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EFFECTS OF TREATING STERPTOCOCCUS PNEUMONIAE WITH CEFUROXIME, VANCOMYCIN OR RIFAMPICIN ON TNF AND NO PRODUCTION BY MACROPHAGES

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Antibiotic therapy has revolutionized the abillity to control infectious diseases and thereby to reduce the associated morbidity and mortality. In recent years, a correlation has been found between the antibiotic therapy of bacterial infections and the intensity of the inflammatory response, which in turn directly correlates with morbidity. Little is known regarding the inflammatory response induced during treatment of Gram-positive bacteria.

In this study we examined the effect of antibiotic treatment of Streptococcus pneumoniae on proinflammatory mediators released by macrophages in vitro. Various antimicrobial agents, with different mechanisms of action, were examined: protein synthesis inhibitors vs. cell wall active antibiotics. Treatment of murine macrophages (J-744) infected with Streptococcus pneumoniae with vancomycin or cefuroxime (cell wall active antibiotics), induced higher levels of TNF than by treatment with rifampicin (bacterial protein inhibitor), in all bacterial concentations examined (105-107 per ml). At a concentration of 107 bacteria/ml, mean TNF levels induced by vancomycin, cefuroxime and rifampicin were 20.3, 16.2 and 2.9 mcM, respectively ($p=0.01$). Also, NO released by the macrophages was higher when the bacteria (106-107 per ml) were treated with vancomycin or cefuroxime as compared to rifampicin. At a concentration of 107 bacteria/ml, mean NO levels induced by vancomycin, cefuroxime and rifampicin were 27.6, 31.7 and 22.3 ngr/ml, respectively ($p<0.05$).

It is concluded that the specific antimicrobial treatment used has an effect on the release of inflammatory mediators by macrophages when treating Streptococcus pneumoniae in vitro. This might affect the clinical course and potential complications of this common infection.