Model of genetic variation in human social networks

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Social networks exhibit strikingly systematic patterns across a wide range of human contexts. Although genetic variation accounts for a significant portion of the variation in many complex social behaviors, the heritability of egocentric social network attributes is unknown. Here, we show that 3 of these attributes (in-degree, transitivity, and centrality) are heritable. We then develop a ''mirror network'' method to test extant network models and show that none account for observed genetic variation in human social networks. We propose an alternative ''Attract and Introduce'' model with two simple forms of heterogeneity that generates significant heritability and other important network features. We show that the model is well suited to real social networks in humans. These results suggest that natural selection may have played a role in the evolution of social networks. They also suggest that modeling intrinsic variation in network attributes may be important for understanding the way genes affect human behaviors and the way these behaviors spread from person to person.

evolution of cooperation | heritability | twins

Human social networks are characterized by rich variation at the individual level. Some people have few friends whereas others have many. Some people are embedded in tightly-knit groups where everyone knows each other, whereas others belong to many different groups where there is little overlap between friends. To explain this variation, scholars have sought simple models of network formation that generate an empirically realistic distribution of network characteristics as an endogenous outcome of a self-organizing process.

The best-known network formation models start with *identical* individuals that are subjected to social processes that create or exacerbate dissimilarity in a network. For example, in the ''scale-free'' physics model (1) it is the process of growth and, in particular, preferential attachment that drives the ''self-organizing'' feature of the power-law distribution in the degree. In the economic ''connections model'' (2–3), individuals who are homogenous *ex ante* endogenously form a star network when actors obtain indirect network benefits and when they are driven by (short-run) economic incentives. And in sociology, actors' preferences for ''structural balance'' (4) and ''homophily'' (5) tend to stimulate transitivity in social relationships and the formation of like-minded cliques.

Although the structural processes in these models generate empirically realistic variation in some network attributes, the effect of individual characteristics has been mainly ignored. There have been extensions to the canonical models that do take into account individual heterogeneity (6–12), but these models are usually presented as ''robust'' versions of the original models, in which the focus still is on the endogenous process (13). In this article, we focus instead on the individual characteristics themselves and explore the possibility that humans are endowed with traits that affect their network attributes. And our most intrinsic characteristics can be found in our genes.

To test the hypothesis that genes play a role in human social network structures, we use a classic twin study design (14–15). This design measures the heritability of a behavioral trait by comparing trait similarity in (same-sex) monozygotic (MZ) twins who share 100% of their segregating genes to trait similarity in same-sex dizygotic (DZ) twins who share only 50% on average. Under the assumptions of the twin study design, if genetic variation is contributing to variation in the trait, then MZ twins should be significantly more similar than DZ twins. Although some scholars object to the assumptions of the design (see SI), it has been widely used to show that genes play a role in personality (16), intelligence (17–18), and several other behavioral traits (14–15, 19–23). Turkheimer suggests as a ''first law of behavior genetics'' that all human behavioral traits are heritable (24).

We should therefore not be surprised to learn that individual social network characteristics have a partly genetic basis. However, as we will show, not all network characteristics are significantly heritable, and, more pertinently, specific estimates of heritability can provide a means to test theoretical models of human social networks.

Results

The fundamental building blocks of a human social network are egocentric properties of each individual in the network: the degree (the number of a person's contacts, or social ties) and transitivity (the likelihood that two of a person's contacts are connected to each other, also called the clustering coefficient). A wide variety of social networks can be constructed by altering the distribution of degree and transitivity between individuals (the nodes of the network), and these two attributes also have a strong influence on other network properties such as betweenness centrality (the fraction of paths through the network that pass through a given node). For example, a higher degree is positively correlated with greater centrality.

To measure how much variation in these node-level measures can be attributed to genetic variation, we used an additive genetic model (see SI) to analyze 1,110 twins from a sample of 90,115 adolescents in 142 separate school friendship networks in the National Longitudinal Study of Adolescent Health (the ''Add Health'' study; see SI for description). The results show that genetic factors account for 46% [95% confidence interval (C.I.) 23%, 69%] of the variation in in-degree (how many times a person is named as a friend), but heritability of out-degree (how many friends a person names) is not significant (22%, C.I. 0%, 47%). In addition, node transitivity is significantly heritable, with 47% (C.I. 13%, 65%) of the variation explained by differences in genes. We also find that genetic variation contributes to variation in other network characteristics; for example, betweenness centrality is significantly heritable (29%, C.I. 5%, 39%).

