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ANIMAL HEALTH

How to control bovine tuberculosis

A model of the transmission and spread of bovine tuberculosis in Britain suggests that controlling the epidemic will require large-scale cattle slaughter or a major rethink of combined control strategies.

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Bovine tuberculosis is a huge economic and social problem in Britain, with control measures costing taxpayers about £100 million (US$170 million) per year. Control is technically problematic and politically controversial because of the involvement of wild badgers in the maintenance and transmission of the infection[1]. Badgers are alternately much-loved and loathed in British society[2] and badger culling has complex positive and negative outcomes for disease in cattle[3–5]. In a paper published on *Nature*'s website today, Brooks-Pollock *et al.*[6] describe a dynamic model of bovine tuberculosis transmission within and between farms that is unprecedented in its scale, realism and approach. By taking bovine tuberculosis by the horns, they have provided an instructive but ultimately disheartening insight into prospects for controlling this chronic disease.

Brooks-Pollock and colleagues modelled the entire national cattle herd, using records of the daily movements of 30,000 cattle between farms and real protocols for bovine tuberculosis (bTB) testing and movement controls. Their approach was computationally intensive, it acknowledged uncertainty and it used real-life evidence of bTB incidents in cattle herds and numbers of infected cattle to 'reverse engineer' the parameters of transmission that could give rise to the observed epidemic (Fig. 1).

The authors also scrutinized cattle-testing protocols that rely on a skin test with a known tendency to miss true positives[7]. They highlight the fact that reliable detection in a herd depends on there being multiple infected individuals and that there is a high probability of infected animals being left behind in the herd, from which subsequent cases can be seeded. Thus, clearing infection from herds is extremely problematic. However, Brooks-Pollock and colleagues worked within this diagnostic constraint by distinguishing between infected herds and herds in which infection had been detected, allowing them to model the consequences of missing and moving infected cattle. They also assessed the importance of infection from the local environment, comprising direct transmission from wildlife (primarily badgers) and indirect exposure to environmental contamination stemming from cattle and/or wildlife.

The model reveals that movement of infected animals was responsible for 84% of newly infected farms and, crucially, that there was a heavy bias towards as few as 10% of farms, characterized by selling many animals, as the source of nearly all secondary cases. Similarly, a small minority of farms seemed to be responsible for spread through the environment. Thus, a key finding of the study is that a small proportion of farms probably function as superspreaders of bTB infection.

The causes of incidents of infection in a herd were evenly spread across three principal processes: introduction of an infected animal; transmission from the local environment, including from wildlife; and an infected animal being left behind after testing and slaughter of test-positive animals. No single cause dominated, so it comes as little surprise that when the authors tested potential control treatments in their model, they found single, discrete strategies to be generally ineffective in checking the growth of the epidemic.

In fact, only one of ten treatments — slaughtering the whole herd upon detection of bTB — was effective in rapidly bringing all measures of the epidemic well below the baseline. The model suggests that this treatment would result, in the year after implementation, in drastic reductions in the number of cattle testing positive for bTB and in removal of restrictions from much of the national herd. However, this is a silver bullet that would cause severe wounding to the cattle industry, because it would come at the cost of a one-off, 20-fold

