

Sub-inhibitory concentrations of vancomycin prevent quinolone-resistance in a penicillin-resistant isolate of *Streptococcus pneumoniae*

ABSTRACT

This finding may have clinical implications, particularly in relation to the risk associated with a bacterial isolate of quinolone-resistant pneumococci.

INTRODUCTION

The most common pathogen in the United States is the gram-negative bacteria, *Escherichia coli*, which is transmitted through the mouth, noses, and eyes. The pathogen is resistant to vancomycin, a bactericide used in penicillin-resistant *Staphylococcus aureus* (*S. aureus*), but resistant to cephalosporins, used in penicillin-resistant *E. coli*. There is also a sub-group of pathogens that are resistant to vancomycin and cephalosporins, including gram-negative bacilli, and enterococci, which are resistant to vancomycin and cephalosporins. In recent years, the worldwide emergence of penicillin-resistant pneumococci has jeopardized the efficacy of β -lactam antibiotics. Furthermore, penicillins are often resistant to multiple drugs, which means they can limit the choice of alternative compounds. New anti-pneumogenic drugs must combine their abilities to rapidly kill target organisms and inhibit their development, as newer quinolones with good anti-gram-positive activity such as trovafloxacin may block resistance development. The combination of vancomycin and trovafloxacin was found to enhance the bactericidal activity of the antibiotic against penicillin-resistant pneumococci in vitro and experimental meningitis in rabbits. However, we have now demonstrated that sub-inhibitory concentrations of 0.03 mg/L vancoramycin also prevented resistance to ciprofloxacin but not per se (this observation deserves special attention).

CONCLUSION

Remarkable conclusions Similar to the synergistic activity of cell wall active antibiotics and aminoglycosides in enterococci and other gram-positive pathogens, the data presented here is comparable. Although not entirely clear, it plays a significant role in both prevention and treatment effectiveness in severe infections. A similar model could also be used with the combination of cell wall inhibitors plus trovafloxacin or other specific quinolones for pneumococcal infections as well. This observation raises the possibility of combining these two drug interactions associated with vancomycin