# The social and political lives of zoonotic disease models: Narratives, science and policy

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## ABSTRACT

Zoonotic diseases currently pose both major health threats and complex scientific and policy challenges, to which modelling is increasingly called to respond. In this article we argue that the challenges are best met by combining multiple models and modelling approaches that elucidate the various epidemiological, ecological and social processes at work. These models should not be understood as neutral science informing policy in a linear manner, but as having social and political lives: social, cultural and political norms and values that shape their development and which they carry and project. We develop and illustrate this argument in relation to the cases of H5N1 avian influenza and Ebola, exploring for each the range of modelling approaches deployed and the ways they have been co-constructed with a particular politics of policy. Addressing the complex, uncertain dynamics of zoonotic disease requires such social and political lives to be made explicit in approaches that aim at triangulation rather than integration, and plural and conditional rather than singular forms of policy advice.

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## Introduction

Zoonotic diseases — transmitted from animals to people — present urgent scientific and policy challenges. Since 1940, 60% of emerging infectious diseases affecting humans have originated from animals, both domestic and wild (Jones et al., 2008). The impacts on poor people's health, lives and livelihoods are increasingly recognised, while if unchecked, many zoonoses threaten global pandemics — as HIV/AIDS and SARS demonstrated so vividly. Complex interactions of epidemiological, ecological, social and technological processes shape zoonotic disease emergence, transmission, risks and vulnerabilities, influenced by wider socio-economic and environmental drivers. Understanding and responding to these, as well as controlling outbreaks, have become crucial imperatives (King et al., 2006), attracting heavy government and international investment. There is growing support for inter-disciplinary and integrative approaches that address human, animal and ecosystem dimensions together — often labelled 'One World, One Health' (e.g. FAO-OIE-WHO, 2010).

Modelling carries growing authority in these efforts, valued to render complexity more legible and handleable, and to provide evidence and predictions for policy. We define 'model' broadly, to refer to a schematic description of a system or phenomenon that accounts for its key properties and may be used as the basis for further exploration or prediction. Modelling takes many forms, shedding light on complex patterns and processes from different perspectives. What then does modelling offer — currently and potentially — to the challenges of addressing zoonotic diseases, especially in dynamic, uncertain, resource-poor settings?

To address this question, we reject conceptions of modelling as an objective, neutral scientific exercise that linearly informs policy. We argue that multiple models that offer different perspectives on epidemiological, ecological and social processes can valuably be combined. Yet such models themselves need to be understood as having social and political lives. Extending Appadurai's original (1986) notion of the social life of things, this refers to the social, cultural and political norms and values that shape the development of particular models, and which they carry and project. Sociologists of science have explored how modelling involves social processes and practices that construct its inevitably selective readings of and gazes on the world (Magnani and Nercessian, 2009; Mansnerus, 2012; Mattila, 2006; Morgan, 2009; Morgan & Morrison, 1999). We connect these insights with understandings of the politics of policy processes (Keeley & Scoones, 2003) and of science and policy as mutually-constructed, or co-produced (Jasanoff, 2004; Shackley and Wynne, 1995). The social and political lives of zoonotic disease models therefore refer to the ways they are developed, shaped and applied in interaction with — or co-constructed with — the politics of policy. Such politics often involve an interplay of 'policy narratives' — simple storylines describing a policy problem, why it

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matters and to whom, and what should be done about it, that drive and justify interventions promoted by, or suiting the political interests, of certain groups (Roe, 1994). Extending our previous analyses of epidemic narratives (Dry & Leach, 2010; Scoones, 2010), here we interrogate their interplay with scientific − and in particular modelling − processes. We explore how modelling contributes to particular policy narratives about zoonotic disease, and how policy narratives uphold the authority of particular models and modelling approaches.

In addressing zoonoses, we adopt a heuristic that distinguishes three broad types of modelling: mathematical/process-based models of epidemiological and ecological relationships parameterised according to available data; pattern-based models which extract relationships from statistical analysis of empirical datasets, and what we term 'participatory' modelling based on anthropological, ethnographic and participatory approaches, including (but importantly going beyond) the established field of participatory epidemiology (Catley, Alders, & Wood, 2012). Such labelling is unconventional, but draws attention to the importance of diverse forms of knowledge and perspective in the schematic descriptions that models provide. This also brings such social science approaches into the same analytical field as more conventional modelling, enabling exploration of their politics.

In the following sections, we explore applications of each type of model in two cases − H5N1 in south-east Asia and Ebola in central Africa. As the cases illustrate, different models also serve scientific and policy purposes within the different 'stages' of understanding and action around zoonotic diseases: from risk mapping, to designing and implementing control measures, to evaluating interventions.

