eter (panel E, green regions) and flow vertically, driven by molecular diffusion of heat. Similarly, where warm, salty water lies underneath cool, fresh water (panel D, blue areas), diffusive convection causes enhanced upward heat flux (panel F).

The fluxes from these phenomena cause density changes in the intrusive layers (panel C) that can both create intrusions and drive them horizontally. In this way, layers as thick as the height of a building are moved hundreds of kilometers by structures that are as thin as an upraised arm.

However, the story is more complicated than this, underscoring the need for the detailed visualization techniques demonstrated by Holbrook *et al*. (*3*). The equatorial intrusions are almost certainly affected by salt fingers. Indeed, many characteristics of the intrusions, such as the density perturbations, suggest that fingers are driving the layers. Yet, a new theory by Richards and Edwards (*4*) shows that inertial instability could convert the kinetic energy of ocean currents into intrusive motions—without the need for salt fingers. And May and Kelley (*5*) have found that the potential energy due to sloping density layers can also drive intrusions.

As the oceanographer Rob Pinkel once said, having multiple explanations for a phenomenon is not an improvement over having a single explanation. Detailed data from the techniques reported by Holbrook *et al.* (*3*) should help to narrow down the mechanisms underlying ocean mixing.

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SOCIAL SCIENCE

Ignorance, Knowledge, and Outcomes in a Small World

Mark Granovetter

Thirty-six years ago, Stanley Milgram concluded from his "small world" experiment that any individual is connected to any other through a short chain of social ties, the average chain length being six people (*1*). On page 827 of this issue, Dodds, Muhamad, and Watts (*2*) provide the first large-scale replication of Milgram's experiment. Their analysis of 60,000 e-mail users—who were asked to reach 1 of 18 target persons in 13 countries by forwarding messages to acquaintances—suggests that Milgram's surprising results are robust. This excellent new study raises but cannot resolve the important question of how much people know about their own social networks and why this matters.

Milgram named the "small world problem" after the obligatory cocktail party response of strangers who unexpectedly discover that they share an acquaintance. Thus, the very name of the phenomenon alludes to surprise at and ignorance of one's social network. Indeed, Milgram liked to recount that before his results became well known, he would ask generally sophisticated audiences to guess how many personal acquaintance links were required to connect randomly chosen endpoints in the United States. Many guessed in the hundreds; hardly any imagined the half-dozen or so of his and later experiments.

Recent attention to "six degrees of separation" has made the naiveté that amused Milgram rare. But it does not follow that people now know more about their social networks. Just 500 acquaintances require more than 100,000 bits of information to track who knows whom, and if each acquaintance knows 500 others, there can be as many as a quarter of a million people at one remove. Overlap in ties reduces these numbers but introduces structural complexity that is equally if differently daunting. Limitations of cognition and time, not to mention competing obligations of everyday life, keep our network knowledge small and nonrandom. Even prolific and determined "networkers" cannot hope for more.

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Yet, enduring fascination with the "small world" phenomenon rests on the perception that we are sharply impacted by the length of network paths. Short paths may bring their endpoints new and better jobs (*3*) or political favors, but also may lead to infection with a dread disease or the unwanted exposure of illicit or shameful acts. The "small world" task of launching a message toward a target through an acquaintance is a clever way to estimate actual chain lengths. But it offers limited evidence of how much people actually know about their networks, and how they normally use them.

Dodds *et al*. suggest that in the "small world" task "all targets may in fact be reachable from random initial senders in only a few steps," but that small differences in incentives and confidence may produce large differences in completion rates. Given the likely high level of network ignorance, few can know the most efficient way to complete a chain. Thus, observed chain lengths are only an upper bound on actual minima. We can expect respondents to fashion strategies from the very limited information offered to them about targets. If occupation and location are the most salient part of this information, it is not surprising that, as Table 2 of the Dodds *et al*. paper shows, respondents channel the message through contacts who match the target on these characteristics. This produces the *appearance* of a network

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where reachability depends on people assigning nodes to "identity categories" [compare (*4*)], but is not good evidence that these cues are the most salient in everyday searches, where different, more complex and subtle information may be available.

