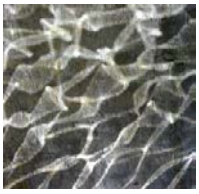

Veterinary Realities: What is Foot and Mouth Disease?

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Abstract

Veterinary science draws on different traditions for knowing and acting, and mobilises different kinds of materials and techniques. This article explores these differences and their tensions for the diagnosis of foot and mouth disease in the UK in 2001. It shows that when they talk of foot and mouth disease, different veterinary traditions refer to the different objects. The clinic looks for deviances in animals, the laboratory detects the presence or otherwise of virus, while epidemiology focuses on patterns of transmission in populations. Despite the fact that they use the same word, clinic, lab and epidemiology are each involved with their own specific ontological variant of 'the' disease. At the same time other figures and configurations shift with the disease. This means that it is not possible to map different versions of foot and mouth disease onto a background of shared co-ordinates. So in 2001 clinic, lab and epidemiology diagnosed foot and mouth disease mobilising different kinds of materials, the entities inhabiting these practices had different qualities and they operated in different ways. Even time lines and spatial relations changed. Such differences are usually treated as a matter of perspective: it is assumed that everyone is looking at a single world. The article challenges this assumption by arguing that different veterinary traditions draw upon and contribute to different worlds in the plural. This shift makes it easier to explore the strengths of these worlds, their drawbacks and their limitations

Introduction

As veterinary science diagnoses, treats and seeks to prevent animal diseases it draws on different traditions for knowing and acting, mobilises different kinds of materials and takes on board different techniques. In this article we explore the character of this heterogeneity and the tensions that arise when the various traditions of veterinary science work together in practice. We do this by analysing a particular case: that of the diagnosis of foot and mouth disease in the UK in 2001.[1]

First we show that there is a problem with the object that lies at the heart of the epidemic, with foot andmouth disease itself.We argue that when they talk of 'foot and mouth disease', different veterinary traditions are not referring to the same 'thing'.

The object they are searching for, measuring or tracing is different. The clinical tradition looks for *deviances* in animals, the laboratory detects whether or not a virus is present in the animal's blood or tissues, while epidemiology focuses on patterns of *transmission* in animal populations.

This does not mean that people trained in different traditions are looking at a single object from different perspectives. Instead, something quite different is going on. Each tradition involves different interactions with the object and different practices of investigation. Each attends to something different. Each does something different, too; it uses different techniques and asks different questions. All this tells us that dealing with a disease in different ways is not a simply a matter of having a different perspective on reality. Instead, it is the *reality* being dealt with that shifts between practices. Our first claim, then, is that despite the fact that they use the same word, clinic, lab and epidemiology are each involved with their own specific ontological variant of 'the' disease. Foot and mouth is not singular. It is a composite.

But, and this is our second argument, it is not just 'foot and mouth disease' that shifts between veterinary repertoires. So, too, do a host of other figures and con- figurations. The consequence is that it is not possible to map different versions of foot and mouth disease onto a background of shared co-ordinates. Instead, those co-ordinates are on the move too – and again this is not just a matter of meanings but has to do with practices. In 2001 clinic, lab and epidemiology diagnosed foot and mouth disease by mobilising different kinds of *materials*. The entities inhabiting these practices had different *qualities* that became important because they worked in different ways. Even time and space did not form a shared backdrop. Clinic, lab and epidemiology each configured its own distinctive *time line* and its own set of *spatial relations*.

All in all, then, there are huge tensions within veterinary science. Though practitioners recognise that there are differences, these are usually treated as a matter of perspective. Turned into ways of seeing, they are assumed to belong to people and to reflect their points of view. The assumption is that everyone is looking at a *single world*. But this is what we want to challenge. Thus, our question is: what happens if we insist instead that different veterinary traditions draw upon and contribute to *different worlds*, worlds in the plural? Our response is that once we do this it becomes easier to explore the relevant strengths of these worlds, their drawbacks and their limitations. As long as perspectivalism reigns these are the kinds of questions that cannot be asked. But this is precisely what we want to do.

In what follows we lay out a few basic but crucial differences between the worlds enacted by the clinic, the lab and epidemiology. Though we do this in what we hope is a balanced way, to be balanced here is not necessarily to be neutral, for as things stand, the ways of working specific to the clinic are under pressure. Instead of receiving the public appreciation and policy investment they deserve they are in the process of being marginalised. In that context, a balanced analysis necessarily becomes a song of praise for the clinic.

