possible that carboxyhaemoglobinemia could account for some of our observed cases exhibiting tachypnoea, despite normal pulse oximetry measurements. This could explain some of the discrepancy between respiratory rate measurements and pulse oximetry readings, but could not account for the 115 cases of croupic hypoxia (pulse oximetry ≤90% with normal respiratory rates).

Furthermore, we tracked pulse oximetry and respiratory rates on a large cohort of non-smokers, namely our paediatric patients (4). Using age specific respiratory rates, we found that among 2127 children, 73 had pulse oximetry readings ≤90%. Only 23 (32%) exhibited tachypnoea (defined as a respiratory rate in the upper 5% by age), and only 35 (48%) had respiratory rates within the upper 20% for their age. Spearman rank correlation coefficients for each age group ranged from -0.476 to 0.799, with a weighted mean of -0.228. These numbers are very similar to the values found in adults, and suggest that respiratory drive is not particularly sensitive to the effects of cigarette smoke. Thus, while smoking may account for some of the discrepancy between respiratory rate and pulse oximetry, other factors appear to predominate.

Clearly more information is needed to assess the importance of cigarette smoking on respiratory drive. In the interim, clinicians must be aware that pulse oximetry overestimates oxygen saturation in smokers, as shown by Dr Gomez and his associates, and that the decreased saturation may not be accompanied by an increase in respiratory rate.

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Dear Editor

Cattle TB crisis? Human implications

Organizations such as the World Health Organization have been warning for some time of a global epidemic of tuberculosis (TB), both human and bovine. Britain is currently experiencing an explosion in bovine TB in the national herd, but this is being overlooked or ignored. In fact it may be attributable indirectly to the BSE (mad cow) epidemic which led to greater stock movement and cost-cutting longer TB test intervals for herds from early 1993. Hence since the start of BSE ‘officially’ in 1986, when there were only 83 TB herds, mostly in tiny pockets of south-west England, by 1996 there were 703 herds, 1537 in 1997, with a spread through most of the south west and into Midlands and Welsh areas where TB had been absent in both cattle and badgers for decades [maps in (1) and (2)]. Up to 70% of badgers with TB after these herd breakdowns suggests a spillover from cattle. And this 18-fold upswing in cattle TB bought in cattle initiating new clusters as shown by DNA fingerprint spoligotypes. In fact the proposed new scientific 5-year badger cull is likely to fail due to lack of funding, impracticalities, and its failure to understand the importance of non-visible lesions in early prepatent cattle TB carriers, e.g. in eventual contiguous cluster spread. Hence, Krebs perpetuates the myth that such NVL cattle are not infectious (1) even though early test-negative NVL cases may be TB excretors, and visible lesions can develop within a month of being infected. The Ministry admit they do not know how badgers might cause a respiratory lung infection in cattle, so the badgers’ ‘guilty’ case is in fact based on the oft repeated claim that badgers can not catch TB from cattle since only VL cases are infectious (3). The Krebs review sadly overlooks this issue of VL/VNL cattle which is the pivotal flaw in the entire TB eradication scheme (4), and also the aspects of badger aetiology and pathogenesis which suggest that they are merely catching TB from previous herd breakdowns (5,6). One TB cow may produce 38 million bacilli in 40 pounds of faeces per day (7), so that badger urine with only 300 000 bacilli ml⁻¹ pales into insignificance as a source of infection. In fact, badger culling is politically inspired, since it does not happen in Ulster which is part of the U.K., and where they have always regarded badgers as a spillover host (8); as with cats, ferrets, or indeed possums in New Zealand.

Sadly, there are issues of urgent and critical importance being ignored amidst this confusion. Astonishingly, milk is not pasteurized by law, and so whilst moves are afoot to ban ‘greenstop’ milk ostensibly in regard to Escherichia coli, Salmonella, and Campylobacter, in fact there is a very real risk that sooner or later TB will get into bulked milk. The

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TB epidemic has already breached EC Directive limitations, so that a return to 1 or 2 year testing in most of the south west will, with farmer compensation, swallow up the whole of the allocated £15 million TB budget. This has trade as well as public health implications.

Finally, it is interesting to consider the 'Great Badgers and Bovine TB Debate' in terms of the history of science, and why progress in science is often so slow. Darwin's bulldog T. H. Huxley said that 'The great tragedy of science is the slaying of a beautiful hypothesis by an ugly fact'. And Darwin himself in the Descent of Man, 1871, noted that 'False facts are highly injurious to the progression of science, for they often endure long'. Indeed, it is often necessary for an older indoctrinated generation to die off before heretical ideas assume mainstream orthodoxy. It was perfectly obvious that cattle are infectious at any stage of the disease (7), but it will be necessary to re-discover that NVL/VL cattle may be infectious and the real source of TB to both cattle and badgers in order to dispel the belief of badger guilt. It has so far proved impossible to get this view of 'false facts' into, e.g. Nature, New Scientist or Veterinary Record. And so editorial predispositions as to what is 'real science' may censor progress. Sadly such facts/false facts become enshrined in accepted wisdom as e.g. in such authoritative works as Cheeseman (3) and Black's Veterinary Dictionary.

Meanwhile, bovine TB is escalating as a bovine problem in Britain, and unless the government, and powerful farming and veterinary unions wake up soon, it may shortly become a public health and trade issue. Cattle TB has now crept back up to 1962 prevalence levels, and there were several thousand human deaths from bovine TB annually in the 1930s. Since the first TB badger in 1971 and two decades of badger culling, there has been no progress in eradicating The White Plague.

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