Latency and the control of bovine TB in man and other animals

A great deal of confusion exists over the measures needed to control the rise of bovine tuberculosis in cattle in Britain and Ireland. Indeed it is still claimed that since transmission is not understood, it is not possible to initiate controls via husbandry risks based on robust science.1–3 Comparison with TB schemes in man are valuable, and one of the key factors being overlooked would seem to be the long incubation period of TB, which dictates the measures available as well as the need for a targeted approach to high-risk groups for the most cost-effective results. This is well documented for human TB in the USA with full recognition that cases may not become skin test reactors until after 2–12 weeks after infection.4 In fact, whilst some 30% of those closely exposed to an 'open' case may become infected, only about 5% of these develop clinical disease in 2–3 years, a further 5% may contain the disease in healed tubercles but can reactivate via progressive TB up to 50 years later.5,6 Reactivation of bovine TB in man can also occur decades later and cause breakdowns in cattle herds TB-free for years.7 Recent studies of cattle TB schemes seem to have overlooked the critical importance of this latency.8,9 Four classic textbooks however clearly note that depending mainly on challenge dose a new case may have six outcomes: fail to become infected, or become infected and then heal fully, remain latent for years, progress slightly with remissions or exacerbations as a chronic TB, progress fairly rapidly (the "normal" situation), or become acute and fatal within months.10–13 Usually bovine TB is a slow but progressive bronchopneumonia which means that if unchecked, it spreads slowly but inexorably in the individual, then through the population, and this spread is respiratory just as with other cattle "pneumonias" whether viral, bacterial (Pasteurella) or mycoplasmal (the pleuropneumonias). And the slow progression determines the two checks needed to control TB: annual testing of all cattle, and movement bans into TB-free areas.1,2 The reason why annual testing is the gold standard worldwide and under EC Directives seems to be poorly understood, so it is worth examining latency in cattle.

Basically one might recognise three stage in TB progression with an early lung microscopic or non-visible lesion/s (NVL) stage with few bacilli shed intermittently; then a stage with one or more lesions visible at gross abattoir inspection (VL) and continuous shedding of increasingly large numbers of bacilli, particularly as in man when lung cavitation occurs; lastly, spread to other organs which may shed externally such as kidney, udder and uterus. On "average" it takes about 2 months to become a reactor to the skin test (8–65 days),8 then about a year to reach the more infectious multi-VL stage (12–15 months).14–16 However, a study of a group of calves infected in 1987 showed 1 reactor in 1987, in 1989: 17 in April; 2 June, 1 August, then 1 each in 1990–1992. So, 24 reactors, 14 VL, over six years showing the danger of leaving early cases in the herd, and that transition from negative to inconclusive to test-positive may take some years.17 Another study suggests that around 9% may be VL infectious by six months, 17% by 12 months, 26% by 18 months, and 34% by 24 months.18 And in fact results from chronic depopulated herds showed that the skin test missed 9.1% of VL cases in 36% of the herds even with up to 6 tests a year, including some cases with generalised lesions which as "superexcretor" non-reactors are a potent risk.19 Even a brief exposure to such cases at a show or auction can result in importing TB into new herds, as with taking TB to Guernsey.13 The depopulation study did not look at NVL early latent cases, but in GB in the 1980s some 70% of NVL "unconfirmed" cases were probably latent carriers.20

And so, the skin test is only about 80% accurate, or 68% on retests, and annual testing is the optimal screening measure to pick up cases as they come "on stream" and before they become the more infectious VL excretors. Retests may pick up most cases within a year or so within the index herd and in contiguous herds, as well as minimise the risk that any sold on stock will be carriers. The bigger the herd the greater the risk of missed cases, in a
1000 strong dairy herd 1–2 may occur in one year, 10–15 in another and the herd becomes chronically infected. This also happened with the Dorset cluster of farms which supposedly proved that badger culls work. At the low point in 1979 with only 89 herds and 600 cases, over half the breakdowns involved a single reactor, so that with the 80% accuracy 2 in 10 singleton herds were missed each time. But it also appeared as if contiguous spread was very limited and blamed on badgers instead. Annual testing hence gets most reactors below the more infectious “critical mass” of VL lesions stage, as well as avoiding non-pulmonary routes of spread congenitally or in milk (stage 3 above). And clearly only a movement ban into TB-free areas will guarantee that TB does not spread via latent “missed” cases.

The tragedy of the present situation is that warnings that foot and mouth would exacerbate the TB crisis were ignored. Cattle TB is back to 1960s levels as predicted since in effect both annual testing and movement bans were abandoned for the last 2 years. Provisional figures for the last year compared to 2000 (pre-FMD) suggest 4047 herds under restriction versus 2511; 1666 confirmed new herds versus 1044, and over 23,000 reactors versus under 9000. And despite the warnings about restocking after FMD, TB has now appeared in areas TB-free for up to 50 years, including Wales: Powys; Scotland: Dumfries, Ayr, Banf (?); England: Cumbria, Northumberland, Durham, Yorks, Leics, Sussex, Hants. It will require one or two full national herd tests to establish where TB has got to, and with a backlog of some 8000 herd tests outstanding it needs several years of annual tests in hotspots and some staggered annual testing in supposedly 2–4 year test areas to arrest the spread, let alone begin to reduce levels.

Sadly both veterinary and farming organisations are still calling for mass badger culls, even though TB has clearly spread amongst cattle way beyond the supposedly important “southwest high density badger TB” 2000 km² hotspots. A study of badger visitation of barns has been hailed as the answer to how badgers are supposedly able to infect cattle, but it overlooked an earlier study showing just how difficult such transmission is even when badgers and calves cohabit long term. In fact badgers are no different to cattle or man. A human super-excretor may shed 4000 million bacilli/day, a cow some 38 million/day so schemes try to catch cases early. In the 300 strong badgers of the 9 km² Woodchester area over 14 years there were only 188 with TB, 41 infectious, 17 superinfectious (58=31%). And so, extrapolating these figures to the Krebs/Bourne trial an eventual cull of 12,500 badgers will yield 2500 with TB, 750 infectious, over some 9 years, in relation to c. 3000 farms in the 2000 km² cull areas, i.e. one infectious badger/13 km² or one per 20 herds. No badger culling, fertility control, or vaccination strategy will ever be practical, cost-effective or meaningful. Scraping the Bourne cull now would release some £30–35 million infinitely better spent on catching up on urgent cattle testing.

References


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