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These results allow us to reject the hypothesis that genes have no effect on human social networks. However, they also focus our attention on what kinds of attributes are heritable. For example, it is striking that in-degree is significantly heritable whereas out-degree appears not to be. There are many potentially interesting causal pathways from genes to human network structure that merit exploration. For example, it was recently shown that the −G1438A polymorphism within the promoter region of the 5-HT2A serotonin receptor gene is associated with variation in popularity (25). However, here we focus on the important implications of such variation—whatever its specific genetic determinant—for models of human social networks.

Network models that do not include intrinsic node characteristics cannot generate heritability in network attributes. The reason is that nodes without their own individual properties can be interchanged without affecting the structure of the network (6). Likewise, genes give people individuality; without genetic variation, human characteristics cannot, by definition, be heritable. Thus, to generate heritability in a model of human social networks, nodes must be endowed with characteristics that actually exhibit variation, and these characteristics must be associated with node network measures.

We surveyed the existing literature for network models that incorporate intrinsic node characteristics. "Hidden variables" models incorporate variation in an attribute regulating the formation of social ties (6). For example, a "fitness" parameter has been used to explore the conditions under which a late entrant might dominate networks constructed via preferential attachment (7, 26). This "fitness" model was proposed to take into account that some nodes are intrinsically more attractive. In an alternative model (8), nodes are placed in a "social space" (9) or a "latent space" (10) where greater social distance reduces the likelihood of a social tie. The social space model (8) in particular generates 3 outcomes that are characteristic of human social networks (as distinct from technological or biological networks): high transitivity, positive degree-degree correlation (popular people have popular friends), and community structure within the network (11). Finally, exponential random graph models (ERGMs) are statistical network characterizations that can incorporate node heterogeneity to explain degree heterogeneity and population-level average transitivity (12).

We developed a "mirror network" method to test whether the "fitness" (7), "social space" (8), ERGM (12), or regular Erdos–Renyi "random" network (27) models generate heritability in degree or transitivity (see SI). In this method, we create one set of nodes with intrinsic characteristics drawn from a probability distribution as defined by each of the models. We then follow the procedures outlined in the network model being tested for connecting nodes. Once that is complete, we create a second set of nodes with intrinsic characteristics drawn from the same probability distribution as before. We randomly choose one node from the first set and copy its characteristics to one randomly chosen node in the second set to create a pair of "twins." We then follow the procedures outlined in the network model being tested for connecting the second set of nodes to create a "mirror network." This is like creating N identical twin pairs and putting one twin from each pair in two separate environments. The initial randomization ensures that twins have uncorrelated environments before the onset of edge formation. Therefore, any resulting correlation in a twin pair's network measures (or the network measures of their friends) is an outcome of the edge-generation process, not the other way around.

Once the two networks have been independently constructed, we record relevant network measures for each of the two twins (in-degree, out-degree, transitivity, and betweenness centrality). We then repeat this procedure 10,000 times. The Pearson correlation between the twins gives an estimate of the proportion of the variation of the network measure that is explained by intrinsic node characteristics, analogous to the phenotypic variance explained by genes in models of identical twins reared apart (16). The "mirror network" method rejects all extant models of social network formation because they do not generate heritability that falls within the confidence intervals of the empirical estimates (Fig. 1). The ERGM comes closest, generating realistic heritability in in-degree and betweenness centrality, but it does not generate realistic heritability in transitivity.

