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Editorial

Drug Resistance in *Salmonella typhi*: Tip of the Iceberg

Shrikala Baliga, M.D.

Associate Professor of Microbiology,
Kasturba Medical College, Mangalore-575001

Address for Correspondence:

Dr. Shrikala Baliga,

Associate Professor of Microbiology,
Kasturba Medical College, Mangalore-575001

E-mail: shrikalab@yahoo.com

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Abstract:

Salmonella typhi is one of the most resistant organisms with multi-drug resistant strains reported from many countries. Initially, individual plasmids were known to code for resistance, but since 1988 a single plasmid has been identified to code for multidrug resistance. It has been found that in certain areas, *S. typhi* has lost this acquired resistance.

Key Words: Drug resistance, Plasmid, *Salmonella typhi*

Salmonella enterica serotype *typhi*, the etiological agent of typhoid fever, is known to be amongst some of the most resistant of pathogens.^{1,2} Typhoid fever causes 20 million cases annually with at least 700,000 deaths. Typhoid fever is endemic in many countries including India and if not treated appropriately has a mortality rate of 30%. Appropriate treatment reduces the mortality rate to as low as 0.5%.¹

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endemic in many countries including India and if not treated appropriately has a mortality rate of 30%. Appropriate treatment reduces the mortality rate to as low as 0.5%.¹

Chloramphenicol resistance is known in *Salmonella Typhi* since 1972, when plasmids of incompatibility group Inc H, coding for chloramphenicol resistance were found in *S. typhi*. Multi drug resistance, (defined as resistance to all the first line antibiotics used to treat typhoid fever, i.e chloramphenicol, ampicillin, co-trimoxazole and tetracycline) has been endemic in the Indian Sub continent and South East Asian countries since 1984. Though initially, individual plasmids were known to code for resistance to each of these antibiotics, since 1988 a single plasmid was known to code for multidrug resistance. This plasmid belongs to incompatibility group H I1 and is highly transmissible. Chloramphenicol resistance, MDR *S. typhi* and now low level fluroquinolone resistance³ have emerged as the newer challenges to treatment of typhoid fever.

There is still some light at the end of the tunnel. It has been seen that in certain areas especially Northern India *S. typhi* has lost this acquired resistance and Chloramphenicol resistance has

reduced from a high of 18% to only 2%. This has been noticed in our Institutes also where Chloramphenicol resistance has reduced drastically over the years (unpublished data, presented at National conference). It has also been seen that chloramphenicol is still being used to treat typhoid fever in some areas in Indonesia where 96% of strains remain sensitive to Chloramphenicol. Low level fluoroquinolone resistance is due to point mutations in the gene coding for DNA gyrase enzyme. This thankfully is not transferable on plasmids. Extended spectrum beta lactamases, coded by transposons and responsible for resistance to third generation cephalosporins have been identified in *S. typhi*. Suitable alternatives to treat MDR *S. typhi* include azitromycin, third generation cephalosporins and carbapenems. Though public health, sanitation and vaccines do have a role to play in control of typhoid fever, it is the antimicrobial therapy which plays a key role in management of typhoid fever.

It is likely that *S. typhi* will continue to acquire genes responsible for antibiotic resistance. The timely detection of these genes and prevention of their spread is the need of the hour to control antibiotic resistance among *S. typhi*. The article by Mandal et al emphasizes the detection of plasmids encoding for MDR *S. typhi* and their transmissible nature. More research could also be directed towards factors responsible for their spread and means to prevent dissemination of such plasmids so as to limit drug resistance.

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