Crisis in arsenic news

The News item by De[1] focused on, as the title would seem to suggest, the health crisis caused by arsenic in India that the rest of the world is now attending to.

The report starts with a reference to the total ignorance of the populace who are affected by arsenicosis, about the reasons of their misfortune. It is suffice to say that this simply is not true. Thanks to the efforts of the School of Environmental Studies, Jadavpur University, the issue has been in highlight and public scrutiny, including appreciable local and international press coverage for more than a decade now. There are specialized arsenic clinics at the Medical College, Kolkata as well as in SSKM Hospital, Kolkata. The Government of West Bengal has specialized posters that are supposed to educate people about the tell-tale signs of arsenic skin lesions.

The statement De makes, I quote, 'but slowly the problem is spreading to other states like Uttar Pradesh' is grossly misrepresentative of the way arsenic contamination of groundwater occurs. From her statement it would seem that arsenic contamination and arsenicosis are spreading westwards, a grotesque representation of the dynamics of contamination. The state of Uttar Pradesh (UP) being affected was not known. Now that it is known, it does not mean that it spreads from West Bengal. De mentions that AIIMS 'confirms' that Ballia district is affected. This does point to a suggestion that there was an original report, which AIIMS only confirmed. There is no mention of that, however. In fact, the very first reports about arsenicosis in Ballia, UP, also came from the School of Environmental Studies, Jadavpur University, first at an International Conference on Arsenic held at Dhaka during February 2004 and subsequently in a paper[2] from the same group. Another report[3] followed. Though Chakraborty and his group are mentioned a number of times later, never are they mentioned in conjunction with Ballia, UP. The issue about first reportage in a scientific platform is of utmost value, as readers of this journal would appreciate.

The rest of the report by De is generously sprinkled with unscientific statements like, 'Scientists report that arsenic is natural' and the quote is not made out of context. The mandate of a journal is to help make educated conclusions about the nature of things and the News section should be treated as seriously as any other. It should not become the 'go-as-you-like' corner, where statements of the kind mentioned above can be made.

1. De, M., *Curr. Sci*., 2005, **88**, 683–684.
2. Chakraborti, D. *et al.*, *J. Environ. Monit*., 2004, **6**, 74N–83N.
3. Sengupta, M. K. *et al.*, *Arch. Environ. Health*, 2003, **58**, 701–702.

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Evidence to support that diclofenac caused catastrophic vulture population decline

Decline in the population of three species of *Gyps* vultures across the Indian subcontinent is among the most rapid ever recorded in birds. Numbers of oriental white-backed (OWBV *Gyps bengalensis*), and long-billed (LBV *Gyps indicus*) vultures have declined[1,2] by a minimum of 97% across northern India since the early 1990s, while those of the recently described slender-billed vulture (*Gyps tenuirostris*) have also declined rapidly[1]. Similar vulture declines have also occurred in Pakistan and Nepal[3]. Two recent papers[2,4] provide further convincing evidence to support the theory that the non-steroidal anti-inflammatory drug, diclofenac is the major cause of decline in vulture population across the Indian subcontinent. Initial results associating diclofenac with such declines came from Pakistan[3], where high mortality was observed in OWBVs, and a high proportion of dead birds had visceral gout (an accumulation of uric acid crystals in and on organs). Oaks *et al.*[3] found that all birds collected dead with visceral gout had detectable diclofenac residues in their kidneys. None of the birds found without visceral gout contained diclofenac. These results were supported by experimental evidence showing dose-dependent mortality in OWBVs fed on the tissues of livestock treated with a normal veterinary dose of diclofenac shortly before death. Experimentally treated OWBVs that died also exhibited visceral gout.

Recently, some researchers[5,6] acknowledged the relationship between diclofenac and vulture mortality discovered in Pakistan, but indicated that conclusive evidence on diclofenac poisoning was lacking for India. We argue that this evidence has been provided in the two recently published papers.

