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Windows and MAC OS.Monocyte activation by endotoxin is mediated by TNF. The mechanism of the enhancement of vascular permeability in endotoxemia remains a
central question in septic shock. We have recently demonstrated that a concomitant reduction in circulating lymphocytes and monocytes can be induced in
endotoxemia by the administration of anti-tumor necrosis factor (TNF) antiserum. Using the murine model of endotoxin, we sought to explore the role of TNF in the
recruitment of monocytes into the circulation of endotoxemic mice. LPS (0.01 microgram/mouse) was administered to mice treated with anti-TNF or anti-interleukin 1
(IL-1) antiserum. Recruitment of monocytes into the circulation was assessed by differential leukocyte counts and evaluation of the hemolysis induced by TNF-
neutralizing monoclonal antibody (MAb). After LPS administration, blood monocytes increased from 2.0 x 10(5) to 5.0 x 10(5) cells/mL, while neutrophils decreased
from 18 x 10(5) to 6 x 10(5) cells/mL. In mice given an anti-TNF antiserum, monocytes increased only 3.6 x 10(5) cells/mL, while neutrophils rose from 12.0 x 10(5) to
20 x 10(5) cells/mL after LPS. A much smaller increase of monocytes occurred after LPS in mice given an anti-IL-1 antiserum (monocytes, 2.2 x 10(5) cells/mL;
neutrophils, 4.0 x 10(5) cells

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