More details/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.

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

Melissa Leach and Ian Scoones

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.

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, shaped 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 interdisciplinary 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. Yet modelling takes many forms. What does it 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 shape its inevitably selective readings of and gazes on the world (Magnani and Nercessian 2009, Mattila 2006, Morgan 2009, Morgan and Morrison 1999, Mansnerus 2012). We connect these insights with understandings of the politics of policy processes (Keeley and 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 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 and 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, three broad types of modelling can be distinguished: 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 et al 2012). Such labelling carries irony, but 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 southeast 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 and 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 2005: 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, work places 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).

Ferguson et al presented their methods and assumptions clearly and transparently, and carried out useful sensitivity analyses on key parameters. Nevertheless they argued definitively that 'we believe that our conclusions are valid for other parts of Southeast Asia' (p.209), and that 'A feasible strategy for containment of the next influenza pandemic offers the potential to prevent millions of deaths... The challenges are great, but the costs of failure are potentially so catastrophic that it is imperative, to ensure that containment is given the best possible chance of success' (p. 213).

Arguably, on the cusp of a potentially major global pandemic detailed questioning of policy-oriented models is neither feasible nor desirable. Reflecting later on managing an emerging influenza pandemic (in this case H1N1), Ferguson and others (Lipitsch et al 2009) highlight the dilemma: the eventual scale is uncertain and 'decisions must be made when the threat is only modest' (p.112). They continue: 'This combination of urgency, uncertainty and the costs of interventions makes the effort to control infectious diseases especially difficult ....in practice, decisions have to be made before definitive information was available on the severity, transmissibility or natural history of the new virus'. However such reflections assume a top-down, expert-led approach, where problems with data exist but can be surmounted through modelling of scenarios and options for policymakers, who make decisions and then 'communicate risks' to the public. But what if the framing and assumptions of the model are off target? In a revealing reflection, Ferguson notes, 'We had to make some assumptions about how a new influenza virus would behave...[but] less detailed statistical work had been done on past pandemics than we hoped' (Nature, 2005b:xi). Addressing this shortfall through multiple model runs and sensitivity analyses cannot redress problems in basic assumptions internal to the framing of the model, or solve the problems of uncertainty (where we don't know the probability of the outcomes), ambiguity (where the outcomes are disputed) and ignorance (where we don't know what we don't know) (cf. Stirling 1999). So - what other modelling approaches might have influenced the debate?

Ducks and rice: an empirical pattern-based model

In 2008 a statistical model, based on a multiple logistic regression framework and published in the *Proceedings of the National Academy of Sciences,* looked at the actual pattern of H5N1 spread in poultry and ducks/geese, again in Thailand (Gilbert et al 2008). The model was

studying empirically what happened, not predicting what might; nevertheless in important respects it qualifies and challenges the Ferguson et al predictions.

The model investigated the statistical association between H5N1 presence and five environmental variables - elevation, human population, chicken numbers, duck numbers and rice cropping density - for three synchronous epidemic waves in Thailand and Vietnam. A statistical spatial risk model was developed for the second epidemic wave in Thailand, and shown to have predictive power in other waves, but also, more surprisingly, in Vietnam.

The analysis was based on a hypothesis, backed by significant data (Gilbert et al 2007, 2006), associating H5N1 prevalence with a particular 'rice-duck farming system', where free-ranging ducks, carrying and shedding substantial amounts of virus, feed in harvested paddy fields. It suggested that H5N1 outbreaks are most likely where such systems exist, notably in lower elevation farming areas with high density of human populations and rice cropping.

This model (and other pattern based analyses, e.g. Pfeiffer et al 2007) had a lesser policy impact than Ferguson et al's. Its timing was later, its focus was birds not people, and its take-home lesson less grand (and profitable) than anti-viral drug stockpiling. However, this work did influence measures, for instance in Thailand, to 'restructure' the duck farming sector – although for good reasons the proposed elimination of free range systems has not been widely adopted (Scoones and Forster 2010).

The Gilbert et al model found associations differing from Ferguson et al's assumptions, so reaching different conclusions. Most notably, while Ferguson et al modelled spread from low population density rural areas, Gilbert et al showed how spread actually occurred from higher population, intensive farming areas. Understanding viral natural history within a particular southeast Asian social-ecological setting enabled evaluation of the potential causal dynamics of H5N1 spread in birds, and so transmission to humans. Gilbert et al and their Asian collaborators knew these systems well, and their model recognised the significance of particular contexts and farming practices in disease ecology.

People's models: local cultural practices and understandings

Alternative perspectives derive from further models, expressed not in statistical formulae or mathematical equations but in arguments about H5N1 from people living with disease emergence, spread and persistence themselves. Studies employing participatory modelling approaches and ethnographic methods have begun to elicit deeper understandings of the often highly specific social relations, cultural practices and ecologies that condition H5N1 dynamics in Asia (e.g. McDermott et al, 2007; Kleinman et al., 2008; Ameri et al 2009; Forster, 2011).

