BROWSINGS

Agriculture: The Potential Consequences of Climate Variability and Change. *John M. Reilly, et al.* Cambridge University Press, Cambridge, 2002. 148 pp. Paper, $30, £21.95. ISBN 0-521-01628-2.

The next 100 years are expected to bring a 1° to 5°C rise in global temperature, shifting patterns of precipitation, and increased climate variability. The authors of this report use scenarios from state-of-the-art climate models to quantitatively assess the effects such changes are likely to have on U.S. agriculture. They predict that production will improve in most regions, although it may decline in southern parts of the country. They also identify less desirable impacts such as increased pesticide use, greater runoff of nutrients, and disputes over groundwater resources.

The Encyclopedia of Historic and Endangered Livestock and Poultry Breeds. *Janet Vorwald Dohner.* Yale University Press, New Haven, CT, 2001. 528 pp. $75, £55. ISBN 0-300-08880-9. Yale Agrarian Studies.

The Narragansett turkey, important in the markets of southern New England throughout the 19th century, is now extremely rare (less than 100 remain). Changes in agricultural practices have brought similar declines to the Dominique chicken, the Cotswold sheep, and many once-important farm animals; others have become extinct. Dohner presents concise accounts of the histories, traits, and qualities of nearly 200 North American and British breeds. Her engaging book testifies to the importance of saving the biodiversity of domestic animals.

Now he hopes to reach a broader audience. His laudable goal is simply "to inform" without passing opinion, in an attempt to enable those supporting or opposing GM to base their arguments on facts. Lurquin believes "the public has a right to know and understand how its food is manipulated at its most basic level, that of the DNA itself." He goes further, saying that "the absence of scientific information [is] the main problem blurring the perception of plant biotechnology." In a brave effort to solve that problem, the author presents an interesting and remarkably even-handed history of the development of GM technology.

If only those who should read the book would. Alas, I fear this will not be the case. Lurquin takes an historical approach that, although accurate and useful for students, is probably not the most efficient means of penetrating the minds of either the general public or the busy policy-makers—for whom the more in-depth approach of this book would nevertheless be useful. Worse, the appearance is dull, with a few black-and-white photographs of boffins and petri dishes. These are hardly the gripping images necessary to capture the public's imagination. There is no reason why attention to presentation would have compromised the quality of content.

Because the layout is not inviting, general readers are, unfortunately, unlikely to open the book. Political advisers will probably be too busy to work their way through the historical approach. And many activists on both sides of the controversy will not want to read Lurquin's account, for his reasoned and critical approach challenges most belief structures. Nonetheless, there is one important constituency for whom I would strongly recommend the book: biology undergraduates. Students should be informed about the debate on GM plants, and they should be able to present logical, reasoned arguments, for and against, to their nonbiologist peers and anyone else who will listen.

Going beyond the brief he set for *High Tech Harvest*, Lurquin includes one chapter in which he analyzes the controversy over GM plants. This he does well, with both scientifically accurate and politically acute commentary. He should expand this element into another book, using a publisher willing to invest in quality reproduction and the provision of clear and eye-catching graphics. Such a book might find the audience it deserves.

PERSPECTIVES: EPIDEMIOLOGY

Controlling Smallpox

Jim Koopman

To face the threat of a smallpox outbreak presented by bioterrorism, we need to answer the question: Should we parry the thrusts of intentional spread with targeted vaccination or raise the shield of mass vaccination? During targeted vaccination, health care workers locate and vaccinate people who have been exposed to the disease. In a mass vaccination campaign, anyone who goes to a vaccination center gets vaccinated regardless of their exposure status. The question of which approach is more effective at eradicating a smallpox outbreak is a crucial one. The demands of mass vaccination in the face of ongoing smallpox transmission are considerable and could prevent targeted vaccination of those who need it most. This raises the question of who should be vaccinated before a smallpox outbreak: the individuals most likely to spread infection or anyone who wants to be vaccinated? On page 1428 of this issue, Halloran *et al.* (*1*) present a model that simulates smallpox transmission in a structured community of 2000 people. They use their model to compare the efficiency of mass vaccination versus targeted vaccination for eradicating a smallpox outbreak. These investigators show that in all scenarios, targeted vaccination would prevent more smallpox cases per dose of vaccine than would mass vaccination. Given that we now have sufficient smallpox vaccine available to vaccinate everyone, the more relevant question is which vaccination strategy would be most efficient at preventing a smallpox epidemic. Halloran and colleagues show that the answer depends on the level of immunity in the population before the outbreak. They demonstrate that both pre-outbreak vaccination and residual immunity from previous vaccinations would increase the effectiveness of targeted vaccination after an outbreak of smallpox.

