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Antibiotic use in animals

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Sir,

We would like to reply to some of the points made by John Turnidge[1] in response to our recent paper on the potential risks to human health arising from the use of antibiotics in food animals,[2] and another on the consequences of the ban of growth-promoting antibiotics in Europe.[3]

Firstly, he characterized our position as being that 'no action is required'. This is incorrect. Our belief is that to achieve better human health, risk managers must base their actions on a full and fair consideration of all relevant scientific evidence, including evidence of both benefit and risk. We also believe that risk managers need to know when there is scientific consensus and when this is lacking—something that has not always been made clear. Furthermore, we believe that evidence has sometimes been used selectively to support a particular viewpoint, and we therefore drew attention to scientific findings that we believed had not been adequately taken into account, particularly those that suggested that the use of antibiotics in animals might *not* pose a risk to human health, and that the abandonment of the use of growth-promoting antibiotics might have *adverse* as well as advantageous effects. We also drew attention to the need for more and better scientific information—to the same extent as those who described the application of the Precautionary Principle as requiring that 'additional information necessary for a more objective assessment of risk' will be sought[4]— agreeing with John Turnidge. We had little to say on the process of risk management, believing this to be the concern of risk managers, although we might agree that actions taken that are uninformed by full consideration of the relevant scientific information might lead to undesirable human health consequences.

Secondly, we agree that resistance is likely to be selected whatever the context of use, but do not presume that an antibiotic used in both animals and humans will be more likely to select for resistance as a consequence of animal rather than human usage. After all, the use of antibiotics in humans, acting both as selector and amplifier, is believed to be the major driver of resistance for most human pathogens. The use of virginiamycin in animals over decades did not appear to result in resistance in relevant human pathogens,[5,6] but when quinupristin–dalfopristin was introduced, streptogramin-resistant *Enterococcus faecium* were immediately observed, but with a resistance mechanism unlike that hitherto reported in animals.[2,6]

Thirdly, we urge that debate should continue, and should indeed be broadened, but not in an adversarial manner. It is simply not good enough for us as scientists to say that 'it is likely that [resistant commensal] species are... important reservoirs of resistance genes that can be transmitted to the human gut flora'. Surely it is not beyond our competence to make appropriate epidemiological, microbiological and clinical observations and devise relevant experiments to provide unequivocal quantitative answers!

Our aim—about which John Turnidge expressed confusion—has been to try to ensure that the scientific debate should be even-handed, making it clear to risk-analysts and managers where there is agreement, where there is disagreement, and where there is a lack of adequate information, in the science on which they base their actions. We remain disinterested in relation to scientific findings. We are neither trying to reverse bans nor to define prudent antibiotic use, merely trying to ensure that scientific data and process are used even-handedly by those who are involved in these activities.

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