For example Padmawati and Nichter (2008) carried out ethnographies of formal and informal commodity chains, from production through distribution to marketing. They found that likely exposure was highly differentiated by age, gender and occupation, while risk perceptions significantly affected people's behaviour, with clear implications for any age or sex-structured model. Forster (2011) highlighted the importance of wet market practices, trading networks and movement movement in potential transmission patterns. Liu (2008) explored chicken raising and consumption practices in South China, reflecting on the underlying social and cultural factors influencing disease exposure. In particular, relations and co-operative practices between

and within households were shown to affect potential disease spread. Phan et al (2010) explored local management practices for free grazing ducks in Vietnam, while Beaudoin et al (2012) examined risk factors in the Thai context. Both studies help elaborate the social and farming system dynamics of emergence and transmission. Meanwhile, Zhang and Pan (2008) examined relationships between chicken producers and the state in eastern China, showing how resistance to veterinary control and public health measures are enwrapped in a wider, historically situated politics.

Each of these studies therefore produced new data, insights and qualifications, relevant to – and sometimes challenging the assumptions in – other models. Moreover they offer different models of H5N1 dynamics, rooted in experience, cultural understandings and social relations, which complement mathematical process-based and pattern-based models. Yet limited in number, scattered and easy to dismiss as anecdote, such studies have remained relatively marginalised in H5N1 science and policy-making.

In the Ebola case that follows, a different interaction between field realities, modelling and policy emerged, which gave greater space – in the end – for perspectives from participatory modelling to gain purchase.

Case 2: Ebola

Viral haemorrhagic fevers associated with wildlife in forested environments have captured popular as well as scientific and policy concern as deadly diseases emerging 'out of Africa' (e.g. Garrett 1995, Preston 1994). Ebola haemorrhagic fever ('Ebola') has attracted exceptional attention given its 'rapid killing' nature (death occurs in 50-90% of clinically diagnosed cases), with outbreaks occurring nearly every year in East and Central Africa. While some policy narratives focus onlocal public health impacts, the spectre of Ebola outbreaks 'going global' has shaped policy and related scientific efforts from the outset, along with the threat of the Ebola virus being used in bioterror (Polesky and Bhatia 2003). The 1995 outbreak in Kikwit, DRC and worry about global spread was, for instance, key to building political momentum for WHO's revised International Health Regulations in 2005 (Heymann et al, 1999). Meanwhile Ebola's natural reservoir remained enigmatic until its recent, relatively clear association with African bats (Feldman and Geisbert 2011, Leroy et al 2009). In this context, Ebola has been the focus of several sorts of modelling, with different approaches and goals.

The course and control of epidemics: epidemiological process-based models

Mathematical epidemiological models have been used to elucidate the transmission dynamics of Ebola epidemics once underway, and to assess the effects of control measures. Two prominent modelling efforts by Chowell et al (2004) in the *Journal of Theoretical Biology,* and Legrand et al (2007) in the *Journal of Epidemiology and Infection* paramaterised their models using data from the well-documented Ebola outbreaks in DR Congo 1995 and Uganda 2000.

These efforts came at a particular policy moment. Justified by both global outbreak narratives and local public health concerns, by the 1990s a standard set of 'at source' control measures dominated the Ebola response programmes of international agencies such as the WHO and Centers for Disease Control (CDC). Once an outbreak was reported, externally-led teams would establish isolation units for the infected; implement barrier nursing techniques; track and

control those who had had contact with infected individuals; limit supposedly 'dangerous' local behaviours such as washing and burying corpses, and provide health education (Hewlett and Hewlett, 2008, p5). A decade on, there was understandable policy interest in evaluating, and hopefully legitimising, this intervention package.

Chowell et al fitted data to a simple deterministic SEIR (Susceptible, Exposed, Infectious, Removed) epidemic model, allowing an estimate of the basic reproductive number R0 for Ebola - the epidemic growth if everyone is susceptible - and the final epidemic size. Using the model they quantified the impact of intervention measures on the disease transmission rate, and the sensitivity of the final epidemic size to the timing of interventions.

The models suggested potentially devastating pandemics in the absence of control measures. But while Chowell et al analysed the impact of several uncertainties, including viral sub-type, incubation period and infectious period, they ignored others. For instance the model assumes uniform population mixing, and so uniform probabilities of infectious contact – neglecting variations in people's social interactions and hence contact in different settings, and by gender, age, and status. Legrand et al refined this assumption, compartmentalising their otherwise similar model into three stages to account for transmission in the community, in the hospital and during traditional burial. They found that the burial component accounted for the highest proportion of R0 in DR Congo, whereas the community component was more significant in Uganda. Yet even this model could not attend to socially-differentiated contacts in each setting, and their possible implications.

