

Antimicrobial resistance in zoonotic bacteria: lessons learned from host-specific pathogens

Trudy M. Wassenaar^{1*} and Peter Silley^{2,3}

¹Molecular Microbiology and Genomics Consultants, Zotzenheim, Germany,

²MB Consult Limited, Lymington, UK and

³Department of Biomedical Sciences, University of Bradford, West Yorkshire, UK

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Abstract

The relative contribution of veterinary and human clinical treatments to the selection of antimicrobial resistance in zoonotic pathogens remains controversial. In this review, we consider bacterial pathogens that differ in host specificity and address their resistance profiles: pathogens that only occur in the human host, pathogens that are specific to particular food-producing animals and pathogens that occur in both host types. Compared with those pathogens restricted to a single animal host, pathogens found in both human and animal hosts appear to have higher incidences of resistance. However, the most urgent and severe resistance problems occur with pathogens exclusively infecting humans. Differences exist in the available genetic repertoire of a bacterial species and these are reflected in the observed resistance patterns; it is important to note that different bacterial species do not automatically result in similarly resistant populations when they undergo comparable selection in different host species. Thus, within a bacterial species, prevalence of resistance can differ between populations isolated from different hosts. For some species, fluctuations in dominant subpopulations, for instance particular serotypes, can be the most important factor determining resistance. The frequently expressed opinion that veterinary use of antimicrobials is at the heart of many resistance problems may be an oversimplification of the complex forces at play.

Keywords: antibiotic resistance, zoonosis, food-borne pathogen, veterinary medicine, human health

Introduction

Antimicrobials provide a tremendous selective pressure on any susceptible bacterial population, whereby cells that are less susceptible are at a great advantage to survive and multiply. Depending on the mechanism of action of the antimicrobial, the severity of selective pressure and the possible genetic strategies that render bacteria either less susceptible or cause partial or full resistance, a population can shift sooner or later from 'completely susceptible' via intermediate stages to one that in the laboratory is determined to be 'resistant'. This can happen

in any environment, including the living host, be it human or animal.

The term 'resistance' can mean different things and even when the minimum inhibitory concentration (MIC) of an antimicrobial is given, this can vary according to the testing methodology. The breakpoint MIC for an antimicrobial agent can be either the threshold above which a given bacterial pathogen no longer survives or multiplies *in vitro*, the MIC value above which it is unlikely to respond to treatment, or a description of the wild-type population (Simjee *et al.*, 2008). These *in vitro* and clinical breakpoints may not be identical, due to the pharmacokinetic parameters resulting from different drug dosing regimens. An epidemiologist interested in resistance development within bacterial populations more likely uses a biological MIC or epidemiological cut-off value to separate a susceptible (wild-type) population from a less

*Corresponding author. Tannenstrasse 7, 55576 Zotzenheim, Germany. E-mail: wassenaar_t@yahoo.co.uk