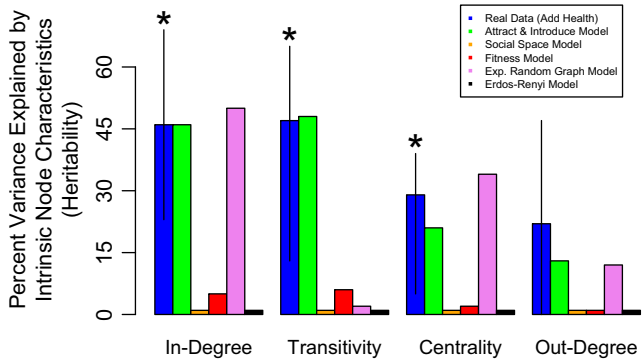

Fig. 1. Heritability of network characteristics in a real social network (Add Health) and simulated networks based on 5 models, the Attract and Introduce model; a social space model (8); a "fitness" model (7); an exponential random graph model (ERGM) (12); and an Erdos–Renyi random network (27). We used additive genetic models of monozygotic and dizygotic twins in Add Health to measure the heritability of network characteristics in real human social networks (see SI). The blue bars show that genetic variation accounts for significant variation in in-degree, transitivity, and betweenness centrality (vertical lines indicate 95% confidence intervals, asterisks indicate which confidence intervals exclude 0). These results suggest intrinsic characteristics have an important impact on the fundamental building blocks of real human social networks. Heritability of out-degree is not significant. To see which network models are capable of generating heritability consistent with the empirical observations, we simulated 10,000 pairs of networks and used the "mirror network" method for each proposed model to measure how much variance in network measures can be explained by intrinsic node characteristics. Compared with heritability estimates from the real social network data, all proposed models are rejected because they fall outside the confidence intervals except ERGM for in-degree and transivity, and Attract and Introduce for all three significantly heritable network properties (in-degree, transitivity, and centrality).

We therefore developed an alternative "Attract and Introduce" model (see SI) built on two assumptions. First, some individuals are inherently more attractive than others, whether physically or otherwise, so they receive more friendship nominations. Second, some individuals are inherently more inclined to introduce new friends to existing friends (and hence such individuals will indirectly enhance their own transitivity). In the Attract and Introduce model, individuals are chosen randomly to form ties and introduce their friends until a fixed number of ties for the whole network is reached (the alternative models either follow the same rule or they establish probabilities of tie formation that yield a fixed number of ties in expectation for a given network size). The model has just 2 parameters, one controlling the distribution of p_{attract} that is the probability of being named as a friend, and one controlling the distribution of $p_{\text{introduce}}$ that is the probability of introducing one's friends to each other.

The Attract and Introduce model generates heritability for in-degree, transitivity, and betweenness centrality that falls within the range of heritability observed in the real data (Fig. 1). The model also yields other important characteristics of human networks. Fig. 2 shows that the tail of the degree distribution falls between the straight line of a power-law distribution (as generated by the fitness model) and the fast cutoff of an exponential distribution (as generated by the social space and Erdos–Renyi models) (Fig. 2). The Attract and Introduce model also gener-

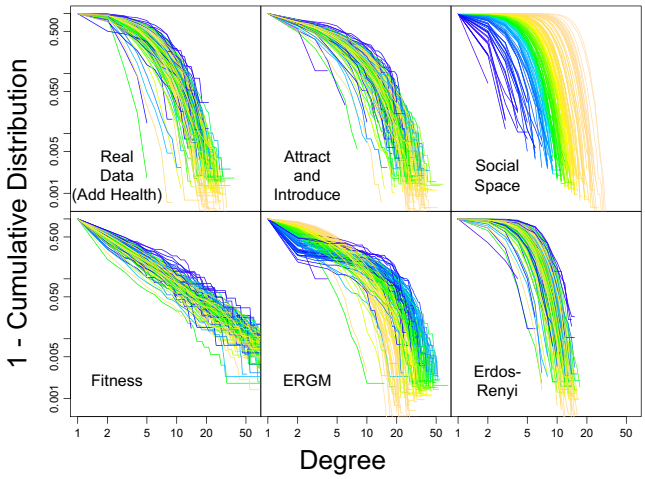

Fig. 2. Comparison of degree distributions in real social networks (within each of the 146 schools in the Add Health sample) and like-sized simulated networks based on 5 models, the Attract and Introduce model; a social space model (8); a fitness model (7); an ERGM (12); and an Erdos–Renyi random network (27). In *Upper Left*, each line indicates the in-degree distribution for each school in Add Health. In other images, line indicates the degree distribution in one simulation that assumes the same number of nodes and edges as each of the 146 schools. The color of each line indicates the size of the network (number of nodes) with yellow shades for small, green for medium, and blue for large networks (total range 9 to 2,724, mean 752). The fitness model generates a power-law tail in the degree distribution that is overdispersed (more nodes with higher degree, fewer nodes with lower degree) compared with the real data, whereas the Erdos–Renyi and social space models generate an exponential cutoff and a degree distribution that is underdispersed. Both ERGM and the Attract and Introduce model are slightly overdispersed, but ERGM in particular shows greater dispersion for larger networks (blue lines are lower for low degree and higher for high degree) in a pattern that does not exist in the real data (the social space model also exhibits an unrealistically strong relationship between network size and dispersion). The Attract and Introduce model produces variation in degree distributions across networks that is similar to Add Health.