Different objects

The clinic

As a veterinary reality, the case of foot andmouth disease in 2001 in the UK has a clear beginning. This was on 19 February. On that day the resident vet of Cheale's abattoir in Brentwood, Essex, Craig Kirby

> was in the abattoir carrying out ... [his] normal duties. We had pigs and sows on site ready for slaughter. As is normal, the pigs were dealt with first, and gave ... [him] no cause for concern. (Food Standards Agency 2001)

> The sows were next, 27 of them grunting and jostling towards the vet in the khaki overalls. Within seconds Mr Kirby spotted something unusual: some had blisters around their mouths. He pulled a recalcitrant swine towards him for a closer look. (Harrison 2001)

Here's what happened next:

> Mr Kirby examined the animals and saw how serious the problem was. Clinical signs alone can not [sic] distinguish swine vesicular disease from FMD. Both are notifiable diseases. He assumed, even hoped it was swine vesicular disease. First he stopped the production line. Then he telephoned the local office of the State Veterinary Service. About an hour later, after inspection by two government vets, one of whom had experience of FMD in Greece, there was no doubt. This was either swine vesicular disease or FMD. Only laboratory work could tell which. (House of Commons 2002, p. 20)

Here, the clinical expertise of the government vets was added to that of the resident vet of the abattoir. Strengthening one another's assessments, they saw that there was 'disease' and narrowed down the many possible diagnoses to two: swine vesicular disease or foot and mouth disease. In order to further differentiate between these, blood and tissue samples from diseased animals were sent to the lab for testing. Thus the clinical signs were not enough for the final verdict, but they were consequential. Mr Kirby stopped the production line on the basis of his clinical assessment and when his colleagues also confirmed that the signs were suspect the lab was put to work. These are the signs that alarmed the vets: the sows *sounded* wrong and they *looked* wrong.

Clinical diagnosis of foot and mouth disease depends on:

> [E]xamination of the visible mucous membranes of the conjunctiva, nose, mouth, tongue and eyes and the external surface of the body and limbs. Recognition in cattle and pigs is usually relatively easy. (Royal Society 2002, p. 78)

Blisters are important. Here is a veterinary manual talking about animals with foot and mouth disease:

> The clinical signs [of FMD] are more severe in cattle and intensively reared pigs than in sheep and goats.... In cattle and pigs, after the incubation period, anorexia and fever of up to 106°F (41°C) may develop.... Vesicles may ... appear on the teats and udder, particularly of lactating cows and sows ... animals show a loss in condition and growth rate that may persist after recovery.... Lesions on the mammary gland and feet frequently develop secondary infections.... In pigs, the complete horn of the toe may be lost. (Merck 2008)

Fever, anorexia, loss of growth and productivity, blisters on the mouths and feet and other areas including the teats, lameness, in some cases extreme, pain on walking and the noisy expression of that pain, together with the onset of secondary infection; these are some of the clinical signs of foot and mouth disease. In farms and slaughterhouses vets tune in to these signs to make their diagnosis. In clinical practice, then, foot and mouth disease is a disease that surfaces in a specific set of signs. Detecting it involves hearing, seeing and touching living animals in a specific and skilled manner (Moreira 2004). Kirby was trained to recognise FMD symptoms at the Edinburgh University Veterinary School (Harrison 2001) but he had never actually seen them before. That one of the governments vets had, albeit in Greece, deserves mention because it adds to the credibility of the diagnosis. Clinical skills, then, depend on lived experience. They are embodied in people with histories that are organised to assemble skills and yet are always particular and idiosyncratic.

In clinical practice foot and mouth disease is something that may be read from signs on the surface of the body. At the same time these deviant signs are significant because they are signs 'of' something: of a disease that troubles the animal that shows the signs. The animal is not well. This calls for immediate action. What kind of action? In the abattoir, where the animals did not come to be cared for but to be killed, the production line is stopped and test samples are collected and sent on to the lab for further specification.

The laboratory

The lab to which the test samples were sent was the Institute for AnimalHealth (IAH) at Pirbright in Surrey, south-west of London. There, other vets and technicians got to work:

> The first ELISA test to detect FMD ... was started at 0900 on Tuesday 20 February It was completed by 1330 and confirmed as a positive.MAFF [Ministry of Agriculture, Fisheries and Food] was informed at 1350. (House of Commons 2002, p. 54).

The lab confirmed the diagnosis of 'foot andmouth disease'. This implied a phone call to London, as it called for immediate action at MAFF. Here we will not explore the actions of the Ministry. Instead, we consider the lab's diagnostic practices, and more particularly the ELISA test.