To quantify the impact of control measures, both models assumed interventions to be an identical 'package' in each epidemic, drawing on data concerning their timing. Chowell et al concluded that control measures 'reduce the final epidemic size by a factor of 2', with speed of intervention critical to minimising final epidemic size (2004, p1). Legrand et also concluded that intervention speed, as well as rapid hospitalization, were key to epidemic control. In these cases, modelling focused on interventions ready in place. Not surprisingly, as they used data from these interventions, the models ended up justifying ex post-hoc exactly this intervention approach, albeit with added exhortations for greater speed and effectiveness. The policy value of modelling here was thus not in framing the form of intervention, but in generating authoritative justification for established, top-down, outbreak control approaches.

Yet this modelling-supported policy conclusion overlooked significant differences between intervention styles in Congo and Uganda. As discussed further below, by Uganda 2000 outbreak control strategies were building on local cultural understandings and community involvement – so intervention effectiveness in the two epidemics might reflect not just timing, but also very different approaches. Also, Chowell et al's model was premised on a clear distinction between R0 'before' and 'after' intervention; yet as we show below, existing community-based social protocols were already in play, potentially undermining such assumptions. Finally, both models assume that public health interventions are introduced smoothly with full local compliance. Yet, again, this assumption is contradicted by local evidence, potentially undermining both the models themselves and their policy recommendations.

The macro-ecology of Ebola risk - pattern-based models

In parallel, pattern-based, macro-ecological models have been developed to create risk maps for Ebola, as part of efforts to identify the natural reservoir for the virus (before it was more firmly linked to bats) as well as to target surveillance.. Dominating the literature are ecological niche modelling approaches developed by researchers at the University of Kansas and CDC Atlanta (Peterson et al 2004), and models of the relationship between Ebola outbreaks and environmental/climatic conditions led by researchers at NASA (Pinzon et al 2004). Pitched at a much larger, African regional/continental scale than models of particular outbreaks, these efforts both exploit the growing availability and sophistication of satellite data.

Thus Peterson et al (2004) related the geo-spatial location of documented Ebola outbreaks during 1981-2003 to 'ecological niches' derived from 11 global datasets covering topographical, hydrological and climatic conditions, generating spatial risk maps.Potential geographic distributions were then modelled using a 'Genetic Algorithm for Rule-set Prediction (GARP)', which relates the ecological characteristics of occurrence points to those of points sampled randomly, thus developing and then testing a series of decision rules that best summarize factors associated with Ebola presence, combining the 20 best predictions into a GIS map. Noting that Ebola outbreaks have indeed been recorded in places across the full geographical extent of these ecological conditions (but not outside them), they suggest that this provides a confident basis for targeting surveillance and 'viral reservoir hunting' efforts.

Peterson acknowledged several uncertainties in the model, including the limits of small sample sizes, and the complex relationships between ecological dimensions and distributional limits. Such models are also framed by the choice of ecological variables. The map focused on climate and topography, but would a rather different one have emerged if, for instance, vegetation or land cover had been included? Moreover as Peterson et al acknowledge, spatial ecological niche modeling correlates outbreaks with a static snapshot of environmental conditions, missing any attention to environmental dynamics over time.

Such temporal dynamics were, however, the focus of Pinzon et al, who linked the same 1981-2003 outbreaks with bi-monthly time series satellite data. They showedthat the majority of Ebola outbreaks wereclosely associated with sharply drier conditions at the end of the rainy season. They suggest that such conditions may act as trigger events to enhance transmission of the virus from its (then unknown) reservoir to humans, and that this link might help unravel the enviro-climatic and vegetational coupling of Ebola outbreaks, informing the development of early warning systems.

Again, the researchers acknowledge a range of uncertainties, including 'outlier' outbreak incidences. They offer the model as a step along the way in highlighting conditions favourable for Ebola virus transmission – information critical, not least, for health care workers in Africa. Indeed, in a subsequent presentation (Campbell and Pinzon 2009), they report on further plans to specify such conditions more precisely, taking advance of the growing availability of higher spectral resolution data. Thus a process is envisaged whereby ever more accurate and highly resolved pattern data over space and time will enable better and better risk predictions. But will they ever eradicate all uncertainties? Can such models ever fully encompass the complex interactions between virus, host, ecological, vegetation, climatic and topographical conditions? And what if non-equilibriium dynamics are acknowledged – non-linear interactions between climatic and vegetation variables (cf. Sprugel 1991)? Non-equilbrium dynamics conceptually challenge predictions founded on linear assumptions or probabilistic notions of risk. Moreover

social, ecological and historical research shows African forests to have been shaped by interacting, non-linear human and environmental influences over centuries and millennia (Fairhead and Leach 1998); dynamics invisible in disease risk maps.

Pattern-based models thus contribute to images of forests as either 'virgin', pristine ecosystems in need of protection, or 'viral', places harbouring dangerous pathogens in need of containment (Hardin and Froment, forthcoming). In policy terms, these images combine in prescriptions focusing on reducing contact between people and wildlife, through protected areas or resettlement. More broadly, Jones et al (2008:xx) suggest that 'efforts to conserve areas rich in wildlife diversity by reducing anthropic activity may have added value in reducing the likelihood of future zoonotic disease emergence'. Thus arguments about forest ecosystems and diseases such as Ebola can be mobilised to support 'fortress' conservation measures - despite their negative effects on local rights and livelihoods (Fairhead and Leach, 1998). There is thus congruence between particular zoonotic disease modelling approaches, and particular policy narratives not just related to disease, but in environment and conservation more generally.