Both these cases involved localised disease outbreaks which some policy-makers and publics, at least, feared would 'go global'. Both illustrate the contested political interests at stake in policy choices. And in each case, these interests interplayed with the application of contrasting approaches to modelling. Drawing on an analysis of original scientific papers, discussions with key actors, and related literature and media reports, we consider for each model the socio-political and policy context in which scientists were working; the values and assumptions deployed; how uncertainties and data limitations were addressed, and the policy conclusions thus supported. In each case, although in very different ways, we show how modelling supported certain policy narratives over others, and how different modelling approaches interacted in a highly-politicised scientific and policy field. In conclusion, we suggest that these social and political lives of disease models cannot be wished away; rather, handling the complex, uncertain dynamics of zoonotic disease requires them to be made explicit in approaches that aim at triangulation rather than integration, and plural and conditional rather than singular forms of policy advice.

## Case 1: H5N1

H5N1, highly pathogenic avian influenza, dominated headlines for much of the decade following the first recorded human deaths in Hong Kong in 1997. Global public health priorities and much science focused on this zoonosis, given the prospect of a global pandemic on the scale experienced in 1918 (Scoones & Forster, 2010). Modelling efforts were central, dominated by one particular set of process-based models which we consider first.

### 'Evidence' for policy: epidemiological process- based models

In September 2005, two papers were published simultaneously in *Nature* and *Science*. Both contained process-based simulation models of the potential spread of H5N1 in humans in Thailand, and the implications of different control measures (Ferguson et al., 2005; Longini et al., 2005). Both argued that 'control at source', especially through a massive use of antiviral drugs combined with other containment measures, would help prevent a global outbreak. The much cited Ferguson et al. (2005) paper has been widely used as the core evidence base for policy thinking, from the WHO to national governments.

The models showed how 'drugs could head off a flu pandemic − but only if we respond fast enough' (Nature, 2005a: 614). As a *Nature* editorial argued:

> They reach markedly different conclusions about how easy it would be to contain an emerging pandemic. But both agree that it would be possible − if the virus was detected quickly, if it did not spread too fast, if sufficient antivirals were deployed quickly and massively around the outbreak's epicentre, and if strict quarantine and other measures were used (p. 614).

Ferguson et al.'s model suggested that containment would succeed if everyone was treated within a five-kilometre radius, involving two to three million drug courses, and if quarantine and movement control were instituted from the start. By contrast, Longini et al.'s model suggested that 100,000 − one million drug courses would be sufficient, administered to the ill and their social contacts.

These variants notwithstanding, the dramatic figures and pleas for urgent action in both models fed perfectly into the 'outbreak' narrative gripping policymakers. In the same month as publication, the UN avian influenza coordinator, David Nabarro, cranked up the scare factor dramatically, arguing that total human deaths could reach 150 million (BBC, 2005). The media had a field-day, and policymakers globally started planning for the worst. The push to boost the current WHO antiviral stockpile of 120,000 courses was high, and pharmaceutical companies happy to oblige. While human-to-human spread did not eventually occur to the feared extent, the power of the models in framing policy was clear. They drove the response to H5N1 − and subsequently H1N1 'swine flu' (Fraser et al., 2009) and indeed other zoonoses − creating the justification for 'at source' control through a massive anti-viral drug intervention.

Nevertheless, the Ferguson model made several questionable assumptions about epidemiological parameters and transmission dynamics, not least due to limited specific data from Thailand. Thus the generation time was assumed to be low (2.6 days) on the basis of data from 2000 in France, age specific attack rates were modelled from 1957 data from Sheffield, UK, and incubation times from a study of infection on an aeroplane. Households were assumed to be randomly distributed, and a ratio assumed between random, place-based and intra-household infections, ignoring any social dynamics in rural village settings. The model chose the country's third least populated rural area to seed the infection and drive the simulation. The resulting slow viral spread was central to the projected success of the model control strategy, requiring local containment within 30 days. Yet as we discuss below, other work suggests that outbreaks are especially common in peri-urban semi-intensive poultry production areas, where the disease may spread much faster. Spread was modelled from a 1994 migration and work survey, but this was restricted to formal workplaces, ignoring movement associated with informal activities. The model assumed no changes in behaviour as the pandemic accelerated, ignoring possible absences from schools, workplaces and other social distancing. Finally, it was assumed that implemented measures for detection and movement restriction would work smoothly − heroic assumptions contradicted by other studies (Safman, 2010; Scoones, 2010).