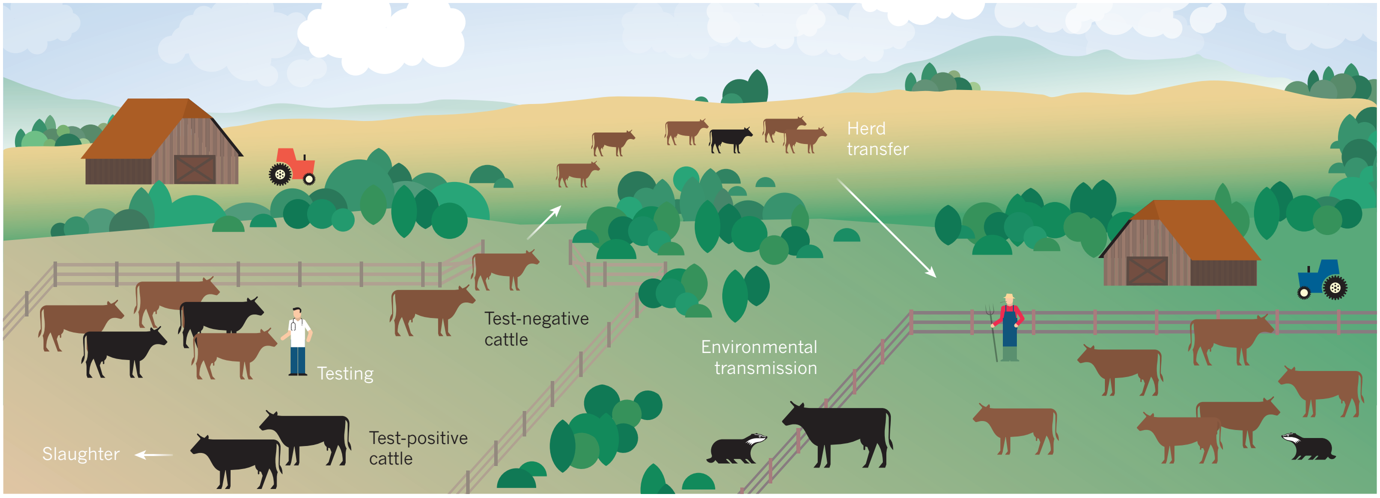

Figure 1 | Bovine tuberculosis spread. Brooks-Pollock *et al.*[6] present a model that parameterizes the complex processes involved in the spread of bovine tuberculosis in Great Britain, including cattle movement, cattle testing (incorporating problems with current diagnostics that miss true positives), environmental exposure and control strategies for cattle and wildlife. Among other findings, the model suggests that a minority of farms act as superspreaders of infection and that control of badger populations is unlikely to stem the growth of the current epidemic.

increase in cattle slaughtered in the first year of the policy. Such a cost is reminiscent of the catastrophic scale of Britain's 2001 experience of controlling foot-and-mouth disease and is likely to be horrifying to policy-makers, farmers and animal-welfare campaigners alike. Faced with the current epidemic trajectory, however, the authors suggest that it might be judicious to contemplate how such severity trades off against ongoing, large-scale culling well into future decades.

The model also revealed that a one-off test of the national cattle herd would bring success in reducing all measures of disease some three to four years later. This treatment was implemented in Wales in 2008–09, and the model's predictions are consistent with current tentative observations of bTB decline in Wales. The model further predicts that improved cattle-test performance would help to reduce trade restrictions, but at a cost of higher numbers of cattle culled, and that cattle vaccination offers some encouraging prospects, although not at the usual level expected for mass vaccination campaigns. Disappointingly for supporters of England's current pilot badger culls, an across-the-board 50% reduction of all sources of environmental transmission, simulating successful culling or vaccination of badgers, was shown by the model to have little impact on any measures of cattle disease, and failed to prevent ongoing growth of the epidemic.

Brooks-Pollock and colleagues' complex modelling approach therefore provides two simple messages: first, that direct, generic controls imposed on cattle provide the greatest leverage for acting on the current mass of the bTB epidemic; and second, that although the environment is an important source of infection, indirectly tackling cattle disease by managing wildlife, by whatever means, is likely to yield unimpressive results and contribute little biologically to controlling a national epidemic.

Although their work is compelling, the authors' job is not complete. Policy-makers often speak of their use of a 'package of measures' and 'every tool in the box' in controlling bTB, particularly with respect to controversial policies to cull badgers. Measures aimed at wildlife could contribute to bTB control in Britain, perhaps as part of achieving consensus among stakeholders in conflict or, most importantly, in the eventual transition from control to eradication of infection. But having shown that individual strategies, short of draconian whole-herd culling, will not rapidly turn this epidemic around, Brooks-Pollock *et al.* now have the perfect opportunity to use their model to test policy-makers' expectations of their various packages and toolboxes and to gauge their overall cost-effectiveness. UK governments and the farming industry badly need such guidance to frame and implement their overall strategy and to manage expectations among their diverse stakeholders. ■

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