ELISA stands for enzyme-linked immunosorbent assay. The standard 2001 international manual (Office Internationale des Épizooties [OIE]) tells us that the 'preferred procedure for detecting FMD' is the indirect sandwich ELISA. This test detects virus in a sample by drawing on the ability of bodies to detect virus immunologically. Bodies learn to recognise particular parts of a virus that immunology calls 'antigens'. In response to the presence of antigens the immune systemmakes 'antibodies'. Blood containing such antibodies is called 'antiserum'. The ELISA test uses antiserummade by living rabbits that have been infected with one of the different kinds ('serotypes') of the foot and mouth virus. This is how it goes:

Step one:

> [D]ifferent rows in multiwell plates are coated with rabbit antisera to each of the seven serotypes of FMD virus. These are the capture sera. The preparation is rinsed and binding sites that aren't specific to FMD are blocked. (Kitching et al. 2000)

A multiwell plate is a plastic surface punched with rows of little test-tubes or 'wells'. These wells are primed with antisera to 'capture' any FMD antigens from test samples. Blocking prevents the capture of antigens of other kinds of virus that might confuse the result, whilst rinsing removes anything that is not stuck to the surface of the wells and might therefore also mess up the experiment.

Step two:

> Test sample suspensions are added to each of the rows, and appropriate controls are also included. (Kitching et al. 2000)

The samples come from the bodies of pigs, cows or sheep suspected of having the disease. If these contain viral antigens they react with the antibodies in the antiserum. Accordingly, two layers are stuck to the walls of the wells, first the rabbit antibodies of the capture sera and second, linked to these, the viral antigens of the sample being tested.

Step three:

> Guinea-pig antisera to each of the serotypes of FMD virus are added. (Kitching et al. 2000)

This is a third layer in the sandwich. Antibodies (made by guinea pigs) in the new layer of antisera stick to viral antigens in the second layer captured by the first antibody layer (taken fromrabbits). As there are various kinds of FMD virus (different 'serotypes'), different antisera are used. Then, in the fourth step, this is

> followed by [adding] rabbit anti-guinea-pig serum conjugated to an enzyme. (Kitching et al.2000)

What is being added in the fourth layer is a 'conjugate' – a molecule combining an enzyme and a rabbit antibody that, this time, is not an antibody to foot and mouth disease virus, but to guinea pig. The rabbit antibody to guinea pig sticks to the guinea pig serum that has itself been captured by viral antigen that bound onto the rabbit antibodies against foot and mouth disease virus. If there is viral antigen in the test sample the binding chain is now four molecules long.

Finally, as a fifth step, a chemical is added that reacts with the rabbit enzyme in layer four. As the OIE Manual puts it:

> A colour reaction on the addition of enzyme substrate, indicates a positive reaction.

In this, the fifth and final layer, a result, positive or negative, can be read from the presence or absence of colouring.

The Manual reveals that the ELISA test involves a whole series of fiddly moves and gestures. It is partly automated (the procedure has developed further since 2001) but it still involves micro-pipetting a lot of liquids containing antibodies and antigens into a large number of wells in conditions that need to be meticulously clean (contamination may lead to false results.) It involves making sure that the right amount of sample of the right

dilution is added to each well (different amounts and different concentrations will affect the result). It involves temperature control (temperature has

a big impact on biochemical reactions). It involves encouraging reactions by gently shaking the plates (during incubation multiwell plates are placed on a rotary shaker). It requires careful attention and record-keeping. (Anyone who has ever been in a lab, knows that it is surprisingly easy to mix up samples). It demands (we only mentioned this once above) meticulous washing between each step in the process with phosphate buffered saline. (The test is not reliable if there are unbound antibodies or antigens present). It demands careful control of pH, that is, of acidity/alkalinity balance. (FMD virus only survives at a very small pH range). It demands accurate timekeeping. (The reactions are time-dependent. So the protocol demands that steps two, three and four last for an hour, while step five, the colour fixation, is stopped after fifteen minutes.) And it demands some kind of assessment of the colour produced by the assay either by eye or, in marginal cases, by photometric light absorption at the appropriate wavelength.

Many of the materials (the microwell plates, the rotary shaker, the antibodies, the buffered solutions) are commercially available, or can be obtained from the OIE/FAO World Reference Laboratory for FMD, (Kitching et al. 2000). However, the ELISA test also depends on the appropriate organisation of laboratory protocols, skills and materials. This means that the practice is fairly robust, but it is not very robust. It has to be done just right, which is why the manuals are meticulously prescriptive and the OIE designates a world reference laboratory to set and monitor standards. So ELISA tests may be used to detect foot and mouth disease. But what is foot and mouth disease in relation to an ELISA test? Since it works by examining the colour of test-tube wells, one might say that the test enacts the disease as a set of coloured wells resulting from circumscribed procedures. But in practice these colours are given meaning by linking them to the virus or, rather, the viral antigen. Here is the OIE manual again:

> Diagnosis of FMD is by the demonstration of FMD viral antigen in samples of tissue or fluid. (Kitching et al. 2000).