The finding that pre-event vaccination boosts the efficacy of postevent targeted vaccination raises several issues. If we do not vaccinate enough individuals before an event, then during an outbreak even the best efforts of our public health personnel might be insufficient to prevent an epidemic that extends way beyond the first intentionally infected cases. However, no level of pre-event mass vaccination will eliminate the need for intensive epidemic control efforts after an outbreak. Even given pre-event mass vaccination, some people will remain unvaccinated because they

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have contraindications or because they are not willing to travel to a public health clinic.

In the absence of ongoing cases of smallpox, new insights into transmission will depend on accurate modeling of a smallpox outbreak. But which model should we use? The Halloran *et al.* results (*1*) are not in accord with the recent findings of Kaplan *et al.* (*2*), who found that postevent mass vaccination would be the quickest and most effective way to halt a smallpox epidemic. Kaplan and colleagues used a continuous population model, whereas Halloran *et al.*'s findings were based on a discrete individual model. A continuous population model does not consider individuals, and so views infection as permeating the entire population. This type of model does not take into account chance events that could stop chains of transmission, whereas discrete individual models do incorporate such chance events.

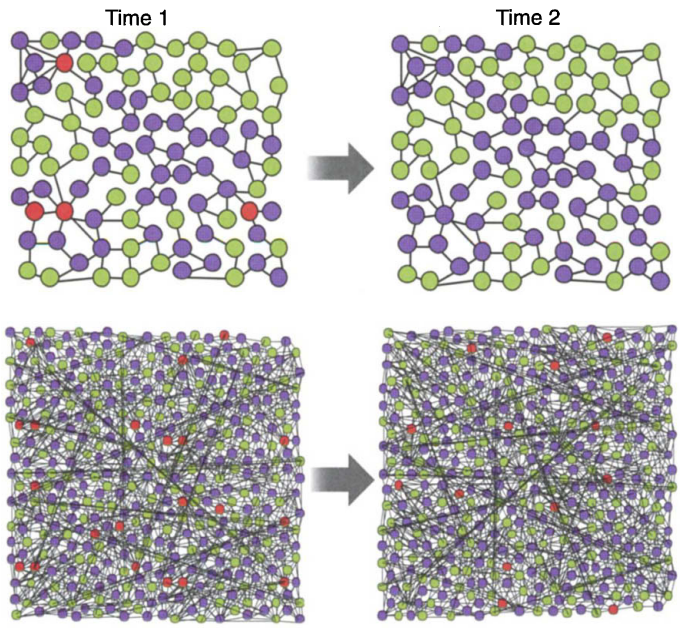
The controversy over discrete individual models versus continuous population models is an old one. More than three decades ago, Fox, Ackerman, and their colleagues used discrete individual models to illustrate how vaccine programs must focus on breaking up potential transmission chains rather than on reaching the magical overall level of vaccination suggested by continuous population models (*3*). Extensive modeling work by these investigators, however, languished because mathematicians did not recognize the unique modeling power of algorithms acting on discrete entities. Consequently the models of Ackerman, Fox, and collaborators were rarely cited and fell into obscurity. But now a revival is taking place. The power of algorithms acting on discrete entities has been recently demonstrated by Wolfram's mathematical modeling of patterns in nature (*4*). Halloran, Longini (Ackerman's student 30 years ago), and co-workers constructed their smallpox transmission model using algorithms similar to those developed by Ackerman and colleagues. Their model accounts for chance events—for example, who comes into contact with an infected person and at what point during the infection, and whether the infectious agent finds a new host before being eliminated—that affect the spread of infection (*1*). This type of stochastic (chance) model that links individuals locally is needed to inform appropriate decisions about the local control of a smallpox outbreak.

Other recent work shows that continuous population models are incapable of capturing the chance effects of different social and geographic environments (*5*, *6*). Such stochastic events underlie the effectiveness of targeted control measures (see the figure). Most chains of smallpox transmission die out locally, and targeted control efforts would ensure that this happens. Continuous population models, however, assume that as long as infectious and susceptible individuals coexist, they will come into contact, thus rendering local targeted control measures far less effective.

