are known to harbour pathogens that have previously emerged and focus efforts on the regions where most contact between wildlife and humans occurs.

A microbe in a primate population is more likely to become zoonotic than is a microbe from a rodent, because we are more likely to have similar cell surface receptors to the primate owing to our shared evolutionary history. But at what point does contact override phylogeny? If a hunter catches a primate once a year, but the staple diet in his village is bush rats, which of these is the high-risk species? These are the questions that disease ecologists can answer, and that are being applied to the new science of pandemic prediction. However, the prediction and prevention of a pandemic is not straightforward. Although molecular techniques exist that can identify novel microbes carried by these high-value wildlife targets, our predictive ability can be overwhelmed by the many novel microbial sequences discovered. For example, how can we identify, from the genetic sequences of ten new paramyxoviruses from bats, which one is most likely to be a virulent pathogen of human beings, capable of spillover and sustained human-to-human transmission? This is the biggest of the grand challenges for pandemic prevention, and one that I believe we are not strategically addressing. Morse and colleagues’ describe a strategy for the so-called known unknowns—novel microbes closely related to known agents. But what of the unknown unknowns—novel microbes that have no known close relative? This challenge, of prediction of viral virulence from a sequence, for example, should be a major focus of basic virology research in every developed country.

A global programme for pandemic prevention based on improved risk forecasting, surveillance, and pathogen discovery will be expensive. Who should pay and how would it work? The answer might lie in the underlying socioeconomic drivers of disease emergence. Pandemics are a product of our economic development—they emerge when we domesticate new species, open up new trade routes, build roads into forests, or expand air travel networks. Perhaps these industries should insure themselves against the rare but devastating pandemics their activities can sometimes cause. Additionally, health-impact assessments, already used in many large development projects, could calculate and assess the pandemic risk of a project. The ultimate public health programme would work with, and be funded by, high-risk development projects to develop better clinics, pathogen discovery, and surveillance programmes that prevent pandemics at their source.

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Emerging infectious diseases: the role of social sciences

Popular and scientific representations of research into emerging infectious disease often focus on the pathogen itself—its molecular machinery, processes of reassortment and mutation, and how these factors indicate risk for human-to-human transmission. However, social and ecological processes that facilitate infection also deserve close attention, as emphasised in the Lancet Series on zoonoses.1–3 Present models of pathogen emergence and spread do not identify underlying drivers with sufficient clarity to allow effective prevention of disease. More robust models that encompass the complex interface between pathogen biology and human,
vector, and reservoir behaviour are needed. These models should address causes of disease emergence, transmission, and spread that are linked to human behaviours, and the sociocultural and political systems that guide and constrain them. The social sciences are important for elaboration of causal assemblages and contextualisation of epidemiologically notable behaviours in social, economic, political, and cultural systems, and thereby contribute to effective behavioural and structural interventions.

A first step in appreciation of the importance of the social sciences in infectious disease research is to consider the effect of human social and behavioural factors on $R_0$, the basic reproductive rate of a pathogen—that is, the expected number of infections caused by one infected individual in a susceptible population. All three components of $R_0$ (contact or exposure rates, probability of transmission, and duration of infection) are partly regulated socially: contact rates are driven by social relationship patterns, which themselves are a product of values and norms in a community that guide social affiliation. Additionally, social factors such as stress and poverty can affect probability of transmission and duration of infection. Social inequality affects susceptibility to, and severity and duration of, infections through the mediating variable of psychosocial stress and risk for co-infection (eg, HIV). Apart from their direct and proximal effects on immunocompetence, poverty and social inequality have harmful effects on people’s health, since they affect access to care, adequate food, suitable housing, and healthy environments.

However, focus only on $R_0$ often restricts analysis to the mechanisms of transmission of established human pathogens. We need to move predictive capabilities closer to the point of species spillover to enable risk-based prevention strategies, in which social and behavioural factors have an important role. In the same way that ecologists talk about ecological cascades that facilitate disease emergence, social scientists speak of the scalar and multifaceted dimensions of influence in social systems—from an individual in a community to the wider political economy—that guide, constrain, or otherwise affect disease risk. The social sciences that emphasise primary data collection in diverse community settings—eg, anthropology, geography, and sociology—are well positioned to identify such influences. Causal complexes, in which social processes play a central part, include: agricultural development and related land use changes, which increase risk for vector-borne diseases; practices in industrial food production systems that facilitate the generation and spread of foodborne diseases, antibiotic-resistant pathogens, and the emergence of viral zoonoses; and social and economic activities, such as gathering of fruits, handling of bushmeat, and rearing of animals, which have brought people into close contact with natural or amplifying hosts, or their tissues.

