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Citation	Fowler, James H., and Nicholas A. Christakis. 2008. Estimating peer effects on health in social networks. <i>Journal of Health Economics</i> 27, no. 5: 1400-1405.
Published Version	doi:10.1016/j.jhealeco.2008.07.001
Accessed	February 17, 2015 11:12:41 PM EST
Citable Link	http://nrs.harvard.edu/urn-3:HUL.InstRepos:3685821
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Estimating Peer Effects on Health in Social Networks

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Journal of Health Economics 27(5): 1400-1405 (September 2008)

We recently showed that obesity can spread socially from person to person in adults (Christakis and Fowler 2007). A natural question to ask is whether or not these results generalize to a population of adolescents. Three separate teams of researchers have analyzed the National Longitudinal Study of Adolescent Health (Add Health) and shown evidence of person-to-person spread of obesity, but they use different methods and disagree on the interpretation of their results. Here, we conduct our own analysis of the Add Health data, provide additional evidence from the Framingham Heart Study on the social spread of obesity, and use Monte Carlo simulations to test the econometric methods we use to model peer effects. The results show that the existence of peer effects in body mass is robust to several specifications in both adults and in adolescents.

The most recent and complete version of this article can be found at the contact author's website <http://jhfowler.ucsd.edu>. For questions, email jhfowler@ucsd.edu. This work was supported in part by National Institute of Aging grant P-01 AG-031093.

In July of 2007, we published a paper in the *New England Journal of Medicine* that used dynamic data over 32 years from the Framingham Heart Study Social Network (FHS-Net) to study the conditions under which obesity might spread from person to person (Christakis and Fowler 2007, hereafter, CF). We found that obese persons formed clusters in the network at all time points, and these clusters extended to three degrees of separation (e.g., to a person's friend's friend's friend). Moreover, statistical analyses suggested that the clusters were not solely attributable to the selective formation of social ties among obese persons. A person's chances of becoming obese increased if he or she had a friend who became obese in a given time period.

Our analyses were restricted to adults, so a natural question to ask is whether or not the results would generalize to a population of adolescents. The existence of social norms regarding weight in both adults and adolescents should not be surprising, (Chang and Christakis 2003) but, of course, causal inference in dyads, let alone in broader social networks, is difficult (Manski 1993). These questions are addressed by two papers in this issue, "Is Obesity Contagious? Social Networks versus Environmental Factors in the Obesity Epidemic," by Ethan Cohen-Cole and Jason Fletcher (hereafter CCF) and "Peer Effects in Adolescent Overweight," by Justin Trogdon, James Nonnemaker, and Joanne Pais (hereafter TNP), and by a third working paper "Identifying Endogenous Peer Effects in the Spread of Obesity" by Timothy J. Halliday and Sally Kwak (hereafter HK). All three of these papers analyze the same dataset and population, the National Longitudinal Study of Adolescent Health (Add Health), albeit with different methods and assumptions. Unlike the FHS-Net, which followed adults over a 32-year period (average age 38 in

1971), Add Health followed adolescents (average age 16 in 1995) into young adulthood over a 7-year period.

All three sets of authors take the possibility of peer effects seriously, advancing the study of this important area. HK summarize their results by noting that they are able to replicate the pattern of results in our study, although their results are sensitive to specification of the dependent variable. If weight is characterized by a dichotomous variable indicating overweight ($BMI > 25$), then an association between friends is significant, but the association in the continuous measure of BMI is not. Similarly, TNP use a variety of econometric strategies to conclude that there are peer effects for obesity in the Add Health sample, especially among females and among adolescents with high BMI. For example, they use an instrumental variables approach and a variety of definitions of endogenous peer groups to control for contextual effects. Here, we particularly address the CCF paper since it is the only one of the three papers that claims to reject the hypothesis that weight status can spread from person to person. To their credit, CCF exploit longitudinal data in a way that TNP and HK do not, but there are important problems in both their analysis and their interpretation of the results.

Getting the Null Right in CCF

In our view, CCF make a classic error in interpreting their own results. CCF seem to be single-mindedly focused on whether one can reject the null hypothesis of zero regarding whether weight gain in a social contact can cause weight gain in an ego. In three of their five specifications, they actually do replicate the approximate magnitude and the significance of the result from Framingham, which is quite remarkable.

However, they interpret their remaining two results as a rejection of the Framingham result because they cannot reject the zero-null hypothesis.