Local cultural models - understanding social and ecological dynamics

In contrast with the 'gaze' respectively from numerical datasets and from space offered by process-based and pattern-based models of Ebola, models derived from ethnographic and participatory research offer contrasting views 'from the ground'.

Concerning the ecology of Ebola emergence and risk, a recent review (Feldman and Geisbert 2011) argued for further field-based studies. Several scientistsnow argue that understandings of pathogen dynamics within wildlife populations should be combined with social and anthropological understanding of people-wildlife interactions and risk perceptions in diverse local settings. Thus for instance in Cameroon, researchers linked to the Global Viral Forecasting Initiative have been studying the interrelationships of bushmeat hunting, local perceptions, land use change and settlement expansion, and the emergence of novel diseases including Ebola (Wolfe et al 2005, LeBreton et al 2006). Extending such approaches to attend more deeply to local people's own cultural logics, alternative, policy-relevant perspectives on ecology and disease might emerge. Thus rather than separate people and wildlife, local understandings could provide the basis for integrated 'One Health' interventions compatible with cultural values and livelihood priorities.

Turning to the management of Ebola outbreaks, through pioneering 'outbreak anthropology' by Barry Hewlett and others since the Uganda 2000 epidemic (Hewlett and Hewlett 2008) Ebola has come to exemplify powerfully in global policy circles the value of attending to local cultural logics in the design and implementation of control measures. However the science-policy context for interest in local Ebola knowledge was not 'where there is no data', the typical trigger for institutional interest in participatory epidemiology in low-income country settings. Rather, it was a context of 'where public health interventions fail.'

As already discussed, many dominant policy narratives about Ebola, and the epidemic models they are co-constructed with, portray local populations as ignorant, blaming their social and cultural practices – such as traditional burial - for hastening Ebola transmission, and targeting them for external reform. Yet such top-down control has often met local resistance. In Gabon in 1995-6, for example, American and French Ebola control measures were perceived as so

inappropriate and offensive by villagers that, when international teams arrived to address a further outbreak there in 2001, they met fierce local armed opposition (Milleliri et al, 2004). It was such experiences – and the growing discomfort of field scientists in control teams so resented – that led to anthropologist Hewlett's inclusion in the team addressing the 1999-2000 Ebola outbreak in Uganda.

Using ethnographic and participatory approaches, Hewlett's Ugandan work explored how Acholi people understood and responded to Ebola – a disease that to them was not 'emerging' but a long-embedded part of life. Local concepts encompassed both endemic and epidemic (*gemo*) disease, integrating biomedical, wind-based and spiritual explanations of cause (Hewlett and Hewlett 2008). Once the Acholi identified *gemo*, they would implement control protocols, including isolating the patient in a marked house; having a survivor feed and care for the patient, and limiting general movement. The 'modelling' approach of Hewlett and colleagues, undertaken in interaction with WHO and CDC outbreak teams, enabled such local cultural logics and protocols to be successfully integrated into response strategies. Over the following years, including anthropologists on Ebola outbreak control teams became more institutionalised within WHO. This has helped to shift outbreak control practices towards a greater focus on community engagement, and on attuning technologies and practices to their particular social contexts (Leach and Hewlett 2010).

Yet the policy – and political – implications of attending to local cultural logics go beyond simply blending local knowledge into standard scientifically-justified responses. Rather, local cultural understandings are co-constructed with alternative policy narratives, such as those upholding customary practices or urging that local rights and ethical concerns be balanced alongside disease control aims (see Calain et al, 2009, Jeppsson, 2002, Bausch et al 2007).

Modelling and the politics of policy

In each of these case studies, we therefore see a range of different models, shaped by particular scientific and social practices, and policy and funding contexts. In each case, particular models offer support to – indeed are co-constructed with - particular policy narratives about the disease problem. These different models have contrasting social and political lives. Yet the ways models and their associated policy narratives have interacted – and the politics of this interaction – have been very different.

In the case of H5N1, a narrative around 'control at source' linked to drug stockpiling and containment recommendations from the Ferguson et al model gained substantial policy traction in the context of heightened public, media and political panic. Spatial ecological models looking at causes and drivers questioned some of the assumptions, and focused attention on particular farming systems as sources of risk, yet the standard framing from the original model persisted. Similarly, more grounded ethnographic understandings barely got a look-in, as they complicated and disturbed the outbreak narrative dominating policy circles. Yet, when 'the big one' didn't happen, more reflection and appreciation of local complexities emerged, along with discussion of more integrated 'One Health' approaches (Pfeiffer et al 2012). That is until the next influenza scare, this time H1N1 fromMexico, when once again an antiviral drug response dominated policy discussions, informed by similar models (Forster 2013).In the case of Ebola, there is a continued dominance of policy approaches to outbreak control at source, underwritten by

policy narratives focused on both global and local public health. However the intervention approaches of the WHO, CDC and related agencies have shifted over the last decade to become more locally culturally sensitive, drawing on anthropological insights and field pragmatism. Policy narratives around local cultural logics and local justice hold greater sway, even if this is fragile. At the same time, there is growing research and policy interest in interdisciplinary understandings of disease emergence, towards One Health approaches.