Social scientists provide local and ethnographic data for behaviour related to vector and host species that are important to inform large-scale studies. A detailed inquiry into local social or ecological contexts is often essential for generation of hypotheses about so-called hot spots for disease emergence. In the causal chain of emergence, substantial gaps or uncertainties exist, which are often related to insufficient social and behavioural

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**Panel: Social factors relevant to prediction of a potential avian influenza outbreak**

Highly pathogenic avian influenza, or influenza A subtype H5N1 (A/H5N1), is mainly a disease of birds and domestic poultry, with outbreaks in people handling or living in close proximity to birds. Clusters of human disease have been identified in southeast Asia and Egypt, where agricultural practices interact with high population density to generate high spillover risk. The increasing size and diversity of the avian host reservoirs, ongoing virus mutations, and social and demographic processes that increase the probability of human transmission have made highly pathogenic avian influenza one of the most feared of the potential emerging zoonotic disease threats.

Similar to all zoonoses, the emergence of highly pathogenic avian influenza as a serious global pandemic depends on cross-species pathogen spillover to human beings and efficient human-to-human transmission (usually where $R_0>1$). Whether this emergence of a serious pandemic occurs will depend on whether the pathogen develops the requisite molecular machinery to allow transmission. Although viral mutation and reassortment are random processes to some extent, the probability of the emergence of a pandemic strain is greatly enhanced by social and demographic factors that increase the size, density, and intensity of interactions in the host species, including human beings. Important social factors that are yet to be fully researched include: details of household-level and worksite behaviours because these factors relate to poultry handling during the day, and by different sex, age, and socioeconomic groups; market conditions and the links between wild and domestic flocks; viral spread that might be facilitated via exports and sales of diseased birds; patterns of social interaction that connect some households more closely than others; and the content of social relationships in terms of the sharing of labour, food, and market responsibilities. These focused local-level studies can be joined with models of higher-level social, demographic, and ecological processes. Even if such work does not prove to be definitive in terms of identification of precise causal linkages, especially in view of the stochasticity inherent to pathogen emergence, it will certainly provide information essential for an effective public health response. Finally, there are clear social, political, and ethical dimensions to some of the present controversies that have arisen regarding highly pathogenic avian influenza, including the debate about whether to release research results that identify genetic mechanisms by which the virus could be transmitted directly through human-to-human contact.
data for epidemiologically significant aspects of the human-animal interface (panel). Another challenge is that many social scientists emphasise questions and methods that, to their counterparts in the biological sciences, might seem peripheral to epidemiological and biomedical discovery (eg, questioning social power, or critiquing assumptions, constructs, and variables), or do not seem to be rigorous (eg, qualitative methods).

Increased interaction between ethnographers and modellers in the development and selection of key aspects of system dynamics would be useful. However, many social scientists do not have skills in epidemiology or ecology. We suspect that barriers to effective collaboration represent communication obstacles that originated from researchers immersed in the traditions of particular disciplines. Yet interdisciplinarity offers hope for groundbreaking research that is needed for better prediction and control of disease emergence.

One promising approach addresses transmission dynamics from the perspective of social networks. The focus is on modelling how different behaviours and social structures affect disease spread, thereby resulting in epidemics, disease containment, stuttering transmission, or burnout. An important finding is that contact networks are characterised by community structure—ie, a tendency for formation of high-connectivity clusters, which can amplify or reduce population transmission.

Social networks can transmit information that increases community cohesion and ability to prevent infection, and also foster social mobilisation and culture change.

Thus, the social sciences are crucial in creation of appropriate public health responses to risk and emergence of epidemics. Examples include socially and culturally appropriate communication campaigns that manifest knowledge about risk perception, the importance of specific message frames to convey risk, and considerations of attenuation versus amplification of outrage in various scenarios with policy makers and the public. Social scientists have also collaborated in interventions to ensure appropriate access to and use of technologies for better prevention and biomedical management of infectious diseases (eg, vaccines, medications, bednets, and condoms). Finally, behaviour change theories have been applied effectively to change the behaviour of consumers and health-care providers.

Continuation and expansion of the engagement of the social sciences, ecology, and epidemiology in infectious disease research is the best route towards full understanding of the complex adaptive systems of emerging infectious diseases. To achieve prediction and control, we should better recognise and account for the fact that parasites and pathogens travel within and between human bodies that are linked in complex but patterned relationships.

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