What is crucial to note here, however, is that the Framingham estimates actually fall inside the confidence intervals of the CCF results *for all five specifications*. That is, CCF cannot reject the null hypothesis that the Framingham estimates are the true values. To put it even more starkly, there is a reasonable probability that the difference in the CCF and Framingham results is merely due to chance. Therefore, an alternative interpretation of their results is that they are actually supportive of the Framingham results (and also consonant with the conclusion reached in TNP and HK).

Moreover, as we will show below, the CCF results reflect a number of idiosyncratic and problematic econometric choices, and there are good empirical and theoretical reasons to be skeptical of their skepticism.

Contextual Effects in the Add Health Data

We attempted to replicate the CCF results but were unable to do so, even after several efforts to acquire information about specific details of CCF's modeling choices from CCF. We first relied on their manuscript and supplement, but it gives an incomplete description of the model; for example, it was unclear how income data were imputed. We then contacted CCF to ask for the code they used to generate their results. They were unwilling to share this code.

We therefore wrote new code to analyze the Add Health data to replicate their results; we sent it to them and asked them to comment. A brief response from CCF stated that a “difference in our approaches is that we ‘lock in’ Wave 1 friends—that is, we do

not allow individuals to switch friends over time.” This is an important and noteworthy omission from their description in the paper since Add Health (like the FHS-Net data) contains *dynamic* information about friendships at later waves, yet they chose instead to ignore this information and rely on a *static* representation of that data. This will cause them to assume some individuals continue to be friends when, in fact, they are not. This assumption by CCF stacks the deck against finding an effect, since it essentially adds “random” non-friend relationships (i.e., people who are no longer friends) to the pool of friends.

Since the CCF results cannot be replicated, and since they are in any case inconsistent with the conclusions of TNP and HK, we present our own results here based on the Add Health data, in an effort to figure out what is going on. CCF use only the first friend named by each subject, but when we interact the friend list order with alter’s contemporaneous obesity, we cannot reject the hypothesis that order is irrelevant to the strength of the effect. We therefore use all observed friendships to maximize efficiency. CCF impute missing income and education data, but it is unclear how they do this. We use expectation maximization with importance sampling to impute all missing data (King et al. 2001). This procedure is widely used and well known to reduce bias relative to alternatives such as listwise deletion (Rubin 2004, King et al. 2001). Like CCF, we also include variables for age, gender, race, ethnicity, and a fixed effect for the wave of the observation. And, as already noted, we use dynamic friendship data available from Waves 1, 2, and 3 to update friendships at each wave, rather than making the assumption made by CCF that friendships are static and all people who became friends in junior high school retain all of their relationships into adulthood.

Table 1. Replication of Add Health Results Show School Trends Do Not Matter and Show Evidence of Directional Effects

	Model 1: Alter is the Named Friend			Model 2: Effect of Named Friend with School Trends			Model 3: Alter is the Person who Named the Friend		
	Coef.	S.E.	p	Coef.	S.E.	p	Coef.	S.E.	p
<i>Contemporaneous</i>									
<i>Alter BMI</i>	0.033	0.014	0.022	0.033	0.014	0.020	0.002	0.014	0.900
<i>Alter Previous BMI</i>	-0.004	0.015	0.762	-0.005	0.015	0.759	0.002	0.015	0.908
<i>Ego Previous BMI</i>	0.952	0.015	0.000	0.952	0.015	0.000	0.937	0.016	0.000
<i>Ego Gender</i>	-0.129	0.085	0.129	-0.132	0.085	0.121	-0.176	0.100	0.078
<i>Ego Age</i>	-0.079	0.030	0.008	-0.079	0.030	0.008	-0.071	0.033	0.033
<i>Ego Hispanic</i>	0.185	0.139	0.182	0.179	0.139	0.198	0.029	0.147	0.845
<i>Ego Black</i>	0.225	0.120	0.060	0.225	0.120	0.060	0.192	0.136	0.158
<i>Wave</i>	2.736	0.158	0.000	2.725	0.159	0.000	2.699	0.174	0.000
<i>Maternal Education</i>	-0.041	0.020	0.037	-0.041	0.020	0.035	-0.023	0.021	0.279
<i>Family Income</i>	0.000	0.001	0.642	0.000	0.001	0.652	-0.001	0.001	0.425
<i>School Trends</i>				0.037	0.032	0.254			
<i>Constant</i>	-2.874	0.537	0.000	-2.854	0.540	0.000	-2.001	0.639	0.002
<i>Deviance</i>	55734			55732			27782		
<i>Null Deviance</i>	240646			240646			107965		
<i>N</i>	8504			8504			4742		

Table 1 presents the results. In model 1, notice that the coefficient of 0.033 on alter’s contemporaneous obesity is significant ($p=0.02$). In model 2, we add school trends as suggested by CCF. Not only does this addition have no effect on the induction effect, it also fails to achieve significance (CCF do not report the coefficient or standard error for their school trend effect, so we cannot compare results). However, what is clear is that the addition of school trends does not matter in a model that uses all available data.