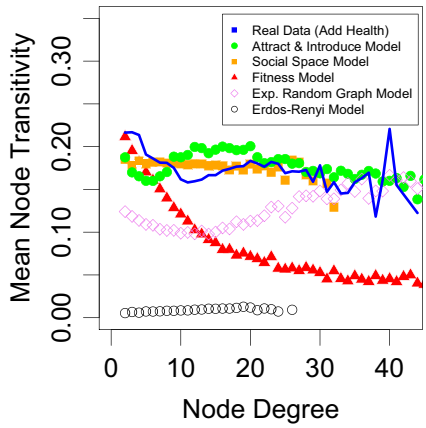

Fig. 3. Comparison of node degree and mean node transitivity in a real social network (Add Health) and simulated networks based on 5 models, the Attract and Introduce model; a social space model (8); a fitness model (7); an ERGM (12); and an Erdos–Renyi random network (27). We used the number of nodes and edges for each observed network in Add Health to generate 1,000 simulated networks for each proposed model and then calculated the mean node transitivity for all nodes of a given degree. The Attract and Introduce model deviates least from the observed data.

ates positive degree-degree correlation ($\rho = 0.18$), high transitivity ($c = 0.18$), a relationship between node degree and transitivity that closely follows the observed relationship in the Add Health data (Fig. 3), and realistic community structure with significant modularity (Fig. 4) (24). Finally, our model also generates motif structures that have a higher likelihood than all of the proposed alternatives (Fig. 5). These motif structures are patterns of ties in sets of 3 nodes or sets of 4 nodes, and their frequency creates a network "fingerprint" that can be used to identify a unique set of observed or simulated networks (see SI).

Discussion

To date, there has been relatively little attention to the role of individual heterogeneity in the formation of social networks. The evidence we present here suggests that egocentric properties are significantly heritable in human social networks. It is therefore important to make individual characteristics just as focal in the modeling of social networks as structural processes. Although it may not be surprising that genetic variation influences network formation, the effects are large enough that it is hard to argue that they can be ignored. Our Attract and Introduce model accounts for the role genes play not only in direct relationships (in-degree) but also in indirect relationships (transitivity and centrality), and as a consequence it is able to generate realistic large-scale community structure. We hope that this approach will generate interest in modeling individual heterogeneity and in using methods like the mirror network technique to test future models of network formation.

In the Attract and Introduce model, genes shape networks; but it also may be the case that networks shape genes. Scholars studying the evolution of cooperation in humans have recently turned their attention to the structure of social networks underlying human interactions (28). For example, in a fixed social network, cooperation can evolve as a consequence of "social viscosity" even in the absence of reputation effects or strategic complexity (29–30). Different network structures can speed or slow selection and in some cases they completely determine the outcome of a frequency-dependent selection process (31). Moreover, adaptive selection of network ties by individuals on evolving graphs can also influence the evolution of behavioral types (32–34). This research provides several theoretical examples of how natural selection can yield stable variation in local network structures. Future work should explore whether social networks may also result from (or contribute to) other sources of genetic variation in humans and other species, such as life history tradeoffs (35), balance between mutation and selection (36), sexually antagonistic selection (37), or the search for desirable partners also sought by others.

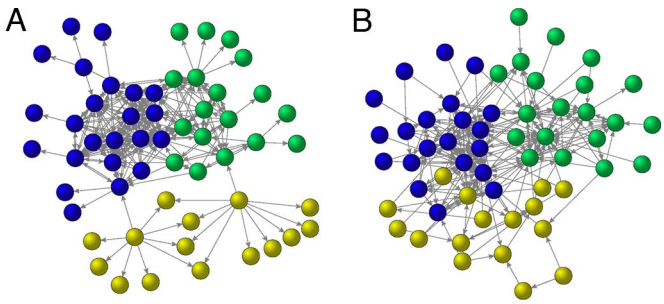

Fig. 4. Comparison of community structure in real and simulated networks. (*A*) School 115 (57 nodes and 252 ties) from the Add Health data. (*B*) Simulated network, using the Attract and Introduce model and the same number of nodes and ties. These networks show significant modularity with well-defined communities that have many connections within their group and few connections to other groups. Colors indicate communities that maximize modularity (11) (modularity = 0.35 in real network; modularity = 0.34 in simulated network).