This suggests that the ELISA test enacts foot and mouth disease as a *viral reality* and *a virus as an object carrying antigens that can be detected by using antibodies as detectors*. Note that this is *not* what foot and mouth disease is 'in general' (in the clinic it is something different) and not even what foot and mouth disease is 'in the laboratory', as there are others tests that enact the disease differently. As it happens, the complement fixation test that preceded ELISA also enacted the disease as one related to a virus that induces an antigen/antibody reaction. But electron microscopy treats a virus as a structure that may be stained or coated, detected and visualised. And then, in the various versions of reverse transcriptase polymerase chain reaction (RT-PCR), a virus is enacted as an entity that possesses a specific and detectable set of ribonucleic acid nucleotides that may be visualised as displaced bands in gel electrophoresis.

Thus, even within laboratories, foot and mouth disease is not a single object but is enacted in different ways. However on 20 February 2001 in the UK an ELISA test at the IAH laboratory at Pirbright confirmed the diagnosis of foot and mouth disease when it concluded that the samples brought in from Cheale's abattoir were 'positive'.

Forensic epidemiology

It was only after all this activity that the Pirbright laboratory informed MAFF that it had established the presence of foot and mouth disease in pigs brought in for slaughter to Cheale's abattoir. In order to act upon this finding, however, the Ministry needed additional knowledge, knowledge of another kind. For if you are a single vet faced with a single diseased animal, clinic and laboratory are telling enough. Their joint diagnosis provides sufficient basis for action. However, the Ministry is not concerned with individual animals but with the national herd. It has a responsibility for all animals (pigs, cows, sheep and goats) that might already be, or shortly get, infected by foot and mouth disease. And it is not just the health of the nation's animals that worries the Ministry, but their economic value as well. And this value drops for all animals, even for those that escape infection. Because as long as there is foot and mouth disease around, the entire UK loses it disease-free status. International agreements and laws stipulate that without a generalised disease-free status a country may no longer generally export its livestock, live or slaughtered.[2]

Against this background it was important to know where the virus came from and where it had spread. This was because it was to be eradicated by culling, by killing all animals that might be vehicles for the further spread of foot and mouth disease. But which animals might these be? In order to get a handle on this, the veterinary offers of the MAFF mobilised *epidemiological* repertoires for knowing about foot and mouth disease. These have to do with *patterns of transmission*. As the textbook puts it:

> FMD spreads most effectively when susceptible animals are closely confined. Virus is present in the excretions, mostly faeces, and secretions such as milk, saliva and breath of infected animals. Animals become infected through inhalation or contact of the virus with mucosal membranes, especially in the mouth and nostrils.
>
> Cattle and sheep are very susceptible to airborne virus.... Airborne FMD virus can be carried great distances on wind plumes depending on weather conditions. (House of Commons 2002, p. 46)

Armed with knowledge of this kind, knowledge about how the foot and mouth virus *might* spread, the veterinary officers engaged in forensic epidemiology.[3] They sought to trace how it actually *had* spread.

> The oldest lesions were seen in two groups of pigs from the Isle of Wight and Buckinghamshire that had entered the abattoir on the 16th February.More recent lesions were seen in the third group of pigs that had arrived from Yorkshire on the 18th February and in a group of pigs that arrived from Suffolk on 19 February, suggesting a very short incubation period.[4] (Department for Environment Food and Rural Affairs (2002, p. 12)

The vets subsequently went to the relevant farms but found no animals with clinical signs of foot and mouth. This suggested that the animals had caught the disease at Cheale's, which meant that it had come from somewhere else. But where? This led to more veterinary detective work. MAFF Veterinary Officers

> visited all premises that had supplied livestock to the abattoir during the previous two weeks. Tracings were prioritised and visits to premises feeding waste food to pigs were undertaken first. (Department for Environment Food and Rural Affairs 2002, p. 13)

This wasn't easy. There were 600 farms, the veterinary service was seriously overstretched and it took 48 hours to go through Cheale's orderly but handwritten records (House of Commons 2002, 1587, 57). In an attempt to trace the infection back to its source, the epidemiologists thought in terms of the *likelihood of transmission* from one animal to another: 'Pigs are relatively resistant to airborne virus but very susceptible to contact infection, such as by eating infected feed' (House of Commons 2002, 1587, 42).

So the infection *might* have been blown to Cheale's but it was more likely that it had arrived in the form of already-infected pigs. And (further epidemiological logic) though all the farms needed inspection, infected feed was a likely source of infection, so it made sense to begin with farms using catering waste. Very few farms (only 92 in the UK) were licensed to do this (Parliamentary and Health Service Ombudsman 2007, p. 17), which is why the vets visited Burnside Farm at Heddon on theWall early on in the search, where 'inspection quickly revealed the presence of widespread FMD, clearly well-established, with old lesions in many pigs. The younger pigs in particular were visibly unwell/unthrifty (Dring 2001, p. 2). At this point the epidemiology branched out in two directions. 'Upstream', the vets tried to work out by process of elimination how the infection had arrived at the farm. All the plausible pathways of possible infection were investigated. The following paragraph mentions seven:

> Investigations have shown no evidence that disease was introduced to the farm by animals, people, vehicles, equipment, vermin, wildlife etc. There was no evidence of disease on premises within 3km of Burnside Farm which predates that found there. (Department for Environment Food and Rural Affairs 2002, p. 3)

An eighth, however, catering waste, was not eliminated. Already a cause for suspicion, the vets quickly concluded that this had not been boiled as the law required. The farmers admitted nothing but this is almost certainly how the pigs caught the disease.