A variety of processes contribute to this co-construction of modelling, power and policy. In relation to any given model, these constitute key dimensions of what we have termed its social and political life; yet they also shape the ways different models interact. First, socio-technical processes - the tools and technologies available to and applied by modellers - are key. These have changed dramatically during the last decade, especially with rises in computer power, virtual and satellite imaging, and information technologies. This has hugely increased the availability of and processing capacity for certain sorts of data – large scale secondary datasets, satellite data – but notably not others. This in turn shapes the 'gaze' on zoonotic disease, favouring the gaze from space, or the gaze from databases – as we saw for risk maps of Ebola, and mathematical models of H5N1 - but not the gaze from the ground.. Second, co-construction is shaped by relations amongst disciplines, and more especially, hierarchies within them. Contextual power relations between natural and social sciences, and between 'central' biomedical/veterinary sciences and those deemed more peripheral such as ecology, are legion in epidemic and zoonotic disease science, despite calls for interdisciplinary approaches. But arguably, even more significant is the ascendancy of quantitative modelling within a whole range of disciplines, from ecology to epidemiology, promoted and valued over and above older more holistic, natural history focused approaches. This is shaped by reflected in forms of prestige, promotion, visibility, publication impact, and success in securing funding applications. The ascendancy of the quantitative in turn renders the qualitative insights from ethnographic and participatory work more easily dismissable as 'mere anecdote' – certainly the case for H5N1 in southeast Asia.

Third, these broader socio-technical and disciplinary processes shape the micro-practices of modelling itself. They condition the details of how scientists draw boundaries, make assumptions, address or black-box issues and uncertainties, choose forms of model parameterisation, select, collect and process data, manipulate technologies, and draw conclusions. Such micro-practices, along with the significance of scientists' social values and positions in shaping their particular readings of the world, are central foci of the sociology of modelling (e.g. Magnani and Nercessian 2009, Morgan and Morrison 1999, Mansnerus 2012). While a detailed exploration for H5N1 and Ebola is beyond the scope of this article, the case studies have hinted at their significance – for instance in the very different ways Asian researchers with embedded experience of rural farming systems, and London-based scientists embedded in global networks, approached the task of modelling H5N1 in Thailand; or the contrasting Ebola modelling practices of NASA laboratories and anthropologists with decades of African experience. .

Fourth, the politics of science advice and funding interplay with these modelling processes. Scientists and modellers need funding for their work, and face growing pressures to demonstrate 'impact'. Policy-makers need justifications for action, and face growing pressures for evidence-based policy. These pressures shape a mutual construction of science and policy that plays out in terms of who and what gets funded, who is invited onto policy committees and

to give briefings, and which forms of evidence are taken up – and which are not. In situations of outbreak emergency or pandemic threat, premium is often on those scientists who can deliver a model quickly, with the right appearance of legitimacy in terms of current norms, and likely to deliver conclusions not too far from established political and policy commitments. Thus for Ebola, epidemiological models that supported well-funded international outbreak control measures found a ready welcome amongst policy agencies; participatory models struggled for legitimacy until dramatic local resistance, provoking policy failure, forced a rethink. For H5N1, in May 2005 a *Nature* editorial bemoaned that the avian influenza response was being conducted 'on a wing and a prayer' (Nature 2005b: 385), yet disaster with millions dead was potentially imminent. This was not 'a Hollywood fantasy', they claimed – it was 'time for action'. Four months later the Ferguson and Longini models were published, into a policy and political context desperate for 'evidence'.

Finally, wider politics shape what these policy commitments are – and hence which kinds of modelling are valued and supported. Details of the politics of zoonotic disease policy processes are beyond the scope of this article, but the stakes and imperatives are broad and diverse. They extend from security and military concerns, to commercial interests in sustaining businesses amidst epidemics or selling drugs and vaccines, to public fears and anxieties, often stimulated by media. In situations of emergency or threat, the political imperative for governments or agencies to 'do something', and advance high-profile claims and actions, may become paramount, perhaps overriding longer-standing political and bureaucratic commitments such as to routine public health.

