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Tumor necrosis factor
(TNF) is a critical
cytokine, which
contributes to both
physiological and
pathological processes.
This mini-review will
briefly touch the
history of TNF
discovery, its family

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members and its
biological and
pathological functions.
Then, it will focus on
new findings on the
molec...

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**Tumor necrosis
factor - National
Institutes of Health**

Medicine And
Biology
Tumor necrosis factor
(TNF, cachexin, or
cachectin; often called
tumor necrosis factor
alpha or TNF- α) is an
adipokine and a
cytokine. TNF is a

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member of the TNF superfamily, which consists of various transmembrane proteins with a homologous TNF domain.. As an adipokine, TNF promotes insulin resistance, and is associated with obesity-induced type 2 diabetes.

Tumor necrosis factor - Wikipedia

F. Atzeni, P. Sarzi-
Page 8/21

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Puttini, in Brenner's
Encyclopedia Of
Genetics (Second
Edition), 2013 Abstract.
Tumor necrosis factor
(TNF), a 17 kDa protein
consisting of 157
amino acids, is a
homotrimer in solution
that is mainly produced
by activated
macrophages, T
lymphocytes, and
natural killer (NK)
cells. Proinflammatory
cytokines such as TNF
and interleukin (IL)-1 β

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play a key role in the ...
Proceedings Of
**Tumor Necrosis
Factor Alpha - an
overview |
ScienceDirect ...**

Biosimilars to these anti-TNF agents have also been developed for use within the United States. Two anti-integrin biologics (natalizumab and vedolizumab) have been approved for use in IBD treatment. And ustekinumab, a

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biologic that targets
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cytokines
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interleukin-12 and
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and interleukin-23 (IL-12
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and IL-23), has been
approved for Crohn's
disease ...

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**Advances in the
treatment of Crohn's
disease and ...**

Medicine And
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About the Societies.
The Association for
Academic Surgery is
widely recognized as
an inclusive surgical
organization. The

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impetus of the membership remains research-based academic surgery, and to promote the shared vision of research and academic pursuits through the exchange of ideas between senior surgical residents, junior faculty and established academic surgical professors.

**Home Page: Journal
of Surgical Research**

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Mitochondria are the target of stress injury. The generation of ROS in mitochondria then induces the release of cyt-c by mechanisms related to Bcl-2 family proteins (Bcl-2, Bcl-Xl, Bax, and Bid). Once cyt-c released, it binds to caspase-9 to form a complex which subsequently activates caspase-3 and other caspases, such as caspase-2, -6, -8 and -10.

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**Advances in the
Studies of Ginkgo
Biloba Leaves
Extract on ...**

Finally, this review will briefly summarize recent advances in understanding TNF-induced cell survival, apoptosis and necrosis (also called necroptosis).

Understanding new findings and emerging concepts will impact future research on the

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molecular mechanisms
of TNF signaling in
immune disorders and
cancer-related
inflammation.

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**Tumor necrosis
factor - PMC**

IL-17RA IL-17 receptor
A, TRAF6 TNF receptor-
associated factor 6,
IL-36R IL-36 receptor,
IL-1RAcP IL-1 receptor
accessory protein,
TWEAK tumor necrosis
factor (TNF)-like weak
inducer of apoptosis ...

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Advances in the pathogenesis of psoriasis: from ...

Tumor necrosis factor (TNF) drives chronic inflammation and cell death in the intestine, and blocking TNF is a therapeutic approach in inflammatory bowel disease (IBD). Despite this knowledge, the ...

Group 3 innate lymphoid cells produce the growth

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factor HB ...

Two theories of the direct initiation of apoptotic mechanisms in mammals have been suggested: the TNF-induced (tumor necrosis factor) model and the Fas-Fas ligand-mediated model, both involving receptors of the TNF receptor (TNFR) family coupled to extrinsic signals.. TNF pathway. TNF-alpha is a cytokine produced mainly by

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activated macrophages, and is the major extrinsic mediator of apoptosis.

Apoptosis - Wikipedia

A plasma cytokine panel [interleukin-1 β (IL-1 β), IL-2, tumor necrosis factor- α (TNF- α), interferon- γ (IFN- γ), and IL-10] also showed no significant differences (fig. S5C). Although more extensive evaluation of

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potential toxicity is
needed for preclinical
development, these
results suggest that
L8-cLNPs are not toxic
or immunogenic when

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genome editing
using targeted lipid
Biology

Severe coronavirus
disease 2019
(COVID-19) can
manifest as a viral-
induced

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hyperinflammation with multiorgan dysfunction. It has been documented that severe COVID-19 is associated with higher levels of inflammatory mediators than a mild disease, and tracking these markers may allow early identification or even prediction of disease progression. It is well known that C-reactive protein (CRP) is ...

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