

Antibiotics in animals

SIR — In his leading article of 4 October¹, Stephen Budiansky takes issue with some comments I made regarding a recent study² linking subtherapeutic use of antibiotics in animals with human disease³. While he obviously hears the clarion differently from me, it is important to clarify some of the facts that he presented. (Those addressed by A.H. Linton⁴ are not repeated here.)

Nobody will disagree with Mr Budiansky that *Salmonella* should not be in meat. These and other less pathogenic organisms will, however, continue to contaminate food products while they continue to be selected for in the agricultural environment. It is precisely their antibiotic resistance that assures this selection and reflects the use of antibiotics.

Mr Budiansky questions "whether animals are a significant generator of resistant human pathogens". It is becoming increasingly more evident that resistant *Salmonella* causing human disease in the United States originate in animals⁵. Moreover, once introduced into a human population, such as occurred in a hospital nursery⁶ or the outbreak described in the *New England Journal of Medicine*², human-to-human spread may also occur. But resistant non-pathogenic strains are also involved. Since transfer of resistance occurs readily among pathogens and non-pathogens, it is not necessary that "human pathogens interact with animal pathogens in order to acquire resistance". This plasmid exchange makes it very relevant to speak about the environmental pool of these resistance plasmids no matter what their host.

Mr Budiansky cites the article by Atkinson and Lorian⁷ as evidence that resistance is not being spread and that resistance in the environment has reached an "equilibrium". But these data are limited to hospitals which have always been a haven of antibiotic use and antibiotic resistance. Furthermore, the study did look "at the big picture", (5.8 million isolates from 242 hospitals in the United States from 1971 to 1972) with the consequence that one cannot relate the findings to any individual hospital or individual patient. In fact, the authors of the report admit that "local outbreaks of bacterial resistance do arise and many have serious consequences". Moreover, they report increased resistance to multiple antibiotics in *S. faecalis* and *S. epidermidis* and sustained high levels of resistance to common antibiotics among many of the strains tested. The fact that some (but not all) resistance has stayed the same and not decreased is hardly grounds for complacency. More importantly, these data do not take into account the new emergence of infectious agents such as multi-resistant pneumococci, penicillin-resistant gonococcus, and ampicillin-resistant *Haemophilus influenzae*

which now appear in the community.

The microbial environment of animals and man is not separate. Consequently, one must look at the sources of these resistant bacteria and resistance factors in the "common" environment. To this viewer, for the reasons stated in my editorial, a major source in the United States is animals.

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Stephen Budiansky replies:

Levy seems to suggest that feeding farm animals antibiotics makes contamination of meat by salmonella more likely. In fact, several studies address this very question and reach the opposite conclusion¹⁻².

But in any event, the burden of my argument was that salmonella is simply the

wrong example to be looking at. Antibiotic resistance is presumably of concern because it is making the treatment of bacterial infections more difficult. Since antibiotics are not normally used to treat salmonellosis, it really does not matter whether the salmonella that is making people sick is resistant or not, or of animal origin or not. Furthermore, salmonella is one of the few pathogens common to farm animals and humans; that animal feeds may be responsible for resistant salmonella does not mean that they are responsible for resistant strains of other, strictly human, pathogens that are one step removed from animal guts.

Indeed it seems unlikely that the blame for ampicillin-resistant *H. influenzae* can be laid to animal feeds, since ampicillin is not used in feeds. And why postulate a subtle origin of penicillin-resistant gonorrhoea, involving multiple transfers of resistance plasmids between bacterial species, when we have staring in our faces the blatant bombardment of humans (and especially those humans with gonorrhoea) by penicillin for the last 40 years.

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Too much hype

SIR — An aggressive publicity campaign has been launched to promote Immunovir (or Isoprinosine), recently approved in Great Britain^{1,2}. The campaign has even found some echo in the columns of *Nature*³.

This is hardly surprising, since this drug is already better known from its promotion rather than its results. For example, in 1982, the US drugstore bookstalls hyped the drug with a paperback, which not only claimed a cure for herpes but also accused the Food and Drug Administration (FDA) of preventing herpes victims from using and benefiting from the miraculous substance⁴. Although the author did not support his claims with any scientific publications, the book claimed him to be "an authority on herpes".

It is even more curious that reports describing the ineffectiveness of Isoprinosine⁵⁻⁷ seem to have been ignored on the occasion of its British premiere, as was also the fact that despite the commercialization of Isoprinosine in France, Germany, Italy and Spain for several years, the number of herpes patients has not decreased there. The purpose of this communication, however, is not to add data based on experience with this drug showing that Isoprinosine clearly has no beneficial effect on herpes patients, but rather, to question the practices of pharmaceutical companies and of the media.

It is therefore incredible to read in the *Sunday Times*: "A pill for AIDS" is available, "but it may come too late"¹. Or in the *Financial Times* that: "Immunovir may

also be able to prevent AIDS, a fatal breakdown of the immune system"². Last but not least, there is the statement: "The drug Immunovir is the first in a new class of drugs which appear to have very promising potential in treating and preventing viral diseases ranging from influenza to shingles to AIDS and to some kinds of cancers"².

It is time to ensure that responsible lay publications prevent the exploitation of the public in such ways. In an era of proliferation of regulatory bodies and ethics committees whose role and motivations are not always obvious, it is unfortunate to have to suggest yet another body to impose restrictions on scientists and journalists concerning the publicity of potential "breakthroughs". But, if it is unacceptable that research hopes are wildly publicized (for more often than not false hopes are more damaging for the public than patience), only self-discipline by scientists and journalists will eradicate the sensational.

Certainly, those who have vested interests in pharmaceutical companies, politicians promoting their re-election or scientists promoting their career and their laboratory budget through the lay press, will reject such limitations, but the majority would accept them with relief.

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