

VIEWPOINT

The cattle TB crisis – a hidden problem?

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Amidst the current cattle tuberculosis (TB) crisis, there is seemingly one pivotal misunderstanding that is responsible for prolonged chronic herd TB breakdowns, this is the inadequacy of current tests in use.

In the current cattle TB crisis, attention has focused so much on badgers as the supposed main reservoir of TB that some basic facts about how eradication schemes work have been forgotten: the two key elements being annual testing plus movement restrictions (Hancox 2006). It has long been known that early and late cattle TB cases are the *usual* cause of recrudescence in herds supposedly tested as clear of the disease. A study some 20 years ago claimed that some 0·30 of cows go temporarily anergic or non-reactor after parturition. Pregnancy certainly modifies the immune response, allowing a proliferation of lesions, followed by their regression post-partum. Young heifers may carry latent TB until their first pregnancy activates the disease (Francis 1947). And so seemingly, a significant minority become permanently anergic yet active TB spreaders (Blood 1989). In fact three such anergic cases caused some 18-herd breakdowns in one parish in the West Penwith or Lands End area of Cornwall (Richards 1972). After selective herd depopulation, this area apparently went clear of TB briefly in the early 1980s without any significant badger culling, which had been focused more in areas where the 'first' TB badger had been found (Gloucestershire in 1971). Local badgers with TB did NOT immediately re-infect the cattle, but local cattle restocking reintroduced TB (Richards 1972; MAFF 1977). Great Britain could have eradicated TB then if the approach had been persevered with.

TB eradication was difficult enough in the 1980s when most UK herds were 50 cows or fewer. Now the average UK herd size is often nearer 150 with accompanying 100 replacement young stock (Defra 2007*a*) and with some herds 250, 500 or 1000 strong. Obviously a test with 0·80 (0·77–0·95) sensitivity will miss some positives every time (Defra 2007*b*). Many

big dairy herds under restriction since the explosion of cattle TB caused by the lack of testing due to foot and mouth disease (FMD) in 2001 will have anergic cases, so tests repeated at short intervals merely remove the latest new cases. It is perhaps not surprising that there are few studies identifying anergic active spreader cases since these must entail detailed autopsy of depopulated herds (Costello *et al.* 1997).

Clearly with such big herds, herd depopulation is a drastic measure. However, it ought to be possible to try and validate two 'new' tests. Gamma interferon will not find late TB cases, but an antibody test would. Ironically, the badger BROCK (TB) Stat-pak (Veterinary Laboratories Agency 2008) would target these late cases as would the similar anamnestic ELISA tried on depopulated herds (Yearsley 1998). Cows can shed 38 million bacilli/day in faeces and so another simple procedure would be to identify TB bacilli using DNA/PCR tests on faecal swabs. PCR will revolutionize rapid confirmation of TB in tissue samples. It is being trialled for persistently infected (PI) bovine viral diarrhoea (BVD) and also for TB (Thomas-Everard 2006; Defra 2007*b*).

Incidentally, the end result of the £50 million 'Krebs' trial involving culling of *c.* 11 000 badgers identified only 1500 with TB and only 166 with advanced lesions. Although these individual badgers might have been an infection risk to cattle, no one has realistically shown how badgers might give cows a respiratory lung disease! It is astonishing that many commentators still claim that TB transmission in cattle is not understood – it was crystal clear half a century ago that some 0·90 of cattle TB is a respiratory 'consumption' as in man, a bronchopneumonia acquired during over-wintering in barns just like other 'pneumonias' be they viral, bacterial or mycoplasmal (Defra 2007*a*). Some 0·10 is via ingested food or water e.g. in slurry runoff in ponds or slow moving streams that may take TB into 'closed' herds downstream, again as in man, a dietary 'scrofula' (Blood 1989; Francis 1947; Hancox 2006). Because of FMD the incidence of cattle TB doubled and so there was twice the spillover to badgers (Bourne *et al.* 2007).

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Badger TB often presents as a dietary ‘scrofula’ with swollen throat, lymph nodes, just as in pig/wild boar (Hancox 1995, 2002, 2006). Thus badgers are innocent victims not villains (Clark 2007).

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