Two results emerge. First, the critical infectiousness r_{half}, at which the disease infects half the population, decreases rapidly for small p (Fig. 3a). Second, for a disease that is sufficiently infectious to infect the entire population regardless of its structure, the time $T(p)$ required for global infection resembles the $L(p)$ curve (Fig. 3b). Thus, infectious diseases are predicted to spread much more easily and quickly in a small world; the alarming and less obvious point is how few short cuts are needed to make the world small.

Our model differs in some significant ways from other network models of disease spreading[20–24]. All the models indicate that network structure influences the speed and extent of disease transmission, but our model illuminates the dynamics as an explicit function of structure (Fig. 3), rather than for a few particular topologies, such as random graphs, stars and chains[20–23]. In the work closest to ours, Kretschmar and Morris[24] have shown that increases in the number of concurrent partnerships can significantly accelerate the propagation of a sexually-transmitted disease that spreads along the edges of a graph. All their graphs are disconnected because they fix the average number of partners per person at $k = 1$. An increase in the number of concurrent partnerships causes faster spreading by increasing the number of vertices in the graph's largest connected component. In contrast, all our graphs are connected; hence the predicted changes in the spreading dynamics are due to more subtle structural features than changes in connectedness. Moreover, changes in the number of concurrent partners are obvious to an individual, whereas transitions leading to a smaller world are not.

We have also examined the effect of small-world connectivity on three other dynamical systems. In each case, the elements were coupled according to the family of graphs described in Fig. 1. (1) For cellular automata charged with the computational task of density classification[25], we find that a simple 'majority-rule' running on a small-world graph can outperform all known human and genetic algorithm-generated rules running on a ring lattice. (2) For the iterated, multi-player 'Prisoner's dilemma'[11] played on a graph, we find that as the fraction of short cuts increases, cooperation is less likely to emerge in a population of players using a generalized 'tit-for-tat'[26] strategy. The likelihood of cooperative strategies evolving out of an initial cooperative/non-cooperative mix also decreases with increasing p. (3) Small-world networks of coupled phase oscillators synchronize almost as readily as in the mean-field model[2], despite having orders of magnitude fewer edges. This result may be relevant to the observed synchronization of widely separated neurons in the visual cortex[27] if, as seems plausible, the brain has a small-world architecture.

We hope that our work will stimulate further studies of small-world networks. Their distinctive combination of high clustering with short characteristic path length cannot be captured by traditional approximations such as those based on regular lattices or random graphs. Although small-world architecture has not received much attention, we suggest that it will probably turn out to be widespread in biological, social and man-made systems, often with important dynamical consequences. □

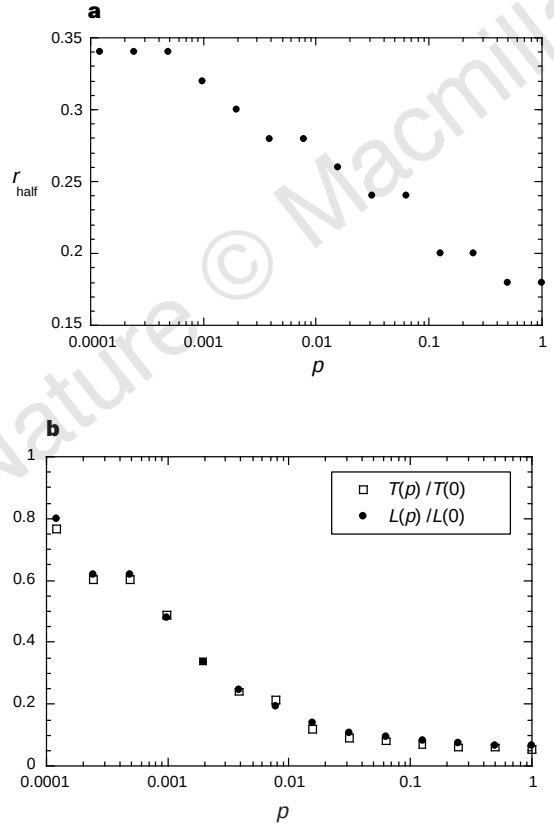

Figure 3 Simulation results for a simple model of disease spreading. The community structure is given by one realization of the family of randomly rewired graphs used in Fig. 1. **a**, Critical infectiousness r_{half}, at which the disease infects half the population, decreases with p. **b**, The time $T(p)$ required for a maximally infectious disease ($r = 1$) to spread throughout the entire population has essentially the same functional form as the characteristic path length $L(p)$. Even if only a few per cent of the edges in the original lattice are randomly rewired, the time to global infection is nearly as short as for a random graph.