Working 'downstream', the vets tried to trace the possible lines of infection leading from the farm to other premises. They knew of the abattoir, but they also quickly discovered 'airborne spread of disease from Burnside Farm to sheep on nearby premises' (Department for Environment Food and Rural Affairs 2002, p. 3). Here they did similar tracing work and found that the chain of infection went on: 16 sheep had been sold from this farm, and (this was the really serious discovery)

> entered the marketing chain and were sold via Hexham and Longtown markets and through dealers where they infected other sheep, people or vehicles thereby spreading FMD virus widely in England andWales and the bordering counties of southern Scotland. (Department for Environment Food and Rural Affairs 2002, p. 3).

This is where the detective work led. It was contacts between animals (24,500 passed through Longtown market in the period in question [House of Commons 2002, 1587, 51]), together with the way in which they had been subsequently moved and mixed with other animals that made the disease spread widely through the relevant animal populations. This all but nationwide set of animal movements implied an all but nationwide spread of the disease. In 2001 animals on over 2000 premises were eventually to contract foot and mouth disease, an impressive epizootic that was the

object of forensic epidemiology. An epidemiology that enacted foot and mouth disease as a *traceable condition that spreads from location to location through susceptible populations*.

Different worlds

The clinic, the laboratory and epidemiology relate to different objects. They all talk about foot and mouth disease but as they do so, the clinic is on the lookout for *deviances* in animals, the lab seeks to detect *virus* in test samples while epidemiology traces *infections* spreading through populations. However, it would be hard to grasp the differences between these ways of objectifying 'the' disease from a simple reading of a textbook. Take the following textbook definition: 'FMD is a highly infectious animal disease, caused by a virus. Its symptoms include lameness and lesions (blisters) on hooves and in or around the mouth' (House of Commons 2002, p. 40).

These short sentences, taken from one of the UK government reports on the epizootic, draw together symptoms, virus and infectiousness as if they were *aspects* of 'foot and mouth disease' seen from one perspective or another. But they do not accord them the same status. It is the virus that is granted the central role, the agency: it is said to *cause* the disease. The symptoms follow from it. That the disease is 'infectious' suggests further activity on the part of a causal virus: it moves from one animal to the other. Thus the textbook stages the object of the lab – the virus – as something that *precedes* clinical signs and achieves epidemiological transmission. This way of narrating the reality of the disease sidelines other possible versions; for instance that of practice. In veterinary practice the lab does not come first. Instead the clinic, that is, the vets who detect deviance as they examine living animals, necessarily *precedes* the work of the lab. Without clinical suspicion the lab is not put to work and without samples provided by clinicians the lab is unable to diagnose anything at all. And when it comes to it, the epidemiological reality of an epizootic is not an achievement of the virus either. Patterns of transmission depend on endless other variables. A virus is one of these, but so are the other factors that epidemiologists investigate; direct contacts between animals, the routes livestock is made to travel, the way the wind is blowing and so on.

Thus, granting the virus the central role in the disease drama is not stating a matter of fact, but staging a specific version of reality at the expense of its alternatives. This is not exceptional. Some version of reality tends to achieve priority over its alternatives in most sites and situations. It is impossible in practice to keep all the versions open all the time. But which version of reality deserves to be foregrounded and worked with? And under which circumstances? What is gained and what is lost if textbooks attribute all the action to the virus? What is gained and what is lost if ministries work with one kind of epidemiology or another? (See Law (2008).) As a part of asking such questions, it is good to know what is at stake. So far we have described how clinic, lab and epidemiology each enact their own specific version of foot and mouth disease. Now we add a further exploration of the *worlds* they draw on and evoke, and to which they contribute. This allows us to show that clinic, lab and epidemiology work with different *materials*, attribute different *qualities* to the entities relevant to their worlds, work in ways that stage *time* differently and engage in different *spatial relations*. Let us explore these issues one at a time.

