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Role of Folate Antagonists in the Treatment of Methicillin-resistant Staphylococcus aureus Infection

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Methicillin-resistant Staphylococcus aureus (MRSA) infection has reached epidemic proportions, and therapeutic options are limited because these strains are often multidrug resistant. However, the new strains of community-acquired MRSA show decreased resistance to trimethoprim-sulfamethoxazole. Clinical and experimental reports show a mixture of successes and failures with trimethoprim-sulfamethoxazole treatment. A reason for failure might be the amount of thymidine released from damaged host tissues and bacteria, a concept strengthened by the fact that S. aureus thermonuclease releases thymidine from DNA. Thus, success or failure with trimethoprim-sulfamethoxazole may well depend on the amount of tissue damage and organism burden, rather than acquisition of a resistance gene. Clinical trials and experimental animal studies show high failure rates, perhaps because of released thymidine. The use of trimethoprim-sulfamethoxazole for community-acquired MRSA infection should not be endorsed without further research.

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