Conclusion

To address the inherent challenges of understanding complex zoonotic disease dynamics where incomplete knowledge persists requires, we suggest, a different approach to modelling and its relationship with policy, as part of an integrated One World, One Health approach. Reliance on a single model is always dangerous, no matter how careful the modellers. No model can claim to capture everything; reality is too complex to model in full. In the heat of an outbreak moment, a simple model often carries much weight, as we saw particularly with the Ferguson et al H5N1 model. But given its limited data and inappropriate assumptions, was the ensuing purportedly 'evidence based' policymaking any less 'on a wing and a prayer' than before? Could it have been improved by more effective deliberation on the assumptions, data sources and parameter estimates? Could a more empirical look at people-ecology-disease relationships have helped refine the models' epidemiological understandings? Would alternative knowledges and perspectives – of people living with poultry, managing free grazing ducks and taking them to wet markets across the region - have provided a better grounding for, and some important questioning of, dominant policy actions? Would this have saved millions of dollars and much more besides? We believe potentially, yes. In the case of Ebola, the incorporation of ethnographically-grounded approaches along with epidemiological models has already changed policy. Would similar attention to local knowledge and cultural logics around environment-disease interactions question dominant policy and open up new opportunities? Again, we believe potentially yes.

Thus a multi-model approach is, in our view, essential. Different models, as we have shown show different things, and are based on different assumptions, world views and sources of information. Of course they come up with different results. Choosing one over another simply does not make sense. Moreover in the heightened politics of decision-making in epidemic, and

certainly pandemic, contexts, choice of model reflects suitability to the prevailing policy narrative, as much as the efficacy of the model itself.

How to organise a process of triangulation across forms of modelling expertise and data sources, and how to facilitate a deliberation between different models, taking account of realities on the ground, remains a challenge – yet one that any integrated, holistic 'One Health' approach must grasp. It requires both model-guided fieldwork and fieldwork-guided modelling. The challenge is just not disciplinary, data and model integration, but more importantly, deliberation around framing assumptions, cultural understandings, policy narratives, politics and values. In this, the selective partiality of particular models – and their social and political lives - needs to be made explicit, so enabling debate, in particular contexts and settings, about what each might contribute, in whose interests and with what social and political implications. This will require reflexivity, humility and interaction amongst modellers, policymakers and those living with diseases, and an embrace of multiple sources of evidence and analysis in policy. Given uncertainties, ambiguities and multiple framings, science advice to policy must be necessarily plural and conditional, even in – and perhaps particularly in – conditions where outbreaks are possible or happening.

References

Ameri, A.A., Hendrick, S., Jones, B., Mariner, J., Mehta, P., & Pissang, C. (2009). *Introduction to participatory epidemiology and its application to highly pathogenic avian influenza participatory disease surveillance: A manual.* Nairobi: ILRI

Appadurai, A. (1986). *The social life of things*. Cambridge: Cambridge University Press.

BBC (2005). Bird flu 'could kill 150m people.' *BBC Online* 30 September 2005 http://news.bbc.co.uk/1/hi/world/asia-pacific/4292426.stm (accessed 14 January 2013).

Bausch, D.G., Feldmann, H., Geisbert, T.W. & the Winnipeg Filovirus Clinical Working Group (2007). Outbreaks of filovirus hemorrhagic fever: Time to refocus on the patient. *Journal of Infectious Diseases,* 196 (suppl. 2), S136-S141.

Beaudoin, A.L., Kitikoon, P., Schreiner, P.J., Singer, R.S., Sasipreeyajan, J., Amonsin, A., Gramer, M.R., Pakinsee, S. & Bender, J.B. (2012). Risk factors for exposure to influenza A viruses, including subtype H5 viruses, in Thai free-grazing ducks. Transboundary Emerging Diseases. Online DOI: 10.1111/tbed.12043

Calain, P., Fiore, N., Poncin, M. & Hurst, S.A. (2009). Research ethics and international epidemic response: the case of Ebola and Marburg hemorrhagic fevers. *Public Health Ethics,* 2 (1), 7-29.

Campbell, P. & Pinzon, J. (2011). Ecological Context for Ebola Outbreaks in the African Rainforest using Hyperspectral Data. Conference presentation, http://www.isprs.org/proceedings/XXXVIII/8-C23/pdf/Campbell.pdf (accessed 14 June 2012).

Catley, A., Alders, R. and Wood, J. (2012) Participatory epidemiology: Approaches, methods, experiences. *The Veterinary Journal*, 191(2), 151-60.

Chowell, G., Hengartnerb, N.W., Castillo-Chavez, Fenimorea, P.W. & Hymanc, J.M. (2004). The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda. *Journal of Theoretical Biology,* 229(1), 119-26.

Dry, S. & Leach, M. (2010). *Epidemics: Science, governance and social justice*. London: Earthscan.

FAO-OIE-WHO (2010). The FAO-OIE-WHO Collaboration: Sharing responsibilities and coordinating global activities to address health risks at the animal-human-ecosystems interfaces: A Tripartite Concept Note. http://www.oie.int/fileadmin/Home/eng/Current_Scientific_Issues/docs/pdf/FINAL_CONCEPT_NOTE_Hanoi.pdf (accessed 19 January 2013).