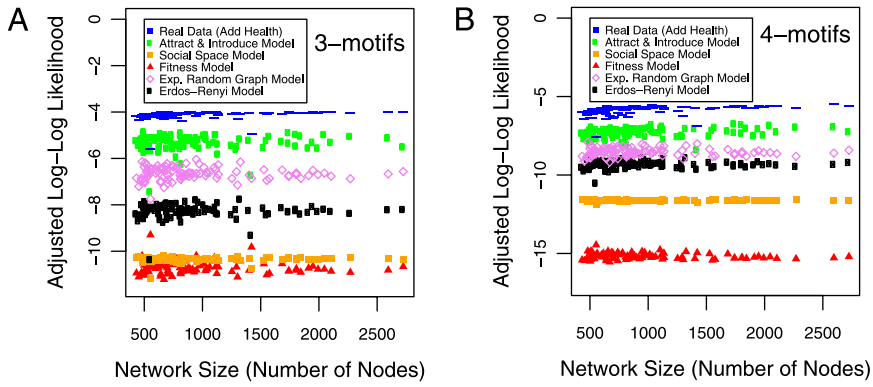

Fig. 5. The Attract and Introduce model best fits the motif structure of observed networks. (*A*) We simulate 100 networks from each proposed model, using the empirical distribution of nodes and edges in each Add Health network. We then count the total number of 3-motifs (isomorphic combinations of ties that connect 3 nodes) in each network and divide by the total number of motifs in that network to generate the empirical probability that 3 nodes form any given motif (the 3-motif fingerprint of the network) (47–50). For each motif, we fit 1 dimension of a multivariate beta density across all simulated networks to characterize the empirical probability of observing a given motif for a given model. We then use this estimated density to calculate the likelihood that the observed network could have been generated from the proposed model. For ease of exposition, we show adjusted likelihoods $-\log(c - LL)$, where c is a constant across all networks and models and LL is the log likelihood of generating an observed network. Each point in the figure represents the adjusted likelihood that a proposed model generates an observed Add Health network of a given size. Among all proposed models, Attract and Introduce (green circles) is the most likely to generate the 3-motif fingerprint in all of the Add Health networks, as shown. (*B*) We repeat the fingerprint procedure for 4-motifs (isomorphic combinations of ties that connect 4 nodes). Attract and Introduce is also the most likely to generate the 4-motif fingerprint in 98 of 100 Add Health networks. Exponential random graph models (violet diamonds) are most likely to generate the 4-motif fingerprint for 2 of 100 Add Health networks. For comparison, we also show adjusted likelihoods generated using a multivariate beta density fit to the actual data (blue dashes). In the SI we show that simulated networks are classified correctly in 10,000 of 10,000 tests, using the fingerprint procedure.

There may be many reasons for genetic variation in the ability to attract or the desire to introduce friends. More friends may mean greater social support in some settings or greater conflict in others. Having denser social connections may improve group solidarity, but it might also insulate a group from beneficial influence or information from individuals outside the group. Although it is possible that variation in individual social network attributes is incidental to natural selection processes operating on other traits, it is remarkable that network traits have significant heritability. Another area of future research should be the identification of mediating mechanisms like personality traits and the specific genes that may be involved.

Finally, social networks may serve the adaptive (or maladaptive) function of being a vehicle for the transmission of emotional states (38), material resources, or information (e.g., about resource or partner availability) between individuals. Some traits that appear to spread in social networks also appear to be heritable (such as obesity (20, 39), smoking behavior (40, 41), happiness (42, 43), and even political behavior (22, 23, 44–46)), suggesting that a full understanding of these traits may require a better understanding of the genetic basis of social network topology. The evidence here indicates that network theorists, behavior geneticists, evolutionary biologists, and social scientists ought to unify their theories regarding the structure and function of social networks, and their genetic antecedents.

