

Rat TNF- α ELISpot Kit

PRODUCT SPECIFICATIONS

Catalogue N°	867.021.001PC - 1 x 96 Discovery (pre-coated plate) 867.021.002PC - 2 x 96 (pre-coated plates) 867.021.005PC - 5 x 96 (pre-coated plates)
Target species	Rat
Specificity	Recognizes natural rat TNF-a
Incubation	3h after cell stimulation
Cross Reaction	No cross reactivity with other rat cytokines
Kit Content	Diaclone Pre-coated ELISpot kits include precoated PVDF plates, Detection antibody, Alkaline phosphatase conjugate, BSA, BCIP/NBT ready-to-use substrate buffer.
Synonym	TNF-a TNF-alpha

BACKGROUND

Tumor Necrosis Factor (TNFa), also known as cachectin, is a polypeptide cytokine produced by monocytes and macrophages. It functions as a multipotent modulator of immune response and further acts as a potent pyrogen. TNFa circulates throughout the body responding to stimuli (infectious agents or tissue injury), activating neutrophils, altering the properties of vascular endothelial cells, regulating metabolic activities of other tissues, as well as exhibiting tumoricidal activity by inducing localized blood clotting. TNFa also inhibits lipoprotein lipase activity resulting in cachexia, a physical wasting condition. Activation of B-cells by the Epstein Barr virus can be inhibited by TNFa. Due to its varied actions throughout the immune system, TNFa may play a role in the pathogenesis of many disease states.

TNFa production is mediated by the action of lymphokines and endotoxins on the macrophage. Purified monocytes produce TNFa within four hours of stimulation by recombinant IL-2 and there is some *in vitro* evidence to suggest that TNFa is expressed at high levels and with prolonged kinetics in T cells stimulated by both CD2 and CD28. Secretion of TNFa is enhanced by gamma interferon. TNFa then induces or enhances the specific production of Class I MHC antigen, GM-CSF, and IL-1. Recent evidence has suggested an intracellular role for this peptide.

TNFa may play a significant role in the pathogenesis of inflammatory disease of the joints and other tissues. Chin et al. found that TNFa, along with IFNg and IL-1 increased cell surface expression of ICAM-1 on synovial fibroblasts. Alvaro-Garcia et al. reported that TNFa stimulates synovial proliferation.

Waage et al. found that increased levels of TNFa in patients with septicemia and meningococcal disease correlated with fatal outcome. Scuderi et al. suggest that increased levels of this cytokine may play a role in the host defense mechanism against parasitic infections. Girardin et al. reported that increased serum TNFa levels correlated

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with the number of risk factors involved in children with gram-negative sepsis and purpura fulminans. Elevated levels of TNF α were also found in individuals suffering from myocarditis.

Role for TNF α in the pathogenesis of AIDS has also been pointed out. Alveolar macrophages (AM) from HIV positive individuals with opportunistic lung infections have been shown to spontaneously produce higher levels of TNF α *in vitro* than those HIV positive individuals without infection and HIV negative controls. Krishnan et al. report that higher TNF α production by AM was associated with lower counts of pneumocystis carinii in bronchoalveolar lavage fluid, indicating that TNF α may play a role in the control of this infection in AIDS. Israel-Biet et al. also reported in *in vitro* studies, that AM that express HIV (p24+) released significantly higher levels of TNF α than p24- alveolar macrophages and controls. Reddy et al. found persistently elevated levels of circulating TNF α in HIV seropositive individuals and suggest a possible involvement of this cytokine in the development of AIDS.

Measurement of TNF α levels has also been shown to be useful in transplant research, where Maury et al. and McLaughlin et al. Both reported TNF α to be markedly elevated in renal allograft rejection episodes. Recent evidence has been presented on increased TNF α levels in Bone Marrow Transplant (BMT). BMT patients with major transplant related complications such as interstitial pneumonitis and severe acute graft-versus-host disease had TNF α levels significantly increase over controls.

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