Fairhead, J. & Leach, M. (1998). *Reframing deforestation: global analyses and local realities – studies in West Africa*. London: Routledge.

Feldman, H. & Geisbert, T.W. (2011). Ebola haemorrhagic fever. *Lancet*, 377, 849-62.

Ferguson, N. (2007). Capturing human behaviour. *Nature*, 446: 733.

Ferguson, N. M. et al. (2005) Strategies for containing an emerging influenza pandemic in Southeast Asia. *Nature*, 437, 209-214.

Ferguson, N.M., Cummings, D.A.T., Fraser, C., Cajka, J., C., Cooley, P., C., & Burke, D. (2006). Strategies for mitigating an influenza pandemic. *Nature*, 442: 448-52.

Forster, P. (2013). To pandemic or not? Reconfiguring global responses to influenza. *STEPS Working Paper* XXX. Brighton: STEPS Centre.

Fraser, C., et al., (2009). Pandemic Potential of a Strain of Influenza A (H1N1): Early Findings. *Science,* 324, 1557-1561.

Garrett, L. (1994). *The coming plague: newly emerging diseases in a world out of balance.* Penguin: New York.

Gilbert,M., Chaitaweesub,P., Parakamawongsa,T., Premashthira, S., Tiensin, T., Kalpravidh, W., Wagner,H. & Slingenbergh, J. (2006). Free-grazing ducks and highly pathogenic avian influenza, Thailand*. Emerging Infectious Diseases*, 12 (2), 227-234.

Gilbert, M, et al. (2007). Avian influenza, domestic ducks and rice agriculture in Thailand. *Agriculture, Ecosystems and Environment*, 119, 409–415.

Hardin, R. and A. Froment, forthcoming, 'From virgin forests to viral forests', in Hardin, R. (Ed), forthcoming, *Socioemergence: Cultural economy and historical ecology of viral disease in tropical forests*. Duke University Press.

Hewlett, B. & Hewlett, B. (2008). *Ebola, culture and politics: The anthropology of an emerging disease.* Wadsworth Books.

Heymann, D.L., Barakamfitiye, D., Szczeniowski, M., Muyembe-Tamfum, J.J., Bele, O. & G. Rodier. (1999). Ebola hemorrhagic fever: lessons from Kikwit, Democratic Republic of the Congo. *Journal of Infectious Diseases*, 179 Suppl 1, S283-6.

Jasanoff, S. (Ed) (2004). *States of knowledge: The co-production of science and social Order.* London: Routledge.

Jeppsson, A. (2002). Defend the human rights of the Ebola victims! *Tropical Doctor*, 32(3), 181-2.

Jones K.E., Patel, N., Levy, M.A., Storeygard, A., Balk, D., Gittleman, J.L. & Daszak, P. (2008). Global trends in human emerging infectious diseases. *Nature,* 451, 990-993.

Keeley, J. & Scoones, I. (2003). *Understanding environmental policy processes: Cases from Africa.* London: Earthscan.

Kleinman, A.M., Bloom, B.R., Saich, A., Mason, K.A & Aulino, F. (2008). Asian flus in ethnographic and political context: A biosocial approach. *Anthropology & Medicine*, 15:1, 1-5.

LeBreton, M., Prosser, A.T., Tamoufe,U., Sateren, W., Mpoudi-Ngole, E., Diffo, J.L.D., Burke, D.S. & N. D. Wolfe. (2006). Patterns of bushmeat hunting and perceptions of disease risk among central African communities. *Animal Conservation,* 9, 357–363.

Legrand, J., Grais, R.F., Boelle, P.Y., Valleron, A.J., & Flahault, A. (2007). Understanding the dynamics of Ebola epidemics. *Epidemiology and Infection,* 135, 610-621.

Leibler, J., Otte, J., Roland-Holst, D., Pfeiffer, D., Soares Magalhaes, R., Rushton, J., Graham, J. & Silbergeld. (2009). Industrial food animal production and global health risks: Exploring the ecosystems and economics of avian Influenza. *EcoHealth*, 6(1), 58-70.

Leroy, E.M., Epelboin, A., Mondonge, V., Pourrut, X., Gonzalez, J-P., Muyembe-Tamfum, J-J., & Formenty,P. (2009), Human ebola outbreak resulting from direct exposure to fruit bats in Luebo, Democratic Republic of Congo, 2007. *Vector-Borne and Zoonotic Diseases,* 9(6), 723-728.

Lipsitch, M., Riley, M., Cauchemez, S. Ghani, A. & Ferguson, N. (2009). Managing and reducing uncertainty in an emerging influenza pandemic. *New England Journal of Medicine*, 361, 112-115.

Liu, T-S. (2008). Custom, taste and science: Raising chickens in the Pearl River Delta Region, South China. *Anthropology & Medicine*, 15:1, 7-18.

Longini, I. M. Jr et al. (2005) Containing pandemic influenza at the source. *Science,* 309,

1083–-1087.

