 277-280.
- Hall, J. E. & Jones, D. W. (2002) What can we do about the "epidemic" of obesity. *Am. J. Hypertens.* 15: 657-659.
- Wadden, T. A., Brownell, K. D. & Foster, G. D. (2002) Obesity: responding to the global epidemic. *J. Consult. Clin. Psychol.* 70: 510-525.
- Hill, J. O., Wyatt, H. R., Reed, G. W. & Peters, J. C. (2003) Obesity and the environment: where do we go from here? *Science (Washington, DC)* 299: 853-855.
- Caspersen, C. J. & Merritt, R. K. (1995) Physical activity trends among 26 states, 1986-1990. *Med. Sci. Sports Exerc.* 27: 713-720.
- Brooks, C. (1988) Adult physical activity behavior: a trend analysis. *J. Clin. Epidemiol.* 41: 385-392.
- Harnack, L. J., Jeffery, R. W. & Boutelle, K. N. (2000) Temporal trends in energy intake in the United States: an ecologic perspective. *Am. J. Clin. Nutr.* 71: 1478-1484.
- Nielsen, S. J., Siega-Riz, A. M. & Popkin, B. M. (2002) Trends in energy intake in U.S. between 1977 and 1996: similar shifts seen across age groups. *Obes. Res.* 10: 370-378.
- U.S. Department of Agriculture (2001) *Agricultural Statistics 2002*. U.S. Government Printing Office, Washington, DC.
- Kuczmarski, R. J., Flegal, K. M., Campbell, S. M. & Johnson, C. L. (1994) Increasing prevalence of overweight among US adults. The National Health and Nutrition Examination Surveys, 1960 to 1991. *J. Am. Med. Assoc.* 272: 205-211.
- Young, L. R. & Nestle, M. (2002) The contribution of expanding portion sizes to the US obesity epidemic. *Am. J. Public Health* 92: 246-249.
- Booth, D., Juller, J. & Lewis, V. (1981) Human control of body weight: cognitive or physiological? Some energy-related perceptions and misperceptions. In: *The Body Weight Regulatory System: Normal and Disturbed Mechanisms* (Cioffi, L., James, W. & Van Itallie, T., eds.), pp. 305-314. Raven Press, New York, NY.
- Edelman, B., Engell, D., Bronstein, P. & Hirsch, E. (1986) Environmental effects on the intake of overweight and normal-weight men. *Appetite* 7: 71-83.
- Rolls, B. J., Engell, D. & Birch, L. L. (2000) Serving portion size influences 5-year-old but not 3-year-old children's food intakes. *J. Am. Diet. Assoc.* 100: 232-234.
- Rolls, B. J., Morris, E. L. & Roe, L. S. (2002) Portion size of food affects energy intake in normal-weight and overweight men and women. *Am. J. Clin. Nutr.* 76: 1207-1213.
- Stunkard, A. J. & Messick, S. (1985) The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J. Psychosom. Res.* 29: 71-83.
- Kral, T. V., Roe, L. S. & Rolls, B. J. (2004) Combined effects of energy density and portion size on energy intake in women. *Am. J. Clin. Nutr.* 79: 962-968.
- Rolls, B. J., Roe, L. S., Meengs, J. S. & Wall, D. E. (2004) Increasing the portion size of a sandwich increases energy intake. *J. Am. Diet. Assoc.* 104: 367-372.
- Rolls, B. J., Roe, L. S., Kral, T. V., Meengs, J. S. & Wall, D. E. (2004) Increasing the portion size of a packaged snack increases energy intake in men and women. *Appetite* 42: 63-69.
- Wansink, B. (2004) Environmental factors that increase the food intake and consumption volume of unknowing consumers. *Annu. Rev. Nutr.* 24: 455-479.
- Geliebter, A. & Hashim, S. A. (2001) Gastric capacity in normal, obese, and bulimic women. *Physiol. Behav.* 74: 743-746.
- Clendenen, V. I., Herman, C. P. & Polivy, J. (1994) Social facilitation of eating among friends and strangers. *Appetite* 23: 1-13.
- Graham, M. A. & Jones, A. L. (2002) Freshman 15: valid theory or harmful myth? *J. Am. Coll. Health* 50: 171-173.
- Feunekes, G. I., de Graaf, C. & van Staveren, W. A. (1995) Social facilitation of food intake is mediated by meal duration. *Physiol. Behav.* 58: 551-558.
- DeCastro, J. (1990) Social facilitation of duration and size but not rate of the spontaneous meal intake of humans. *Physiol. Behav.* 47: 1129-1135.
- Redd, M. & De Castro, J. M. (1992) Social facilitation of eating: effects of social instruction on food intake. *Physiol. Behav.* 52: 749-754.
- DeCastro, J. M. (1994) Family and friends produce greater social facilitation of food intake than other companions. *Physiol. Behav.* 56: 445.
- Birch, L. L. (1980) Effects of peer models' food choices and eating behaviors on preschoolers' food preferences. *Child Dev.* 51: 489-496.
- Birch, L. L. & Billman, J. (1986) Preschool children's food sharing with friends and acquaintances. *Child Dev.* 57: 387-395.
- Hobden, K. & Pliner, P. (1995) Effects of a model on food neophobia in humans. *Appetite* 25: 101-113.
- Hendy, H. M. & Raudenbush, B. (2000) Effectiveness of teacher modeling to encourage food acceptance in preschool children. *Appetite* 34: 61-76.
- Levitsky, D. A. (2002) Putting behavior back into feeding behavior: a tribute to George Collier. *Appetite* 38: 143-148.
- USDA (2004) *Agricultural Statistics 2004*. United States Government Printing Office, Washington, DC.