Materials and Methods

For the Attract and Introduce model, we assume there are N nodes and E edges. Each node is permanently endowed with 2 characteristics, p^i_{attract} and $p^i_{\text{introduce}}$, with values randomly drawn from fixed distributions. The distribution of p^i_{attract} is based on a single parameter $\alpha \in [0,1]$ such that $\Pr(p^i_{\text{attract}} \sim \text{Uniform } [0,1])$ and $\Pr(p^i_{\text{attract}} = 0) = 1 - \alpha$. The distribution of $p^i_{\text{introduce}}$ is based on a single parameter $\beta \in [0,1]$ such that $\Pr(p^i_{\text{introduce}} = 1) = \beta$ and $\Pr(p^i_{\text{introduce}} = 0) = 1 - \beta$.

At each time period, nodes i and j are randomly chosen from the population, and with probability p^i_{attract} a social tie from i to j forms. If this occurs, then with probability $p^i_{\text{introduce}}$, i chooses to introduce j to all of his ''friends'' (the other nodes to which i is already connected). If i does introduce, then each friend sends a tie to j with probability p^j_{attract} and j sends a tie to each friend with probability p^j_{attract} that corresponds to each kth friend. This process is repeated until at least E ties are generated. In the SI we show code used to generate this model and test it using the ''mirror network'' method to assess heritability.

To establish the best fitting distributions for p^i_{attract} and $p^i_{\text{introduce}}$, we optimized one parameter for each to generate the empirically-observed average transitivity and heritability of in-degree and node transitivity in a network where $n = 750$ and $E = 3150$ (reflecting the typical school network in the Add Health data). The distribution of attractiveness that fit the data suggests about 1/10 of the individuals have very low attractiveness whereas the remaining 9/10 are approximately evenly distributed between low, medium, and high attractiveness. The probability a person will have the desire to introduce is about 3/10.

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1. Barabasi A-L, Albert R (1999) Emergence of scaling in random networks. *Science* 286:509–512.
2. Jackson MO, Wolinsky A (1996) A strategic model of economic and social networks. *J Econ Theory* 71:44–74.
3. Bala V, Goyal S (2000) A non-cooperative model of network formation. *Econometrica* 68:1181–1231.
4. Cartwright D, Harary F (1956) Structural balance: A generalization of Heider's theory. *Psychol Rev* 63:277–292.
5. McPherson M, Smith-Lovin L, Cook JM (2001) Birds of a feather: Homophily in social networks. *Annual Rev Sociol* 27:415–444.
6. Park JY, Barabási AL (2007) Distribution of node characteristics in complex networks. *Proc Natl Acad Sci USA* 104:17916–17920.
7. Bianconi G, Barabási AL (2001) Competition and multiscaling in evolving networks. *Europhys Lett* 54:436.
8. Boguñá M, Pastor-Satorras R, Díaz-Guilera A (2004) Arenas A models of social networks based on social distance attachment. *Phys Rev E* 70:056122.
9. Watts DJ, Dodds PS, Newman MEJ (2002) Identity and search in social networks. *Science* 296:1302.
10. Hoff PD, Raftery AE, Handcock MS (2002) Latent space approaches to social network analysis. *J Am Stat Assoc* 97:1090–1099.
11. Girvan M, Newman MEJ (2002) Community structure in social and biological networks. *Proc Natl Acad Sci USA* 99:7821.
12. Snijders TAB, Pattison PE, Robins GL, Handcock MS (2006) New specifications for exponential random graph models. *Sociol Methodol* 36:99–153.
13. Boccaletti S, Latora V, Moreno Y, Chavez M, Hwang DU (2006) Complex networks: Structure and dynamics. *Physics Rep* 424:175.
14. Evans DM, Gillespie NA, Martin NG (2002) Biometrical genetics. *Biol Psycho* 61:33.

