

physicians are inclined to recommend its use in his fashion. In describing the methodology of our study, we specifically state that 'the experimental conditions were intended to reflect the clinical circumstances under which patients take supplemental oxygen, accepting that an absolutely constant  $FI_{O_2}$  could not be guaranteed'. Our conclusions are therefore practical and based on how we believe oxygen is commonly used.

We agreed that it would be interesting to test the effect of an assured, constant  $FI_{O_2}$  on dyspnea recovery time avoiding any increase in respiratory muscle work. Such a study would require a high flow or reservoir oxygen delivery system with application of positive pressure such as CPAP and the likelihood of such systems gaining approval for clinical practice is remote. Even if meticulous attention to these factors revealed a positive effect of supplemental oxygen on post-exercise dyspnea, we believe that such an effect is likely to be small. Of far greater consequence, as shown in our study, is the effect of prior exercise, and presumably pre-existing metabolic factors such as lactic acidosis or respiratory muscle fatigue, in determining the duration of dyspnea recovery. Patients whose breathlessness scores had returned to baseline between 20 and 30 min after exhausting walking exercise were unable to exercise again for the same duration, indicating that although they felt they had recovered symptomatically, physiological recovery was incomplete.

J. A. MARQUES-MAGALLANES, T. W. STORER AND  
C. B. COOPER

*Departments of Medicine and Physiology,  
UCLA School of Medicine, U.S.A.*

Dear Editor

### **Of Bourne, badgers, and a bovine 'TB alert'**

In response to the global upswing of tuberculosis, a new initiative 'TB Alert' was launched by two Liverpool doctors, P. Davies and B. Squire, on 5 June at the Royal Society of Tropical Medicine & Hygiene, London. Mainly focused on human TB, attention was also drawn to the worsening bovine TB epidemic in British cattle, already increased by 45% over last year, and my warning of a risk to man, particularly via unpasteurized bulked milk (1,2), and of the value of cross-fertilization of ideas. The new Bourne badger/cattle initiative launched on 17 August (3) belatedly recognizes this risk, whilst a further possible link with another *Mycobacterium* (*M. paratuberculosis*), which is not invariably killed by pasteurization, is noted as a cause of Johne's scouring disease in cattle and perhaps Crohn's bowel disease in man (4). Ubiquitous in cattle, sheep, goats, pigs and horses, this may be one cause of false-positive 'TB reactors' in cattle (5). In fact, there is an exact parallel between the difficulty in diagnosing and treating drug-resistant TB in man early enough to prevent spread and the

inadequacies of current cattle test/trace procedures: the skin test is only approximately 80% sensitive, thus missing perhaps one in five TB carriers, whilst in late herd eradication situations up to 80% of reactors may be later found to have TB false-positive results (due to *M. avium*, *M. paratuberculosis*, etc.).

Progress in science is most rapid where often very simple 'right questions' yield 'right answers', and is greatly hampered by asking the 'wrong questions' or by what Charles Darwin described as 'false facts' (1). Koch discovered the tubercle bacillus in 1882, but then did great damage by claiming that bovine TB was of little relevance to man. It took a Royal Commission a decade or so to re-establish the risk of unpasteurized milk in 'scrofulous' children particularly, and it is ironic that pasteurization is still not mandatory in law in the U.K. (apart from in Scotland).

Sadly, a very quick and simple resolution to 'The Great Badgers and Bovine TB Debate' is being overlooked as politically incorrect (2), and instead the Bourne/Krebs 'scientific' badger cull (3,6) is unlikely to resolve matters since it raises the wrong questions, will be costly, impractical, and the statistics 'won't be able to cope with so many bodge factors' (7). This is political expediency pursued to the point of absurdity', as an ex-Ministry vet said over a decade ago, whilst the last independent scientific review then found what Bourne will 're-discover' after 5 years; that badger culls are simply a waste of money because they do not work (8,9). The debate arising from this initiative has at least highlighted the simple flaws in the Krebs/Bourne review. It is worth noting where the 191-page Krebs Report is weak (6): it does recognize that cattle are infectious at any stage of the disease; at the early microscopic or non-visible lesion (NVL) or late gross visible lesion (VL) stage which could be reached within a month (pages 100, 104) (1,2, 10,11). However, despite noting very briefly that spill-over from cattle to badgers and other wildlife might occur (pages 24, 152-155), it fails to appreciate the implications of this pivotal flaw in the protocol for deciding 'Attribution of source' of herd breakdowns (pages 59, 90, 163), as well as the flaw in claims that badger culls 'work' (page 30). The claim that badger TB is of respiratory aetiology is repeated (page 45), even though it often starts as a cervical lymphadenopathy of the submandibulars, i.e. dietary 'scrofula' from eating invertebrates from under cow pats, or as in the 1950s in Swiss badgers, feeding on roe deer carrion (1). Lastly, Krebs noted, but did not explain, the dramatic rise of TB in new Midlands and Welsh herds, and was not made aware of the rise in incidence of cattle TB, even though such data can be found in the Ministry of Agriculture, Fisheries and Food (MAFF) annual Badger and Animal Health Reports ('data unavailable', pages 13, 56, 141, 156).