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1. Winfree, A. T. *The Geometry of Biological Time* (Springer, New York, 1980).
2. Kuramoto, Y. *Chemical Oscillations, Waves, and Turbulence* (Springer, Berlin, 1984).
3. Strogatz, S. H. & Stewart, I. Coupled oscillators and biological synchronization. *Sci. Am.* **269**(6), 102–109 (1993).
4. Bressloff, P. C., Coombes, S. & De Souza, B. Dynamics of a ring of pulse-coupled oscillators: a group theoretic approach. *Phys. Rev. Lett.* **79,** 2791–2794 (1997).
5. Braiman, Y., Lindner, J. F. & Ditto, W. L. Taming spatiotemporal chaos with disorder. *Nature* **378,** 465–467 (1995).
6. Wiesenfeld, K. New results on frequency-locking dynamics of disordered Josephson arrays. *Physica B* **222,** 315–319 (1996).
7. Gerhardt, M., Schuster, H. & Tyson, J. J. A cellular automaton model of excitable media including curvature and dispersion. *Science* **247,** 1563–1566 (1990).
8. Collins, J. J., Chow, C. C. & Imhoff, T. T. Stochastic resonance without tuning. *Nature* **376,** 236–238 (1995).
9. Hopfield, J. J. & Herz, A. V. M. Rapid local synchronization of action potentials: Toward computation with coupled integrate-and-fire neurons. *Proc. Natl Acad. Sci. USA* **92,** 6655–6662 (1995).
10. Abbott, L. F. & van Vreeswijk, C. Asynchronous states in neural networks of pulse-coupled oscillators. *Phys. Rev. E* **48**(2), 1483–1490 (1993).
11. Nowak, M. A. & May, R. M. Evolutionary games and spatial chaos. *Nature* **359,** 826–829 (1992).
12. Kauffman, S. A. Metabolic stability and epigenesis in randomly constructed genetic nets. *J. Theor. Biol.* **22,** 437–467 (1969).
13. Milgram, S. The small world problem. *Psychol. Today* **2,** 60–67 (1967).
14. Kochen, M. (ed.) *The Small World* (Ablex, Norwood, NJ, 1989).
15. Guare, J. *Six Degrees of Separation: A Play* (Vintage Books, New York, 1990).
16. Bollabás, B. *Random Graphs* (Academic, London, 1985).
17. Achacoso, T. B. & Yamamoto, W. S. *AY's Neuroanatomy of C. elegans for Computation* (CRC Press, Boca Raton, FL, 1992).
18. Wasserman, S. & Faust, K. *Social Network Analysis: Methods and Applications* (Cambridge Univ. Press, 1994).
19. Phadke, A. G. & Thorp, J. S. *Computer Relaying for Power Systems* (Wiley, New York, 1988).
20. Sattenspiel, L. & Simon, C. P. The spread and persistence of infectious diseases in structured populations. *Math. Biosci.* **90,** 341–366 (1988).
21. Longini, I. M. Jr A mathematical model for predicting the geographic spread of new infectious agents. *Math. Biosci.* **90,** 367–383 (1988).
22. Hess, G. Disease in metapopulation models: implications for conservation. *Ecology* **77,** 1617–1632 (1996).
23. Blythe, S. P., Castillo-Chavez, C. & Palmer, J. S. Toward a unified theory of sexual mixing and pair formation. *Math. Biosci.* **107,** 379–405 (1991).
24. Kretschmar, M. & Morris, M. Measures of concurrency in networks and the spread of infectious disease. *Math. Biosci.* **133,** 165–195 (1996).
25. Das, R., Mitchell, M. & Crutchfield, J. P. in *Parallel Problem Solving from Nature* (eds Davido, Y., Schwefel, H.-P. & Männer, R.) 344–353 (Lecture Notes in Computer Science 866, Springer, Berlin, 1994).
26. Axelrod, R. *The Evolution of Cooperation* (Basic Books, New York, 1984).
27. Gray, C. M., König, P., Engel, A. K. & Singer, W. Oscillatory responses in cat visual cortex exhibit inter-columnar synchronization which reflects global stimulus properties. *Nature* **338,** 334–337 (1989).

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