Materials

Each of the practices is materially heterogeneous. Nevertheless, the materials most relevant to the clinic are bodies: animal *bodies* with or without signs of disease and human bodies with the skill to recognise the relevant signs. Clinical diagnosis depends on the *proximity* between these bodies. Indeed, the arrangements at Cheale's were precisely configured to secure that proximity. More generally, UK law requires that slaughter be supervised by a qualified vet who needs to get close to every animal to look for clinical signs of disease. The possibility of making a diagnosis from a photo may suggest that bodily proximity is not necessary. And indeed, photos, although they lack sound and smell, may sometimes show clinical signs sufficiently clearly for a good clinician to make a diagnosis from a distance. However, the person who took the photos was necessarily close to the animals and moreover knew what to look for, what to depict. The nearby photographer stood in for the far away clinician.

Large living animals with foot and mouth disease never enter the laboratory. They do not need to come near it. Instead, laboratories work with small test samples that clinicians take from sick animals with needles and knives. While such *specimens* are crucial materials in the laboratory, what comes into focus first are the technologies used to process them: multiwell plates, rotary shakers, temperature control systems, thermometers and photometric measuring devices. Reagents and other laboratory supplies are important as well. These are partly made by small animals: laboratory rabbits and guinea pigs produce the antisera on which laboratory diagnosis depends. A supply of foot and mouth virus with a known serotype, one that was established earlier, is necessary to infect these small animals. These materials do not need to be produced in every single lab. They may be bought in from elsewhere, sometimes in kit form. Skilled technicians are indispensible even so. Their individual idiosyncrasies, however, are not important. Indeed, so far as possible, they are excluded. Ideally, technicians are as meticulous and standardised as their machines.

In epidemiology it is records that are crucial. The forensic epidemiology of 2001 drew on data from the slaughterhouse, Burnside Farm and the markets where possibly infected sheep were bought and sold. The variously crafted records that these institutions had produced contained traces of the movement of animals and their feed. Traces of other movements and routes were relevant too: meteorology gave wind directions and geography showed the locations of roads. So records were central –though tracking traces and turning them into epidemiological insights again depended on a range of additional technologies – computers, statistical interferences, cartography. And human skill.

Qualities

Not only do the materials crucial to the three knowledge traditions differ, but so, too, do their most relevant *qualities* (see Thévenot 2001). In the clinic animals are large and the signs of the disease are *large* enough for human beings to see, hear and smell. At the same time, the quality of a deviance that has most clinical relevance is not its size but the level of trouble or distress that it implies. A deviance may disturb an animal

just a little or rather a lot: its symptoms may be *mild or severe*. These gradients may be important to the animal but they are not relevant to the question of whether or not the animal 'has' foot or mouth disease. Thus, they are not relevant when it comes to passing on diagnostic facts to epidemiologists. But they are relevant to how difficult or easy it is to diagnose. Mild symptoms create a grey zone, a zone of insecurity where clinicians cannot be sure. Asking colleagues may help but with mild symptoms they may not be sure either. Clinically, then, the disease hits animals in different *degrees*.

In the laboratory the most important entities – viruses, antibodies and antigens – are tiny. Indeed, without laboratory technologies they are invisible. The laboratory thus enacts *sub-microscopic entities* and makes these available to human perception.[5] Those entities are *definite* in form, at least in the present case where the ELISA test produces a binary answer. Yes, virus is present in the sample or no, it isn't. Grey zones or degrees of severity are absent. Insecurity gets controlled by repetition. An indeterminate result is a reason to re-run the test: 'Values close to 0.1 should be confirmed by retesting or by amplification of antigen by tissue culture passage and testing the supernatant once a cytopathic effect (CPE) has developed' (Kitching et al. 2000). An unexpected result may also be a reason to run a second confirmatory test. [6] Hesitation and ambivalence are not in order. ELISA enacts sub-microscopic entities that, absent or present, are discrete and definite.

Epidemiological practice is different yet again. From the records that it uses epidemiology has to reconstruct the entities it seeks to know: its *pathways of infection*. These are neither ambivalent nor definite but take the form of *likelihoods*. In predictive epidemiological modelling (which we have not explored here) these take the form of quantified statistical probabilities. In the case at hand the epidemiologists concluded that the most likely pathway for the initial infection in the UK was catering waste that had contained virus coming from overseas. Such a pathway is neither big nor small, neither severe nor mild. Instead, to say that it is 'likely' is an indication of the strength of the possible links between the most recently infected sub-population of susceptible animals and a sub-population affected by the disease at an earlier time. In the case at hand, the contact between these populations was digestive. The carriers and their victims did not meet face to face but the pigs who were the first animals to be infected in the UK were given meat to eat that had been illegally imported from a country where foot and mouth disease is endemic, and that, accordingly, could not lay claims on the status 'disease free'.

