CORRESPONDENCE

The Spread of Obesity in a Social Network

TO THE EDITOR: In the report on the spread of obesity in a social network, by Christakis and Fowler, and the accompanying editorial by Barabási (July 26 issue),[1,2] variables of social and economic status (SES) are not mentioned. Social networks conceal a high degree of social homogeneity — that is, people are more likely to network with people at similar income and educational levels — and we suspect that they may operate differently in different contexts. For example, the way a social network affects the incidence of obesity in a community where most people have a low educational level and cannot afford a healthy diet is likely to differ from the way it affects an affluent and well-informed community. In 2000, obesity in the United States was reported to be highest among black women and people who had not completed high school,[3] and among black women, a low socioeconomic position predicts an early onset of obesity.[4] SES variables should be included in any interpretive model, and public health policies for the prevention of obesity should take into account the SES characteristics of the communities they are supposed to benefit.[5]

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1. Christakis NA, Fowler JH. The spread of obesity in a large social network over 32 years. N Engl J Med 2007;357:370-9.
2. Barabási A-L. Network medicine — from obesity to the "diseasesome." N Engl J Med 2007;357:404-7.
3. Freedman DS, Khan LK, Serdula MK, Galuska DA, Dietz WH. Trends and correlates of class 3 obesity in the United States from 1990 through 2000. JAMA 2002;288:1758-61.
4. Bennett GG, Wolin KY, James SA. Lifecourse socioeconomic position and weight change among blacks: the Pitt County study. Obesity (Silver Spring) 2007;15:172-81.
5. Stafford M, Cummins S, Ellaway A, Sacker A, Wiggins RD, Macintyre S. Pathways to obesity: identifying local, modifiable determinants of physical activity and diet. Soc Sci Med (in press).

TO THE EDITOR: Christakis and Fowler describe how weight gain occurs in social clusters and stress that people are influenced by the appearance and behaviors of others. In behavioral economics, this mechanism is referred to as "anchoring," meaning that judgments are based not on absolute values but on comparison with implicit reference points ("anchors").[1] We collected data on the sense of urgency with respect to weight control that show how subtle and powerful anchoring can be. With the use of different anchors for the same scenario, 154 subjects were asked to judge the importance of taking action concerning their weight if 35% of the population was heavier than they themselves were (scenario A) versus the importance of taking action if 65% of the population was thinner than they were (scenario B). The importance of weight control was judged to be significantly lower

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(P<0.001 by a paired t-test) in scenario A, in which subjects implicitly compared themselves with heavier people. This finding illustrates how anchoring may have contributed to the clustering of obesity but also raises concern that increasing weight in the general population may increase our anchors — the implicit reference points for what we perceive as normal. This could turn overweight in society into a self-reinforcing process.

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1. Tversky A, Kahneman D. Judgment under uncertainty: heuristics and biases. Science 1974;185:1124-31.

TO THE EDITOR: I believe that the article on the spread of obesity in a large social network has important public health implications. It is also of concern for those of us involved in health technology assessment and firm believers in the advantages of randomized, controlled trials.

The study focused on obesity, but the findings probably hold for nearly all types of lifestyle interventions. By using the concept of etiologic fraction and using data on smoking behavior among smokers and nonsmokers, I previously estimated that for every 10 people who stop smoking, there will be another 2 who in the long run do not smoke as a consequence,[1] an indirect effect of 20%. Trends toward a decline in smoking around the world have surely been reinforced by social diffusion.

It is obvious that randomized, controlled trials with individual allocations do not take these indirect social effects into consideration and thereby underestimate the effects. Health technology assessments have so far not considered these indirect effects, and organizations such as the one I represent may have somewhat hampered the development of lifestyle interventions.

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1. Rosén M. On randomized controlled trials and lifestyle interventions. Int J Epidemiol 1989;18:993-4.

THE AUTHORS REPLY: We investigated the interpersonal spread of obesity as a possible factor in the obesity epidemic. Tamburlini and Cattaneo are concerned about the role of SES. Our reported analyses did account for education. Additional analyses (not reported in the article) also accounted for income. Adjustment for either or both of these measures did not alter our finding that weight gain in one person was associated with weight gain in others. Moreover, we found no significant difference in the likelihood of spread according to whether the level of education was high or low.

