WIDE RANGE OF ACTIONS OF THE TUMOR NECROSIS FACTOR (TNF)

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Tumor necrosis factor (TNF) and lymphotoxin (TNF-alpha, TNF-beta) are two structurally related cytokines. Many of their functions are identical to those of IL-1, but to achieve a biological effect, their concentration must be 100 times greater than that of IL-1. They are capable of killing certain sensitive cell types, most often tumor cells, in vitro. IL-1 and TNF act synergistically on fibroblasts, enhancing the production of prostaglandin E2 (PGE2). They cause neutrophil aggregation and thromboxane synthesis.

Tumor necrosis factor (tumor necrosis factor or cachectin) is produced mainly by macrophages, as well as endothelial cells, neutrophils, and lymphocytes. A wide range of cells, in particular cells of the reticuloendothelial system (RES), have specific receptors for TNF. The half-life of TNF in plasma is 15 minutes. Under normal conditions, TNF production is very low, but in the event of trauma, inflammation, burns, or endotoxemia, massive TNF production occurs within minutes by all cells, especially macrophages. Although high levels of TNF in the circulatory system are maintained for a very short period of time, the response to this increase is prolonged. This is due to the initiation of secondary cascades, such as the activation of oxidants and proteases released from white blood cells, and these cascades can now be self-sustaining. FNO has a wide range of actions that can be attributed primarily to the stress response.

FNO also influences the formation of the immunological response: 1) the release of neutrophils from the bone marrow; 2) the marginalisation and

activation of neutrophils, including the release of oxidants and enzymes; 3) activation of macrophages to release oxidants and arachidonic acid metabolites, as well as the production of other cytokines, such as IL-1 and IL-6.

Thus, these reactions are beneficial because they are aimed at protecting the body against bacteria, but they can also have negative effects due to their ability to cause self-destructive inflammation.