It will be necessary to 're-discover' the basics of cattle TB before any progress can be made towards eradication, with cattle TB as bad today as it was in the early 1960s, before badgers became 'implicated' (page 141). The classic studies note that cattle are infectious whether NVL or VL, so TB can spread inexorably within and between herds, forming clusters of herds or TB 'pockets'. The test/slaughter policy brought the incidence of cattle TB down from Midlands

blackspots areas (Irish imports?) in the 1940s (10), to tiny south-west pockets. However, relaxation of the programme as a spin-off to the BSE epidemic, including greater stock movement and cost-cutting by initiating longer herd-test intervals, has simply allowed TB to spread into new Midlands areas where it has been absent from both cattle and badgers for decades. This is exactly what might have been predicted with hindsight: the spread from south-west dairy- and calf-producing areas to non-south-west net importers of stock areas, including TB carriers which have been mistested or are untraced, and, contrary to MAFF claims with spill-over to badgers (75–100% TB plus on index or epicentre farm of cluster). Classic work in Ulster showed, thanks to fully computerized cattle tracing, that up to 70% of new breakdowns were due to contiguous spread, and up to 30% to bought-in stock, i.e. 100% of cattle with modest spillover to badgers (12, 13). Few studies in England have attempted to show how difficult tracing stock is without such computerization, which is being belatedly introduced in response to the BSE export ban and which, apparently with astonishing blindness, will not include TB test history (14,15).

Sadly, the Bourne badger cull is based on the official MAFF view that only VL cattle are infectious, and so the whole perspective is based on how and to what extent badgers *might* infect cattle (16,17), rather than the reverse. Ironically it was noted that 'it is possible to postulate an efficient mechanism for cattle to infect badgers in the past. . . . The mechanism by which badgers infect cattle may be much less efficient', and yet the whole badger culling and research programme after 27 years is based merely on the assumption that transmission is one-way only: badger to cattle (16,17).

Given the honesty and political will, it would be feasible to confirm within weeks, from data already on MAFF computers, why this NVL/VL misunderstanding is pivotal.

1. *Cattle to badger spill-over*. This is not supposed to happen, but the new Midlands clusters clearly show most spill-over to badgers on epicentre farms, with many VL cattle; few or no TB badgers are found on outer farms, with a few NVL cattle; and a clean ring is found outside with no TB badgers or cattle. This gives a direct correlation graph of severity of cattle TB versus yield of badgers with TB from the same farms (which, notably, is the opposite of Krebs' graph predicting background badger TB supposedly underlying TB in cattle herds page 63).
2. *Contiguous/closed/ongoing herds and passive vectors*. These clusters clearly show spread amongst herds but MAFF only recognize one to two contiguous cases a year if a VL case has nose to nose 'kissing' contact (6: page 163, 13). The badger zone of influence, however, is supposed to extend 2–3 km (page 163). Passive spread (including into closed herds) may be as wind/water/vehicle slurry, via starlings, rats or hire bulls (though the latter two species can also catch TB, 18). The arbitrary gulf between ongoing and new herds after being TB-free for 15 months is also flawed as test-'negative' carriers can remain undetected for years (15,19).

3. *Badger culls 'work'*. None of the 'proof' cases cited segregates the effect of culling badgers from removal of all TB cattle VL/and NVL (6: page 30). The Dorset case was merely a cluster of chronic TB herds (17), whereas Thornbury explicitly discounts ongoing, contiguous or NVL factors although noting at least 70% of NVL cases were exposed to *M. bovis* (5). Cattle have been the ideal maintenance host of bovine TB all along.

M. HANCOX  
17 Nouncells Cross,  
Stroud, Glos. GL5 1PT, U.K.

## References

1. Hancox M. Cattle TB crisis? Human implications. *Respir Med* 1998; **92**: 990–991 and **90**: 184, 371.
2. Hancox M. Cattle TB crisis: will the Krebs cull work? *Sci in Parliament* 1998; **55**(3): 3.
3. Bourne J. *Towards a Sustainable Policy to Control TB in Cattle*. London: Ministry of Agriculture, Fisheries and Food, 1998.
4. Boyce N. Milk theory stirs up bowel disease experts. *New Scientist* 1998; 7 Feb: 12.
5. Clifton-Hadley R. The occurrence of *M. bovis* infection in cattle in and around an area subject to extensive badger control. *Epidemiol Infect* 1995; **114**: 179–193.
6. Krebs JR. *Bovine Tuberculosis in Cattle and Badgers*. London: Ministry of Agriculture, Fisheries and Food, 1997.
7. Pain S. Badgers on trial. *New Scientist* 1998; 22 Aug: 13, 10 Jan: 20–21.
8. Coffee DJ. Badgers 'I told you so'. *Vet Pract* 1985; **18**: 3.
9. Dunnet GM, Jones IM, McInerney JP. *Badgers and Bovine Tuberculosis*. London: HMSO, 1986.
10. Francis J. *Bovine Tuberculosis*. London: Staples, 1947.
11. Myers JA. *Man's Greatest Victory Over Tuberculosis*. Springfield, IL: Thomas, 1940.
12. McIlroy SG. Pulmonary lesions and *M. bovis* excretion from the respiratory tract of tuberculin reacting cattle. *Vet Record* 1986; **118**: 718–721.
13. Wright R. The guilty secret of kissing cows. *New Scientist* 1985; 24 Oct: 19.
14. Hewson PI, Simpson WJ. Tuberculosis infection in cattle and badgers in an area of Staffordshire. *Vet Record* 1987; **120**: 252–256.
15. Richards RA. *Inquiry Into Bovine TB in West Cornwall*. London: Ministry of Agriculture, Fisheries and Food, 1972.
16. Cheeseman C. In: Neal E. *Badgers*. London: T & AD Poyser, 1996: 202–203.
17. Little TWA. Bovine tuberculosis in domestic and wild mammals in an area of Dorset. *J Hyg Camb* 1982; **89**: 211–224, 195–210, 225–234.
18. Hancox M. The great badgers and bovine TB debate. *Biologist* 1995; **42**: 159–161.
19. O'Connor R. *A Study of the Bovine Tuberculosis Eradication Scheme*. Dublin: ESRI, 1986.