CCF appear dismissive of evidence that the induction effect in our Framingham data is *directional*. That is, if Mark names John as a friend, we expect John to have an effect on Mark. However, if John does not reciprocate by naming Mark as a friend, then John may not be affected by Mark’s opinions or health behaviors. We hypothesize that influence flows from the named friend to the person who named him, but not necessarily vice versa, and in Framingham we find exactly that. The named friend has a significant

effect on the namer but the namer has no significant effect on the named. New work in the econometrics of networks confirms that exploiting directionality in networks is a useful identification strategy (Bramouille, Djebbari, and Fortin 2007). Fortunately, Add Health also allows us to test the directional hypothesis. In model 3 of Table 1, we show that there is no evidence that named friends are influenced by namers ($p=0.90$), confirming our results in the FHS-Net and providing additional evidence in favor of the causal interpretation regarding social influence in weight behaviors.

It is interesting to note that CCF did not report this result, since it speaks directly to their main point. If contextual effects are spuriously driving the relationship between ego and alter, then there is no reason to expect a directional result. The context should cause the named friend and the namer to move up and down simultaneously; hence, if we find a significant effect in one direction, we should also find it in the other: the namer should appear to have an influence on the named friend. Since we do not find such a significant effect, we believe the evidence in Add Health is suggestive of a causal effect, just as it is in Framingham. Obesity appears to spread from person to person.

CCF assert that our model specification in Framingham does not capture any contextual effects that vary across geographic space. It is important to note that friends in Add Health are all physically proximate (they are in the same school), whereas they are not in Framingham. If our estimates are biased because they capture community-level correlation, one implication is that the increased distance between friends will reduce the effect size (since distant friends are not contemporaneously affected by community-level variables). We specifically find that the relationship does *not* decay with physical distance, even up to hundreds of miles away (Fig. 3 in CF). We strongly emphasize this

point in our article, and it was widely reported in the popular press, so it is difficult to understand how CCF could have missed this. The implication of this observation is that any contextual effects that are geographic in nature probably do not have an effect on the association between ego's and alter's obesity. In addition, as reported in CF, we also analyzed whether there was a relationship in weight behaviors between individuals and their immediate neighbors living at adjoining housing units. If contextual effects were driving the association in weight between friends, we would expect neighbors to appear to have an effect on each other, but we found that they do not.

Finally, in this regard, it is also worth noting that in Framingham we include dummy variables as controls for each exam, which effectively controls for average weight change in the whole population (time-specific effects). This might not capture regional variation within time blocks, but we also control for ego-specific factors that would account for some regional variation, including age, gender, and education.

Fixed Effects in the Framingham Heart Study

Adding fixed effects to dynamic panel models with many subjects and few repeat observations creates severe bias towards zero coefficients. This has been demonstrated both analytically (Nickell 1981) and through simulations (Nerlove 1971) for OLS and other regression models and has been well-known by social scientists, including economists, for a very long time. In fact, CCF even note that they do not add fixed effects to their logit regression model for this reason, but they strangely assert that fixed effects are necessary in the OLS model.

Table 2. New Model of Framingham Heart Study Social Network Data Shows Framingham BMI Result is Robust to the Inclusion of Ego Fixed Effects

	Alter is the Named Friend		
	<i>Coef.</i>	<i>S.E.</i>	<i>p</i>
<i>Contemporaneous Alter BMI</i>	0.053	0.018	0.003
<i>Alter Previous BMI</i>	-0.016	0.019	0.387
<i>Ego Previous BMI</i>	0.209	0.020	0.000
<i>Ego Age</i>	0.134	0.075	0.073
<i>Wave 3</i>	-0.192	0.346	0.578
<i>Wave 4</i>	-0.308	0.603	0.610
<i>Wave 5</i>	-0.373	0.874	0.670
<i>Wave 6</i>	-0.588	1.174	0.617
<i>Wave 7</i>	-0.879	1.380	0.524
<i>843 Fixed Effects Not Shown</i>
<i>Constant</i>	14.547	10.567	0.169
<i>R²</i>	0.88		
<i>N</i>	2396		

Since fixed effects generate bias towards zero coefficients, an especially conservative test of the Framingham results would be to subject them to this model. If we get a significant result in spite of the downward bias, then it would indicate our results are particularly robust. Table 2 shows that when we include fixed effects in our FHS-Net analyses, we find a significant influence of friend’s BMI on ego’s BMI, and the size of the effect is about the same at 0.05. Thus, our results in Framingham are robust to the CCF specification. Their assertion that a fixed effects model would change the Framingham result is thus not correct.

