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 \( P_{\text{O}_2} \) 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 \( P_{\text{O}_2} \) 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 70 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.

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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 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 \( M. \text{avium} \), \( M. \text{paratuberculosis} \), 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 the cull culls not are 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 VL 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 calls ‘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.

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