Time lines

Clinic, laboratory and epidemiology also situate themselves differently in time, or rather they enact 'time' in different ways. The clinic, to start with this again, is marked by *shifts*. In February 2001 in no more than 36 hours the animals shifted from being noisy sows into sows with blisters, then becoming sows with a notifiable disease before ending up (with feedback from the lab) as sows with foot and mouth disease. Along the way the art was not to be sure but to be careful. As a part of this, different possibilities had to be taken simultaneously into account: the burden for the slaughterhouse of stopping what everyone was doing because disease might be present, the burden on the animals if they were to have the disease, the burden on the animal

population if the disease were to spread, the burden on the economy if diseased animals were not to be culled. In clinical work various uncertainties have to be entertained at the same time. And since so many variables are variable, the clinic works not by fixing reality but in a chronic process of *tinkering* or (if the term can be stripped of its pejorative connotations) of *doctoring*. (This is developed in Mol 2008; see also Law 2010). Thus, clinical time is characterised by shifts, simultaneities and ongoing adaptations.

In the laboratory time is different. The ELISA test, more particularly, moves towards closure. At the beginning its samples are underdetermined. Ambiguity intolerant, the lab characterises reality by *fixing* it. This implies a chronology with a beginning (uncertainty), a middle (the process of testing itself) and an end (a secure result in which reality is stabilised). The only way to change the definite conclusion is to start again from the beginning. If this leads to a contradictory result then a third test may be needed to indicate which of the two earlier tests was wrong. There must be an end and the end comes with a definite *conclusion*.

Epidemiology *traces* its objects. It searches for clues and maps the links thrown up by those clues, attributing different degrees of likelihoods to them. The logic is that of a whodunit that works by *elimination*. The most likely links are traced first, like the farms feeding catering waste to their pigs. Most were eliminated, but not Burnside Farm. Then the process of tracing and eliminating started again: where did the disease on Burnside Farm come from? Was it indeed catering waste? Various other possible sources of infection were ruled out. As it appeared that the catering waste at Burnside was not boiled, this became the prime suspect.[7] And then the search went downstream to where the infection had spread. The detective work of forensic epidemiology thus proceeds iteratively, continuously remaking the grounds and conditions of its own investigations. Time goes on: there is no closure.

Spatial relations

Finally, clinic, lab and epidemiology imply different *spatial relations* (for related discussion of the spatialities of biosecurity see, for example, Enticott 2008). The clinical repertoire is not bound to a specific site. Indeed, we have been talking about the clinic without going near a hospital or a surgery. Trained vets work in slaughterhouses and on farms, so 'the clinic' goes with them. Because they embody the clinic, the law requires their presence in the slaughterhouse, farmers call them out when their animals fall sick and they are sent on forensic epidemiology missions. Interestingly, the clinic is able to spread not because it is stable but because it is malleable. It adapts to a great variety of variables. Clinical practices, then, are *fluid*:

> The clinical signs [of FMD] are more severe in cattle and intensively reared pigs than in sheep and goats, and FMD has frequently been ignored or misdiagnosed in small ruminants. (Merck 2008).

What vets look for depends on a whole range of factors: on the animals they deal with (cattle, pigs, sheep and goats), on the epidemiological context (if an infectious disease is prevalent its diagnosis is more likely); on the serotype known to be present (some generate more symptoms than others) and on the stage of the disease (is it florid or

not?). The ‘same’ condition looks, sounds and feels different depending on such specificities. The clinic is able to attune to this variety and this adaptability allows it to travel. Its *mutability* makes it mobile.

For the laboratory this is different. Laboratories may be geographically far apart but they are also meant to be *identical*. Pirbright IAH, the World Reference Laboratory for foot and mouth disease, sets the gold standard for its ELISA tests. It tells other labs which reagents to use and which protocols to follow. Solutions need to be buffered (to fix pH) and everything else needs to be likewise stabilised. Laboratories isolate themselves from contextual contingencies. Ideally, their practices are similar from one location to another: they are *immutable*.[8] Because this is a demanding achievement, it has been realised in only a few locations. Over the last 10 years considerable effort has been put into automating the ELISA procedure. If the test were made available in kit form, ‘the immutable lab’ might become mobile enough to travel beyond the walls of laboratory buildings (for an example of the large literature on this subject, see Ferris et al. 2008). This is work in progress; its success depends on the possibility of rendering the components of the lab sufficiently immune to their surroundings.

Thus, while the clinic travels by adapting itself, the lab can travel only if it manages to stay stable. Epidemiology enacts a third spatial pattern. It gathers traces from dispersed sites into a single location and in doing so, seeks to create an *overview*. (For the logic of drawing things together and of ‘obligatory points of passage’ see Latour [1990]). As a part of this, it draws cartographic maps that allow it to depict relations between sites, and statistical maps that allow it to establish likelihoods. As these maps are being drawn, epidemiology enacts space as a *flat surface*. Links are depicted, ranked, measured and accorded probabilities. Qualitative homogenisation is combined with quantitative discrimination. Reality is spread out on the two dimensions of a sheet of paper or a whiteboard, or the screen of a computer. The overview thereby created makes decisions about where to intervene possible.

