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The Role of Overproduction of Nitric Oxide in Apoptosis of BALB/C Mice Macrophages Infected with LeishmaniaMajor in Vitro

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Abstract:

Nitric oxide (NO) derived from activated macrophages has been shown to be crucial for the host's leishmanicidal activities. Excess NO, however, can in¬duce apoptosis in some cell types, including macrophages. In the present inves¬tigation, we studied the role of NO in inducing apoptosis of BALB/c mice mac¬rophages infected with Leishmania major in vitro. The peritoneal macrophages were harvested and cultured with or without L.major in the presence of a donat¬ing reagent (s-Nitroso-N-Acetylpenicillamine (SNAP)) or an inhibitor of NO synthase (NG -Methyl-L-Arginine (NMMA)). The concentration of NO in cul¬ture supernatants was measured after 18 hours incubation. Simultaneously, mac¬rophages undergoing apoptosis were identified by fluorescence and electron mi¬croscopy. The results showed an increase in apoptosis rate in parallel to nitrite production in macrophages cultured in the presence of SNAP. Although mac¬rophages infected with L.major had no significant increase in NO production, they showed a significant increase in apoptosis rate. Besides, macrophages cul¬tured with NMMA, had a decreased NO production but the apoptosis rate in¬creased. Therefore, mechanisms involved in apoptosis induction in the last two groups may be different from NO overproduction.

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