In the latest published paper, Shultz *et al.*[4] show a statistically significant association between the presence of gout and contamination of kidney or liver tissues with diclofenac in a sample of dead and dying OWBV and LBV collected across India and Nepal. Arun and Azeez[6] have argued, quite correctly, that visceral gout can occur for a variety of reasons other than diclofenac poisoning. However, it seems that, in reality, a negligible proportion of deaths of wild vultures that have gout at post-mortem involves some factor other than diclofenac. If wild vultures are dying with visceral gout from a cause other than diclofenac poisoning, it is difficult to explain why all 14 vultures collected with visceral gout across India and Nepal had traces of diclofenac in their tissues (like those tested in Pakistan[3]), whereas none of the dead birds without visceral gout did so. Sample sizes of dead vultures collected in India were admittedly small (28 birds examined), primarily due to the time delays and difficulties in obtaining vulture-collections permits

in some areas. However, there is no reason to assume that the samples collected were unrepresentative. Populations have now declined so much that it is unlikely that large sample sizes can be collected in future. The recent study of Shultz *et al.*[4] extends the geographical area of coverage by more than 2000 km eastwards from the Pakistan study, into northeastern India and Nepal. Taken together, the results of this and the earlier Pakistan study indicate that diclofenac is the major cause of population decline across most of the geographical range.

However, as indicated by Arun and Azeez[6], this information alone may still be insufficient to convince those with a healthy scientific scepticism that diclofenac is the 'universal' cause for the decline in vulture population. Questions arise as to whether sufficient livestock carcasses are likely to contain diclofenac at levels lethal to vultures to have caused such a rapid population decline, especially as the post-administration half-life of diclofenac in mammalian tissue is a matter of days[7]. The recently-published simulation modelling study[2] does much to answer this question; less than 1% (between 0.13 and 0.75%) of ungulate carcasses available to vultures would need to have contained levels of diclofenac lethal to vultures to have resulted in the observed rates of vulture decline. There are about 503 million livestock (projection based on Basic Animal Husbandry Statistics, 2004, Government of India) in India and estimated 5 million of them are given diclofenac injections annually. If we assume that 10–20% of ungulates (50–100 million) die per year, then between 5 and 10% of carcasses would be contaminated with high levels of diclofenac, if death occurred soon after treatment in all cases. Of course, it is unlikely that death really occurs soon after treatment in every case. However, comparison of these figures with those from Green *et al.*[2] shows that only a small proportion of treatments (between 1 and 15%) would have to be administered just before death to account for the decline in vulture population.

There is, we believe, still more persuasive evidence. Results from India, Nepal and Pakistan show that a similarly high proportion (75% in India/Nepal and 85% in Pakistan) of vultures found dead had visceral gout at post-mortem. This proportion is considered in detail in the recent publication[2], where the proportions of dead vultures that have symptoms of diclofenac poisoning are compared with those we would expect to see if diclofenac was the only cause for the decline. A model incorporating the best information available on vulture demography and foraging, and the latest vulture population survey data from the Bombay Natural History Society and The Peregrine Fund, is used to calculate the expected proportions. The results show that the proportion of dead vultures with signs of diclofenac poisoning is consistent with diclofenac being the only cause of the declines both in India and Pakistan and for OWBV and LBV[2]. Furthermore, the hypothesis that there is another cause for the decline, more important than diclofenac, is explicitly tested and is rejected both in India and Pakistan.

Although a multitude of factors listed by researchers[5,6] can cause mortality in vultures, none listed has the potential to cause the observed catastrophic decline in the vulture population over such a large and diverse geographical coverage. The results of the recent papers[2,4] along with the pioneering work in Pakistan[3], conclusively showed that diclofenac was responsible for the catastrophic mortality amongst vultures throughout the subcontinent.

We concur with the final conclusions of Arun and Azeez[6]. They nevertheless advocate the immediate replacement of diclofenac with other drugs, whilst cautioning against the use of drugs whose toxicity to vultures is unknown. In addition, we suggest that captive holding and breeding of vultures is essential, given current rates of population decline. Birds held and bred in captivity would be reintroduced within their historical range once their environment is diclofenac-free.

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3. Oaks, J. L. *et al.*, *Nature*, 2004, **427**, 630–633.
4. Shultz, S. *et al.*, *Proc. R. Soc. London Ser. B* (*Suppl.*), 2004, **10**,
5. Chhangani, A. K. and Mohnot, S. M., *Curr. Sci.*, 2004, **87**, 1496–1497.
6. Arun, P. R. and Azeez, P. A., *Curr. Sci.*, 2004, **87**, 565–568.
7. The European Agency for the Evaluation of Medicinal Products, Veterinary Medicines and Inspections, Committee for Veterinary Medicinal Reports, EMEA/MRL/885/03-FINAL, September 2003.

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