EPIDEMIOLOGY OF TRIMETHOPRIM RESISTANCE MEDIATED BY R-PLASMIDS

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When trimethoprim resistance conferred by resistance factors (now termed R plasmids) was first reported (Fleming, Datta & Grüneberg 1972) the percentage of organisms containing such plasmids was very low being less than 1%. Later the percentage of such organisms in urinary isolates increased but still remained low (less than 2%) in the United Kingdom (Amyes, Emmerson & Smith 1978). In France and Italy, although the initial frequency of plasmid-containing organisms resistant to trimethoprim (Tp^r) was low (less than 2%), rapid increases in the frequency of Tp^r organisms occurred (Acar & others 1977; Romero & Perducca 1977). This communication reports the proportion of Tp^r organisms in three hospitals, the therapy patients had been given and examines the persistence of Tp^r bacteria in patients.

From October 1978 until April 1979 urinary tract isolates from three separate hospitals were examined for trimethoprim resistance. Cultures of those organisms, insusceptible to trimethoprim, were mated with a rifampicin-resistant Escherichia coli recipient strain for 24 hours. Plasmid transfer was detected on Davis-Mingioli minimal medium plates containing both rifampicin and trimethoprim.

Hospital	Percentage of organisms insusceptible to 10μg/ml Tp	Percentage of organisms detected with Trimethoprim R plasmids
1 Convalescent	46.3	13.4
2 General	20.4	4.5
3 General	12.5	5.7

The results show that there is considerable variation among hospitals, all of which are within five miles of one another and that one hospital had the highest frequency of Tp^r organisms yet recorded in the literature. Twenty two plasmids have been examined further. Fifteen plasmids possessed the same resistance pattern, namely to trimethoprim, streptomycin, ampicillin and sulphonamide, but they were not identical as they were not all from the same compatibility group. Five other plasmid types were found among the remaining seven plasmids. Unusually, ampicillin resistance was linked to trimethoprim resistance in all but six plasmids.

In order to examine the persistence of transferable Tp resistance, the urinary isolates of two patients not being given antimicrobial therapy were examined twice-weekly for a year after the initial identification of a Tp^r plasmid in bacteria isolated from their urine. All isolates from patient A retained the transferable trimethoprim-streptomycin plasmid throughout the year, although a separate kanamycin-sulphonamide R plasmid was lost after two months. Isolates from patient B retained the Tp^r plasmid for ten months.

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