That is not to say that continuous population analyses are not needed. They have the advantage of being easier to analyze than discrete individual models. A particular advantage of the Kaplan *et al.* continuous population model is that it includes consideration of public health personnel and the time they take to vaccinate individuals. This is an important point because it takes a very short time to vaccinate individuals who voluntarily arrive at a vaccination site. It takes much longer to locate and vaccinate targeted individuals who have been exposed to those infected with the disease. Thus, the comparative benefits of vaccinating targeted individuals must be large to justify a targeted control strategy. Some epidemiologists who have worked on smallpox eradication feel that the benefits are in fact large enough to justify targeted control. The principal benefit comes from stopping chains of disease transmission and is not captured by a continuous population model.

Another distinction between these two types of model relates to how they account for contacts that transmit infection. One possibility is to model a mixing process that brings individuals together for only an instant when infection can take place. This scenario assumes that the population is thoroughly mixed the instant after each contact occurs. Both the Kaplan *et al.* and Halloran *et al.* models incorporate such a mixing process, the difference being that the mixing is more structured in the Halloran *et al.* model. Another possibility is a network model that describes a pattern of fixed links between individuals (*7*). Because the network model forges enduring links between individuals, it is easier to visualize what happens over time. Network models incorporate features of both discrete individual and continuous population models (see the figure). The ideal model both elucidates large-scale issues and captures the finer details of targeting decisions. Adding finer details to the Kaplan *et al.* model will not enable the capture of the microlevel effects seen by Halloran and colleagues. Combining various models will enable us to explore issues that are difficult to address with only one model (*8*).

Regardless of how effectively we link different models, the answers that models can provide are constrained by the available data. Without appropriate data, models cannot indicate whether we should target contacts for quarantine or vaccination when those contacts have been made in households, schools, workplaces, at public events, or under other circumstances. Which kind of exposure is associated with the most risk? Data on the risk of contacts in modern settings has never been gathered for small-



Transmission networks. **(Top)** A network interpretation of the discrete individual model of smallpox infection devised by Halloran *et al.* (*1*). Each individual is considered to be susceptible (green), infectious (red), or immune (blue). The figure shows the progression of infection with time during which a randomly infected individual could infect on average three others if everyone else is susceptible. In this scenario, a smallpox infection would readily die out. **(Bottom)** A network interpretation that approximates a continuous population model of smallpox infection, such as that developed by Kaplan *et al.* (*2*). The number of individuals modeled is close to infinite: Each individual is connected to every other regardless of the distance between them, and links are activated to transmit infection at a constant rate. A continuous population model corresponds to an infinite number of individuals, and so a smallpox infection would not die out.

pox. The relevant risk is not only that individuals get infected in those settings, but that these individuals themselves promote subsequent transmission of the disease.

One solution to the lack of data about smallpox outbreaks is to gather data on influenza epidemics. Indeed, Halloran *et al.* use insights from influenza outbreaks to guide construction of their smallpox transmission model. But even for influenza outbreaks, data are limited. Huge studies that follow individuals closely enough to document their exposure and infection are very expensive. However, there is another promising but untried data source: nucleotide sequences of infectious agents taken from individuals with documented contact points in the transmission system. The idea is not to determine which individuals infected other individuals, but rather to fit transmission models to genetic distances (estimated using nucleotide sequence data) in a pathogen population.

The Halloran *et al.* model simulates a smallpox outbreak in a population of 2000 individuals. They find that under a number of conditions, more infections are prevented by extending vaccination beyond immediate social contacts. This supports the notion that targeted control measures will need to involve vaccination of a broader range of individuals. Future work will need to focus on scaling up the Halloran *et al.* model so that it effectively simulates a population as large as that of the United States. There are other kinds of information that will also be needed by policy-makers. For example, we need to determine ahead of time the most important data to collect during a bioterrorist attack. How will the various new vaccines proposed for development fit into simulated control efforts like those examined in the Halloran *et al.* and Kaplan *et al.* studies? Neither study will put to rest the current debate over the best policy for protecting the U.S. population against a bioterrorist attack. But publication of these studies may help to orient future research that will provide the information needed by policy-makers.

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PERSPECTIVES: GEOLOGY

Serpentinite Seduction

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Serpentinite, a valued green decorative building stone and the official California state rock, forms through hydrothermal alteration of peridotite, the rock of Earth's mantle. It is common in ophiolites, which are exposed fragments of oceanic crust and subjacent mantle.