Handling difference

In most instances veterinary traditions do not act alone. Instead, clinic, lab and epidemiology tend to work together. What we have described above is typical. Thus, in 2001 the clinic came first; clinical skills put to use in observing animals are indispensible to the detection of disease. The lab was then asked to give its definitive diagnostic verdict but it could not do this without the test samples sent in from the clinic. Epidemiology, in its turn, could trace patterns of infection only because it had diagnostic reports from the clinic as well as the lab in the files and records that it worked with. To the diagnosis of individual animals, it added the patterns of transmission necessary to intervene in the health of populations. But even while they collaborated, clinic, laboratory and epidemiology were also drawing on and contributing to different worlds.

Drawing upon different worlds as they do, clinic, lab and epidemiology do not know the same ‘foot and mouth disease’. Each enacts a different version. They do so by attending to and thus giving importance to different materials, fostering different qualities, staging different time lines and engaging in different spatial relations. This

tells us that the ontological realm each opens up, explores and strengthens is different. Pragmatists might want to explain this by pointing to the fact that clinic, lab and epidemiology have different goals and serve different purposes. This is right, but the problem is that goals are not given separately from the practices that make them conceivable. Take the clinic: does this have embodied and fluid ways of working because it has to be able to move in a world composed of large animal bodies, plagued by disease in different degrees? Or is it rather able to move around in these settings because it happens to be fluid, adaptable and good at detecting unstable signs? Should we be in awe of the lab's ability to make definite diagnosis or is this a simple result of the all but endless energy and money that have been invested in rendering its tools, procedures and techniques fixed, discrete and conclusive? And what about epidemiology: is this designed to attend to populations rather than to individuals or does it happen to allow for a type of veterinary practice that addresses and attends to the UK's 'livestock' as a single collective?

That clinic, lab and epidemiology enact different worlds implies that their differences are relevant to the way the world comes to be shaped. It implies the importance of the question: 'what to make of the world?' This is an ontological question. What it implies is the possibility of, and the need for, an ontological politics. This is not a politics that works to establish goals, leaving questions of means for subsequent implementation by experts and technicians. Instead, in an ontological politics technical questions are at stake from the beginning. What does it mean for a 'fact' to be 'established'? What is it to 'act' or to 'implement'? In its handling of many simultaneous questions, ontological politics resembles the clinic. In the clinic, too, there is always an endless number of issues at stake more or less simultaneously, all of them open to adaptation and tinkering. By contrast, in the linear timeline of the laboratory, an activity, like a diagnosis, tends to move in a sequential way from an insecure beginning to a definite conclusion, while the iterative time line of epidemiology keeps on shifting its own conditions of possibility. It implies a notion of action marked by an acute sensitivity to the new situation that has just been established. In complex situations such as those of a widespread infectious disease epidemic the definite character of lab actions might seem to offer safety and security. However, it is good to remember that the clinic comes with a repertoire marked by adaptability. It is able to attune to specific local needs and circumstances. Does this call for praise? Or should it just be respected, attended to and carefully developed?

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[1] This is in line with ways of working in science and technology studies. See, for instance, Latour and Woolgar (1986), Hacking (1992) and Mol (2002).

[2] The rules are complicated, but in the UK the loss of disease free status effectively prevented exports.

[3] The term 'forensic epidemiology' is conventionally applied to epidemiological investigations that have legal import. Though the epidemiological evidence was used in a criminal prosecution after the 2001 foot and mouth outbreak, we are using the term more generally to explore a logic of tracing based on what we call definite likelihoods. There are other epidemiological genres as well. Some, for instance, generate models that predict how the disease will further spread through populations. For an account and further references see Law (2008).

[4] The text goes on to tell us that the virus recovered from all these pigs was of the same serotype. This claim involves a move back to the laboratory where tissues from the pigs were tested not just for the presence of a virus but also for serotype (seven of which are currently known).

[5] Famously, Latour (1983) describes the laboratory as a device for reworking scale relations.

[6] So much was at stake that MAFF asked for this after the initial result was phoned through to London on 20 February 2001 (House of Commons 2002, pp. 54–56).

[7] The circumstantial evidence included thousands of pieces of cutlery. ‘Over and above cutlery in the pens, circa 1,300 pieces were retrieved from the bottom of the first Burnside swill holding tank (i.e. the input point into the automated feeding system of the supposedly processed swill). As noted above, if only processed swill had gone into this tank, the number of pieces of cutlery retrieved from its bottom should have been nil’ (Dring 2001, 16).

[8] For discussion of immutability see Latour (1983). For an account of how the laboratory and clinic travel see Mol and Law (1994), and Law and Mol (2001).