Overview

Cattle TB schemes: control or eradication—a critical reappraisal

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Introduction

Both human and bovine tuberculosis is on the increase worldwide (Mycobacterium tuberculosis, and M. bovis or M. africanum). In particular, bovine TB is rising rapidly in incidence in both Britain and Ireland, and yet paradoxically, it is seriously questioned as to whether cattle-to-cattle transmission is important (1–3).

The answer to this curious conundrum is complicated by the ‘highly complex and emotive issue’ of badgers (Meles meles or possums Trichosurus vulpecula) as part of the ‘undisclosed’ source of cattle TB (1). Many of the answers lie in somewhat forgotten classic studies reviewed comprehensively by Francis (4), and are usefully complemented by contrasting human and cattle TB problems.

The two factors which determine the spread of TB or other infectious diseases are exposure (particularly challenge dose) and susceptibility (particularly how immunocompetent the host is to overcome infection). Ironically, the Industrial Revolution facilitated the spread worldwide of both forms of TB. Poor housing and nutrition helped human TB to peak in Europe around 1800, and it was exported by colonization thereafter. Similarly, cattle TB may have become significant in the first domestication in Asia and the near East, then increasing in Europe, but it was the Industrial Revolution with the need to feed increasingly urban populations which led to intensive town milksheds, whilst new crop rotations meant more than just the breeding nucleus of herds could be overwintered indoors, also facilitating spread of bovine TB. Export of shorthorn, then fiesians and channel island breeds, took bovine TB worldwide in the 19th century.

Both cattle and humans can have avian, bovine and human TB. But in man, only the latter tends to be progressive. Bovine TB in man can be progressive enough to be a significant source (pulmonary or renal) of TB in cattle, but it is seemingly only anecdotally recorded as spreading from human to human. With HIV however, both avian and bovine TB may become progressive and self-maintaining diseases within the human population (2,4). Similarly in cattle, human and avian TB disease tends to be non-progressive although avian TB may cause abortion, and both can cause a false-positive reaction to the skin test (4). Whilst there may be some 2 billion humans infected with TB, only 10 million have progressive disease with perhaps 3 million deaths a year, according to World Health Organization (WHO) data. The real problem—complicated by HIV and multi-drug resistance—is to find ‘sputum-smear positive’ cases early enough, and to help the immune system regress TB below this infectious stage by vaccination or chemotherapy. Similarly, in cattle bovine TB is the equivalent of human respiratory ‘consumption or phthisis’—many may be infected but few are infectious—and the whole point of control schemes is to remove ‘sputum positive’ cases early enough, by test and slaughter of reactors.

Becoming infected/infectious

Classic studies reviewed by Francis (4), suggested that the development of ‘tubercle’ lesions was the key to understanding transmission routes, and how TB spreads within the cattle population ‘reservoir’ of the disease. Primary complex lesions suggest some 90% of transmission is by inhalation, 10% by ingestion and under 1% is congenital. A low challenge dose might result in only incipient lesions (granuloma), and be latent or only slightly progressive for months or years. A high dose could produce multiple lesions, acute TB and death within a month. Unlike in man however, tubercles usually fail to become fully encapsulated, but grow by peripheral proliferation (tuber like), whilst haematogenous spread yields further lesions in the lungs and other organs (4). Thus, a natural low dose of infection may result in infection subclinically (4, 5–8). A classic study showed that when cattle have been on annual testing for some years, some 40% of ‘reactors’ do not have lesions because they do not have bovine TB but carry avian or human strains (false positive) (1, 4). Amongst the true bovine TB cases nearly three quarters had lung lesions, but over half of these were single lesions under 1 cm in diameter (9). Only some 20% of these were sputum positive, i.e. significantly infectious to other cattle (9). This is why cattle are ‘apparently’ not important in passing TB to other cattle (1–3). These early lesion cases may only have been infected for some 11 months (range 5–15) which is why annual testing is the gold standard worldwide, removing most but not all reactors before they get to the highly infectious gross lesion stages (4, 9). It is unsurprising that in Britain the worst TB parishes, i.e. those on annual testing, are more likely to have repeat breakdowns, and at least 70% of reactors may be subclinical latent TB carriers (1,10).
Cattle TB: control or eradication

A review of cattle TB schemes in over 100 countries worldwide suggests that there are two phases. If left unchecked, TB develops in the individual from microscopic or non-visible lesion (NVL) to gross macroscopic visible lesion (VL) to clinical TB with emaciation. Hence such cases increase progressively in the national herd reservoir of TB. In effect, in phase one, this progression is reversed. Annual testing of all cattle eliminates the clinical and VL cases and a ban of stock movement into TB free areas prevents spread (4,11–14). Britain had a textbook demonstration of this process up until the 1970s (4, 15–18). It is fairly easy to get from very high TB levels (90% of herds, some with 100% reactor rate) to around 1% of herds infected, within 10 years.

In phase two however, with TB at such low levels, the same error has been repeated worldwide. As actual TB cases fall, the proportion of false-positive reactors rises. Previously ‘masked’ by the true bovine TB positive, these can be a reaction to human TB as in Finland, or to skin TB, Johne disease (M. paratuberculosis), other infections (brucellosis, liver fluke), or even pregnancy (4,10,13,16,17). From a true reactor rate of 98% in the U.S.A., by the 1940s false positive or ‘unconfirmed’ or non-specific reactors reached 41%, particularly where they were the sole reactor in the ‘singleton’ herd (13). Exactly as in other countries where singleton herds predominate after some years of intensive testing (1). These NVL false positives can rise to 80% or more of reactors, and many countries hence relax the annual test/movement bans prematurely. But this merely allows TB to escape from containment as happened in the 1970s in Ireland and Britain now (11). Longer herd test intervals simply allows TB to build up in herds, such that under EC rules on up to 4 yearly testing massive breakdowns pose a real risk to other stock, wildlife, and the farmers themselves e.g. from raw milk (4,13,20). The problem is that current tests are a compromise between picking up all TB cases (sensitivity) and picking up too many non-TB cases (specificity) (1,19).

Since a minimum dose of 400 000 bacilli may be required even by inhalation (3) and badger sputum only contains 200 000/cc, badgers are an unlikely source of cattle TB herd breakdowns (21,22).

References