It has been assumed that serpentinite is widespread in oceanic plates undergoing subduction (*1*–*3*). If true, this would have important implications for earthquake and volcano activity in subduction zones. During subduction, water is released from serpentinites by metamorphic dehydration reactions. It has been suggested that serpentinite provides a particularly fertile water source for magma generated in subduction-related arc volcanoes (*1*). Furthermore, water released by metamorphic dehydration could trigger subduction-zone earthquakes (*4*).

In concert with previous studies (*2*, *3*), Dobson *et al.* contend on page 1407 of this issue (*5*) that earthquake hypocenters in the lower segments of double seismic zones (see the figure) can be attributed to serpentinite dehydration. Is serpentinite indeed widespread in the subducted oceanic plates, or is it a green herring?

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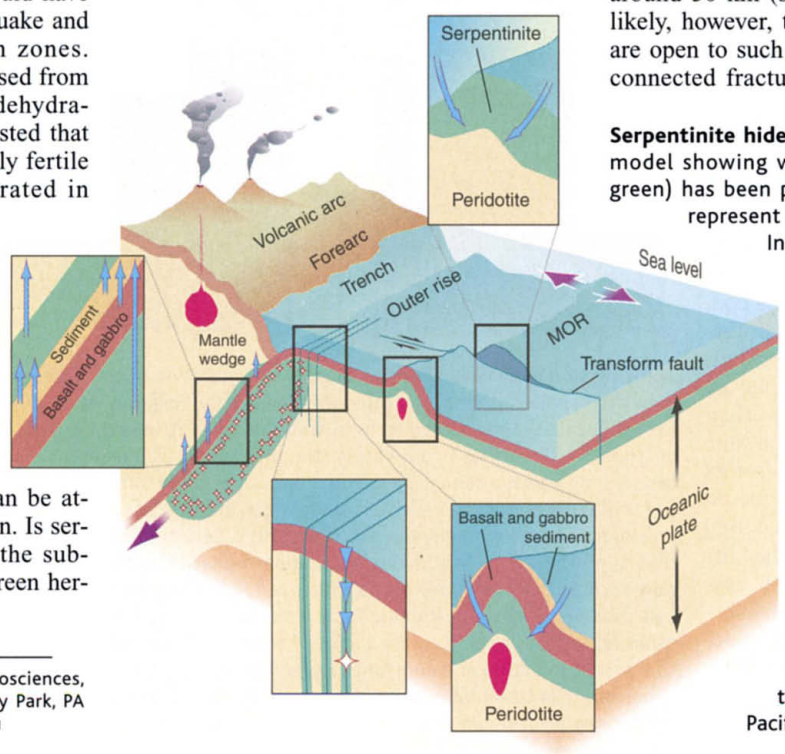
In the nonsubducting Atlantic oceanic plate, serpentinite is common where transform faults intersect and offset the slow-spreading Mid-Atlantic Ridge system. Hydrothermal circulation of seawater transforms the shallow peridotites into serpentinite. Seismic data suggest that the serpentinite may be 2 to 3 km thick (*6*).

In contrast, with rare exceptions (*7*), serpentinites are virtually nonexistent in the fast-spreading ridges of the Pacific oceanic plate and in ophiolites believed to be associated with fast-spreading centers (*8*). Nonetheless, serpentinization of oceanic mantle entering subduction zones around the Pacific (where most of Earth's subduction occurs) has been postulated to arise from infiltration of seawater into transform faults (*3*) or along deep faults at the "outer rise" where oceanic plates bend upon entering subduction zones (*2*).

The formation of serpentinite in the lower part of double seismic zones would require ingress of seawater to depths of around 50 km (see the figure). It is unlikely, however, that the outer rise faults are open to such depths, or that an interconnected fracture network allows deep



Serpentinite hide-and-seek. Plate tectonic model showing where serpentinite (dark green) has been postulated to form. Stars represent earthquake hypocenters. In the subducted plate, the hypocenters outline a double seismic zone. Large purple arrows indicate relative plate motion. Spreading occurs at the mid-ocean ridge (MOR). Blue arrows depict fluid ingress or expulsion. Magma is shown in red. Serpentinite along the transform fault is relevant to the slow-spreading Mid-Atlantic Ridge, whereas the other serpentinite locations are relevant to the Pacific plate.