Homophily in Networks

CCF imply that our model does not account for selection effects or homophily, which is the tendency of people with similar attributes to form ties. If obese people befriend other obese people, it might create a correlation in weight status that is not driven by induction. CCF also assert that their method works in a way ours does not

when they say it “accounts for self-selection of friends (homophily) according to weight status by looking only at the change in BMI from the time of declaration of friendship until the subsequent weight measurement.” In fact, we did exactly the same thing as CCF in our Framingham analysis, including only individuals who were friends both at time t and $t+1$. In addition, we used generalized estimating procedures to account for individuals’ repeated appearances in the data. And, unlike CCF, we tracked the friendship status of people across time.

CCF point out the problem of homophily, noting “friendship selection could directly lead to the correlation between friends’ weight or weight gain without an individual’s weight causally affecting his friend’s weight through a social network effect.”(p.2) They then go on to make a specific claim about our method of controlling for homophily: “the CF dynamic model as estimated produced coefficients with large degrees of bias.”(p.2) This second claim, in particular, is not supported since it requires knowing the true value of the coefficient. To assess CF’s claim, we performed Monte Carlo simulations of the model we use to see if homophily would bias the estimate of the induction effect. We show here that it does not.

In our simulations, we generate a population of individuals, each with a BMI drawn from the empirical distribution observed in Framingham. We then permit individuals to form ties with a probability that is a sum of two variables, (1) a variable that is inversely proportional to the absolute difference in their BMI, and (2) a random uniform variable with support on $[0,1]$. We vary the weight on these two variables so that ties are either formed purely due to homophily, purely due to the random variable, or some combination of the two. This weight can be described as the percentage contribution homophily

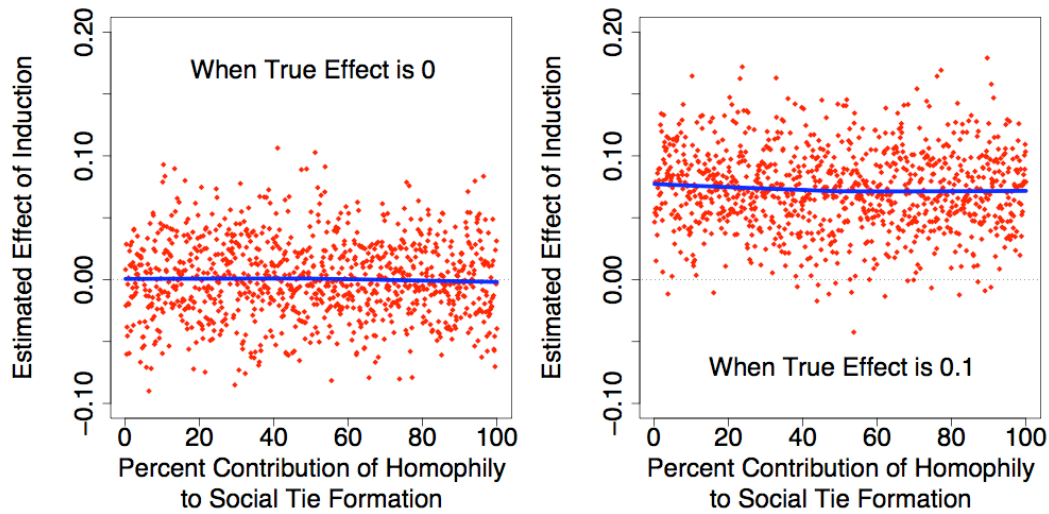
makes towards the formation of social ties.

We then give each individual an exogenous shock to their BMI that is drawn from the empirical distribution of *changes* in BMI observed in Framingham. Finally, we assume that the ego's new BMI is equal to a weighted combination of their own previous BMI with the shock and the average of their friends' BMIs (with their shocks). The weight on friends' BMIs is the induction effect. We can then use our regression model on the simulated data to see whether the degree of homophily affects the estimated induction effect. We do this by repeating these steps thousands of times and then comparing the "true" parameters used to simulate the data to the estimated parameters inferred from the regression model.