15. Neale MC, Cardon LR (1992) *Methodology for Genetic Studies of Twins and Families.* (Kluwer, Dordrecht, The Netherlands).
16. Bouchard TJ, Lykken DT, McGue M, Segal NL, Tellegen A (1990) Sources of human psychological differences: The Minnesota Study of Twins Reared Apart. *Science* 250:223.
17. Plomin R (1999) Genetics and general cognitive ability. *Nature* 402:C25–C29.
18. Devlin B, Daniels M, Roeder K (1997) The heritability of IQ. *Nature* 388:468–471.
19. Defries JC, Fulker DW, Labuda MC (1987) Evidence for a genetic etiology in reading-disability of twins. *Nature* 329:537–539.
20. Herbert A, *et al.* (2006) A common genetic variant is associated with adult and childhood obesity. *Science* 312:279–283.
21. Fox PW, Hershberger SL, Bouchard TJ (1996) Genetic and environmental contributions to the acquisition of a motor skill. *Nature* 384:356–358.
22. Fowler JH, Baker LA, Dawes CT (2008) Genetic variation in political participation. *Am Political Sci Rev* 102:233–248.
23. Settle JE Dawes CT, Fowler JH (2009) The Heritability of Partisan Attachment. *Political Res Q*, in press.
24. Turkheimer E (2000) Three laws of behavior genetics and what they mean. *Curr Dir Psychol Sci* 9:160–164.
25. Burt SA (2008) Genes and popularity: Evidence of an evocative gene-environment correlation. *Psychol Sci* 19:112–113.
26. Kong JS N Sarshar, and V.P. Roychowdhury (2008) Experience versus talent shapes the structure of the Web. *Proc Natl Acad Sci USA* 105:13724–13729.
27. Erdős P; Rényi A (1959) On random graphs. I. *Publicationes Mathematicae* 6:290–297.
28. Szabo G, Fath G (2007) Evolutionary games on graphs. *Phys Rep* 446:97–216.
29. Ohtsuki H, *et al.* (2006) A simple rule for the evolution of cooperation on graphs and social networks. *Nature* 441:502–505.
30. Nowak MA (2006) Five rules for the evolution of cooperation. *Science* 314:1560–1563.
31. Lieberman E, Hauert C, Nowak MA (2005) Evolutionary dynamics on graphs. *Nature* 433:312–316.
32. Pacheco JM, Traulsen A, Nowak MA (2006) Coevolution of strategy and structure in complex networks with dynamical linking. *Phys Rev Lett* 97:258103.
33. Santos FC, Pacheco JM, Lenaerts T. Cooperation prevails when individuals adjust their social ties. *PLOS Comput Biol* 2: 1284–1291.
34. Skyrms B, Pemantle RA (2000) dynamic model of social network formation. *Proc Natl Acad Sci USA* 97:9340–9346.
35. Wolf M, van Doorn GS, Leimar O, *et al.* (2007) Life-history trade-offs favour the evolution of animal personalities. *Nature* 447:581–584.
36. Barton NH, Keightley PD (2002) Understanding quantitative genetic variation. *Nat Rev Genet* 3:11–21.
37. Foerster K, *et al.* (2007) Sexually antagonistic genetic variation for fitness in red deer. *Nature* 447:1107–1110.
38. Gervais M, Wilson DS (2005) The evolution and functions of laughter and humor: A synthetic approach. *Q Rev Biol* 80:395–430.
39. Christakis NA, Fowler JH (2007) The spread of obesity in a large social network over 32 years. *N Engl J Med* 357:370–379.
40. Carmelli D, *et al.* (1992) Genetic influence on smoking—A study of male twins. *N Engl J Med* 327:829–833.
41. Christakis NA, Fowler JH (2008) The collective dynamics of smoking in a large social network. *N Engl J Med* 358:2249–2258.
42. Lykken D, Tellegen A (1996) Happiness is a stochastic phenomenon. *Psychol Sci* 7:186–189.
43. Fowler JH, Christakis NA (2008) The Dynamic Spread of Happiness in a Large Social Network. *British Med J* 337:a2338.
44. Fowler JH, Schreiber D (2008) Biology, politics, and the emerging science of human nature. *Science* 322:912–914.
45. Nickerson DW (2008) Is voting contagious? Evidence from two field experiments. *Am Politica Sci Rev* 102:49–57.
46. Fowler JH, Dawes CT (2008) Two genes predict voter turnout. *J Politics* 70:579–594.
47. Middendorf M, Ziv E, Wiggins CH (2005) Inferring network mechanisms. *Proc Natl Acad Sci USA* 102:3192–3197.
48. Milo R, *et al.* (2002) Network motifs: Simple building blocks of complex networks. *Science* 298:824–827.
49. Holland P, Leinhardt S (1975) In *Sociological Methodology*, ed Heise D (Jossey-Bass, San Francisco), pp 1–45.
50. Wasserman S, Faust, K (1994) *Social Network Analysis* (Cambridge Univ Press, New York).