Magnani L. & Nersessian, N. (Eds.) (2009). *Model-based reasoning: Science, technology, values*. New York: Kluwer & Plenum.

Mansnerus, E. (2012). Using model-based evidence in the governance of pandemics. *Sociology of health & illness*, online ISSN 0141-9889

Mattila, E. (2006). Interdisciplinarity in the making: Modelling infectious diseases. *Perspectives on Science: Historical, Philosophical, Sociological,* 13 (4), 531-53.

Milleliri, J.M., Tevi-Bennissan, C., Baize, S., Leroy, E. & Georges-Courbot, M.C. (2004). Epidemics of Ebola haemorrhagic fever in Gabon (1994-2002): Epidemilogic aspects and considerations on control measures. *Bulletin de la Societe Pathologiques Exotiques*, 97(3), 199-205.

Morgan, M.S. (2009). *The World in the Model.* Cambridge: Cambridge University Press.

Morgan, M. & Morrison, M. (Eds.) (1999). *Models as mediators: Perspectives on natural and social science*. Cambridge: Cambridge University Press.

McDermott, J., Mariner, J., Rich, K. & Omore, A. (2007). Combating avian influenza in developing countries: the role of applied research in improving rural livelihoods. *Bulletin of Animal Health and Production in Africa*, 47-54.

Nature. (2005a). Editorial: On a wing and a prayer. *Nature,* 435, 385-386

Nature. (2005b). Making the paper: Neil Ferguson. *Nature* 437, xi.

Padmawat, S. & Nichter, M. (2008). Community response to avian flu in Central Java, Indonesia. *Anthropology & Medicine*, 15:1, 31-51.

Peterson A.T., Bauer, J.T. & Mills, J.N. (2004). Ecologic and geographic distribution of filovirus disease. *Emerging Infectious Diseases,* serial online, URL: http://wwwnc.cdc.gov/eid/article/10/1/03-0125.htm (accessed June 14 2012).

Pfeiffer, D., Minh, P., Martin, V., Epprecht, M. &, Otte, J. (2007) An analysis of the spatial and temporal patterns of highly pathogenic avian influenza occurrence in Vietnam using national surveillance data. *The Veterinary Journal*, 174, 302–309.

Pfeiffer, D., Otte, J., Roland-Holst, R. & Zilberman, D. (2012) A one health perspective on HPAI H5N1 in the Greater Mekong sub-region. *Comparative Immunology, Microbiology and Infectious Diseases*, http://www.sciencedirect.com/science/article/pii/S0147957112001294

Phan, M.Q., Stevenson, M.A., Schauer, B., Morris, R.S, & Quy, T.D., (2010). A description of the management of itinerant grazing ducks in the Mekong River Delta of Vietnam. *Preventive Veterinary Medicine*, 94(1-2), 101-107.

Pinzon, J.E., Wilson, J.M., Tucker, C.J., Arthur, R., Jahrling, P.B. & Formenty, P. (2004). Trigger events: enviroclimatic coupling of Ebola hemorrhagic fever outbreaks. *American Journal of Tropical Medicine and Hygiene,* 71(5), 664-74.

Polesky , A. & Bhatia, G. (2003). Ebola hemorrhagic fever in the era of bioterrorism. *Seminar on Respiratory Infections,* 18(3), 206-15.

Preston, R. (1994) *The Hot Zone*. New York: Anchor Books.

Roe, E. (1994). *Narrative policy analysis: Theory and practice.* Durham, NC: Duke University Press.

Scoones, I. (Ed.) (2010). *Avian influenza: Science, policy and politics.* London: Earthscan.

Scoones, I. & Forster, P. (2010). Unpacking the international response to avian influenza: Actors, networks and narratives. In Scoones, I. (Ed.). Avian influenza: Science, policy and politics, pp. 19-63. London: Earthscan.

Shackley, S. & Wynne, B. (1995). Global climate change: The mutual construction of an emergent science-policy domain. *Science and Public Policy*, 22.

Sprugel, D.G. (1991). Disturbance, equilibrium, and environmental variability: What is 'natural' vegetation in a changing environment? *Biological Conservation* 58, 1-18.

Stirling, A. (1999). *On science and precaution in the management of technological risk*. EC Joint Research Centre: Seville.

Van Kerkhove, M.D., Mumford, E., Mounts, A.W., Bresee, J., Ly, S., Bridges, C. & Otte, J. (2011). Highly pathogenic avian influenza (H5N1): Pathways of exposure at the animal-human interface, a systematic review. *PLoS ONE* 6(1), e14582. doi:10.1371/journal.pone.0014582

Wolfe, N.D., Daszak, P., Marm Kilpatrick, A. & Burke, D.S. (2005). Bushmeat hunting, deforestation and prediction of zoonotic disease. *CDC Perspective*, 11(12).

Zhang, L. and Pan, T. (2008). Surviving the crisis: Adaptive wisdom, coping mechanisms and local responses to avian influenza threats in Haining, China. *Anthropology & Medicine*, 15:1, 19-30.