Figure 1 shows two sample tests from this analysis. In the first test, we hold the induction effect constant at 0, and we gradually change the way people form friends, going from random formation of friendships to those that are purely driven by homophily. The results of this test are shown on the left. In the second test, we hold the induction effect constant at 0.1 and once again we gradually change the way people form friends, going from random formation of friendships to those that are purely driven by homophily. These results are shown on the right.

The first thing to notice in the left panel is that when the true induction effect is 0, the regression model produces unbiased estimates of this effect that average to 0. But more importantly, it does this even in the presence of network formation that relies 100% on homophily to generate social ties! Thus, even if people in our sample form friendships *solely* on the basis of similarity in weight, we would not tend to find a peer-to-peer influence effect if none truly existed.

Figure 1. Monte Carlo Simulations of the Regression Model Used in Framingham Show Homophily Does Not Affect Estimate of Induction Effect



Note: Each point represents the estimated effect of alter BMI on ego BMI in one simulation of a network with the homophily weight indicated on the x-axis. The dark blue line shows best fitting LOESS curve to the observed points. “True effect” refers to induction parameter used in model to simulate the influence of alter on ego. 1000 simulations shown.

The right panel shows what happens when we repeat this test with a “true” induction effect of 0.1. Here we find that peers exert a small effect on average, and once again the result is not biased upward by the presence of homophily. Even if people in our sample tend to form friendships solely of the basis of similarity in weight, the model we use will not tend to overestimate the size of a peer-to-peer influence effect. In fact, the main drawback to our model is that the estimated effect tends to be *lower* than the true effect on average, but, in this setting, this just means that the model is conservative for effects of this size. This simulation evidence suggests that the CCF assertion that homophily causes us to overestimate the size of the induction effect is false.

Network Science Methods

Our work on social networks also involves a variety of further methods, including both graphical methods and network science methods that examine the role of topology in the kinds of effects of interest here (see, for example, Christakis and Fowler 2008). These methods provide additional evidence regarding the likely existence of peer effects with respect to obesity, smoking, and numerous other health outcomes.

Inter-Personal Health Effects

Social scientists have increasingly been exploring inter-personal health effects in the last few years (Burke and Heiland, 2006; Hammond and Epstein, 2007; Cutler and Glaeser 2007; Clark and Loheac, 2007). And the extent of such effects may extend considerably beyond health behaviors such as obesity and smoking. For example, it is not hard to imagine that people emulate each other when it comes to symptoms as well. When a person's friends, co-workers, and family come to complain of depression, back pain (Raspe et al, 2008), itching, cough, or headaches, they might do so as well.

Such peer effects are of obvious policy significance. First, it means that clinical and policy interventions may be more cost-effective than policy-makers may have previously supposed and that some interventions may gain more than others in the accounting (Christakis 2004, Rosen 1989, Powell, Tauras, and Ross 2005, Harris and López-Valcárcel 2008). Interventions that have greater positive externalities may rise in the analyst's estimation. If it costs \$25,000 to replace a man's hip and he gains four quality-adjusted life years from this intervention, and if his wife also gains one quality-adjusted life year as a result of having a more active partner, then the cost-effectiveness

of the surgery has just gone up by 25%. But if a knee replacement does not benefit a spouse, then *its* cost-effectiveness does not rise. If we spend \$500 to get a person to quit smoking and if this person's quitting in turn results in one out of ten of her social contacts quitting, and if that leads to one out of *that* person's social contacts quitting as well, we can see that three people have quit for the price of one, tripling the cost-effectiveness of the intervention.

These kinds of effects are rarely taken into account by policy makers or even by entities with a collective perspective, such as insurers. Yet they probably should be.

A second implication of our embeddedness in social networks is that group-level interventions may be more successful and more efficient than individual interventions. In fact, programs such as Alcoholics Anonymous, runners' clubs, symptom support groups (e.g., for chronic fatigue), and weight loss groups are explicitly designed to create a set of artificial social network ties.

Finally, a social network perspective suggests that it may be possible to exploit variation in people's social network position to target interventions where they might be most effective in generating benefits for the group. For example, if funds are limited, it may be best to target people who are most likely to influence others. This has long been a focus in sociology (e.g., Valente and Pumpuang, 2007) and is increasingly becoming a focus in both health economics (e.g., Rao, Mobius, and Rosenblat, 2007; Banerjee R, Cohen-Cole E, and Zanella G, 2007) and development economics.

People are interconnected, and so their health is interconnected.

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