Knecht et al. provide very nice evidence for the effects of both "framing" and "anchoring." The experiment they describe shows the importance of how framing the context of an individual respondent affects responses (i.e., the otherwise equivalent description of the group as being composed of 35% of people who are heavier than the respondent versus 65% who are lighter). Even more telling results, we suspect, would arise if the scenarios were manipulated so that 45% or 55% or 65% of the group was described as being lighter than the respondent.[1] As suggested in our report, we suspect that increasing weight gain in a population may affect reference points for what people perceive as "normal" weight,[2] and this might be one mechanism for the interpersonal spread of obesity. Moreover, personal assessments of weight vary, perhaps in keeping with the sociodemographic group to which a person belongs.[3]

We agree with Rosén both with respect to the likely relevance of our findings to other health behaviors (e.g., smoking) and also with respect to the relevance of our findings for technology assessment. The existence of interpersonal health effects has substantial implications for the analysis of health policy, since outcomes in individuals to whom a person is connected should, in many situations, be included in enumerating costs and benefits of interventions.[4] This is not limited solely to lifestyle interventions: replacing a hip, preventing a stroke, or curing a cataract in one person may reduce the disability not only of that person but also of his or her spouse, for example.

Social networks are relevant to health and health care. One point worth emphasizing, however, is that social support is well known to be important.[5] It is therefore unlikely that severing ties with people on the basis of any of their par-

ticular traits — as some have supposed that our results might suggest — would necessarily be beneficial.

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1. Solnick SJ, Hemenway D. Is more always better? A survey of positional concerns. J Econ Behav Organ 1998;37:373-83.
2. Burke MA, Heiland MA. Social dynamics of obesity. Econ Inq 2007;45:571-91.
3. Chang VW, Christakis NA. Self-perception of weight appropriateness in the United States. Am J Prev Med 2003;24:332-9.
4. Christakis NA. Social networks and collateral health. BMJ 2004;329:184-5.
5. Berkman LF, Syme SL. Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda County residents. Am J Epidemiol 1979;109:186-204.

Partial Thrombosis of the False Lumen in Aortic Dissection

TO THE EDITOR: The main limitation of the study of a large cohort of patients with type B acute aortic dissection from the International Registry of Acute Aortic Dissection (IRAD), reported by Tsai et al. (July 26 issue),[1] is the exclusion of patients who died in the hospital. Fifty patients (24.9% of the 201 patients examined) died within 3 years after discharge from the hospital, whereas 66 patients must have died in the hospital because 466 of the 532 patients enrolled in the IRAD who had type B acute aortic dissection were discharged from the hospital alive. In a previous study of 384 patients with type B acute aortic dissection from the IRAD,[2] the in-hospital mortality among patients with a patent false lumen, those with a partially thrombosed false lumen, and those with a completely thrombosed false lumen was 12.7%, 12.1%, and 8.3%, respectively (P=0.71). We would like to know the false-lumen status of the 66 patients who were excluded from the study by Tsai et al. and would like to know the midterm mortality with the in-hospital mortality included rather than excluded.

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1. Tsai TT, Evangelista A, Nienaber CA, et al. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. N Engl J Med 2007;357:349-59.
2. Suzuki T, Mehta RH, Ince H, et al. Clinical profiles and outcomes of acute type B aortic dissection in the current era: lessons from the International Registry of Aortic Dissection (IRAD). Circulation 2003;108:Suppl II:II-312–II-317.

TO THE EDITOR: Tsai et al. report an independent adverse relationship between partial thrombosis of the false lumen and long-term mortality among hospital survivors of acute type B aortic dissection. However, focusing on postdischarge mortality rather than total mortality (i.e., both in-hospital and postdischarge mortality) provides an incomplete view. Hypothetically, there is less time for thrombosis of the false lumen among patients who die in the hospital. This may seem to contradict the theory proposed by Tsai et al. Do the authors have any information on this important subgroup of patients to either substantiate or refute their hypothesis? Furthermore, according to their conceptual risk model, the group of patients who had complete thrombosis of the false lumen should have had the lowest mortality during follow-up. However, this was true only up to about 600 days of follow-up, after which there was a rather steep rise in the mortality rate observed only in this group. This suggests that there were probably other, unexplained factors contributing to late mortality besides false-lumen status.

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THE AUTHORS REPLY: Takagi et al. request information on the false-lumen status of the 66 patients who were excluded from the study because they died in the hospital. Of these patients, 39 (59.1%) had a patent false lumen, 19 (28.8%) had partial thrombosis of the false lumen, and 8 (12.1%) had complete thrombosis of the false lumen. This distribution closely resembles the distribution of false-lumen status in the study population. With the inclusion of these patients in the survival analysis, the mean (±SD) 3-year mortality rate for patients with a patent false lumen was 14.7±7.3%, for those with partial thrombosis it was 32.4±12.2%, and for