We should also interpret with much caution the conclusion of Dodds *et al.* that hubs (nodes with many links to others) are less relevant to social searches than has been suggested by Barabasi (*5*) and others. This conclusion relies on identifying hubs by respondents' reports of whether they chose a particular acquaintance to send on the message because that person had many friends. But when giving a single response justifying one's choice of contact, location and occupation may seem more salient than number of ties. Moreover, peoples' information about how many contacts one's friends have is generally poor. Yet, it is likely that the choices respondents made were typically for individuals with a greater than average number of ties, because such individuals are considerably more likely than others to be chosen in network relations (*6*). Thus, the data may seriously understate the number of hubs, making it difficult to assess the part they play in social searches. More generally, close analysis of the characteristics of the *specific* nodes that ended failed chains, in comparison to specific nodes that reached intended targets, might tell us something beyond what can be learned by treating all nodes in all chains equally.

"Small-world studies offer tantalizing leads about connectivity and processes in natural networks..."

The broadest issue this research raises is what chain-length estimation experiments tell us about natural social processes. They suggest the need to extend our study to a wide range of situations where network chain length actually matters. This requires far more information than we now have on what people know about their networks, how they use this knowledge during searches, and how chain length impacts individuals *even when the search is absent*, as is common. Studies of how people find jobs through social networks show that while chain length is important, information reaches prospects who did not seek it in about one out of three cases—more for better jobs (*3*). Though people contract diseases when network distance to the already-infected is short, few searched their networks to achieve this outcome. Chain length and knowledge are critical, but the search complicated, when chain endpoints have opposing goals, as when searches are actively resisted by criminals, mob informers, missing deposed dictators or terrorist icons. Small-world studies offer tantalizing leads about connectivity and processes in natural networks, but then need confirmation and enrichment by studies that emerge from the laboratory to track actual networks. As in other branches of science, progress in understanding requires that tightly controlled experiment and real-world complexity regularly and systematically inform one another.

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DEVELOPMENT

Longing for Ligand: Hedgehog, Patched, and Cell Death

Isabel Guerrero and Ariel Ruiz i Altaba

Dependence on developmental signals, like dependence on love, can have catastrophic effects. Being unloved or lost in a multicellular organism can lead to self-inflicted death that people call suicide and cells call apoptosis. But how do lost cells recognize that they lack direction from developmental signals? That job may belong to ligand dependence receptors that induce cells bearing them to undergo apoptosis if the receptor remains unoccupied by ligand. These receptors are thought to ensure the survival of cells that remain close to the source of the appropriate developmental signal (the ligand), and the death of those cells that do not. On page 843 of this issue, Thibert *et al.* (*1*) provide evidence suggesting that the Patched1 (Ptc1) receptor is a dependence receptor that induces programmed cell death during chick neural tube development in the absence of its ligand, the signaling molecule Sonic hedgehog (Shh).

During neural development in vertebrates, Shh is produced first by the notochord and later by the floor plate (see the figure). A ventrodorsal gradient of Shh directs ventral patterning and cell differentiation [reviewed in (*2*)]. The Thibert *et al.* results provide insight into how the neural tube is shaped during development. In multicellular organisms, cells that are poorly positioned as a result of developmental errors can be eliminated because of failure to receive cues instructing them that they are in the correct location [reviewed in (*3*)]. Given the widespread importance of hedgehog (Hh) signaling during development, the results of Thibert *et al*. suggest how Ptc1 and Shh signaling may control tissue sculpting through selective cell death and survival.

To initiate the cell death program, proapoptotic dependence receptors require preliminary cleavage of their intracellular domain (at the DXXD site) by caspase enzymes. Thibert *et al.* present several lines of evidence indicating that Ptc1 is a proapoptotic dependence receptor. They show that overexpression of Ptc1 in cultured cells induces apoptosis, which is blocked by addition of Shh. In the developing chick neural tube, removal of the ventral source of Shh causes massive cell death, which is rescued by expression of a dominant-negative form of Ptc1 that interferes with the proposed function of wild-type Ptc1 in apoptosis. Cleavage of Ptc1 by caspase-3 exposes a carboxyl-terminal apoptotic domain. Transfecting cultured cells with the carboxyl-terminal region of Ptc1 is sufficient to induce cell death. In this region, there is a conserved aspartic acid residue in human, mouse, and chicken; mutation of this site (D1392N) in mouse Ptc1 prevents apoptosis when this receptor is unoccupied by ligand. Transfection of cultured cells with Ptc1 truncated at the caspase-cleavage site induces apoptosis that cannot be rescued by addition of Shh.

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