Maximizing Patient Results in AI Rheumatic Conditions

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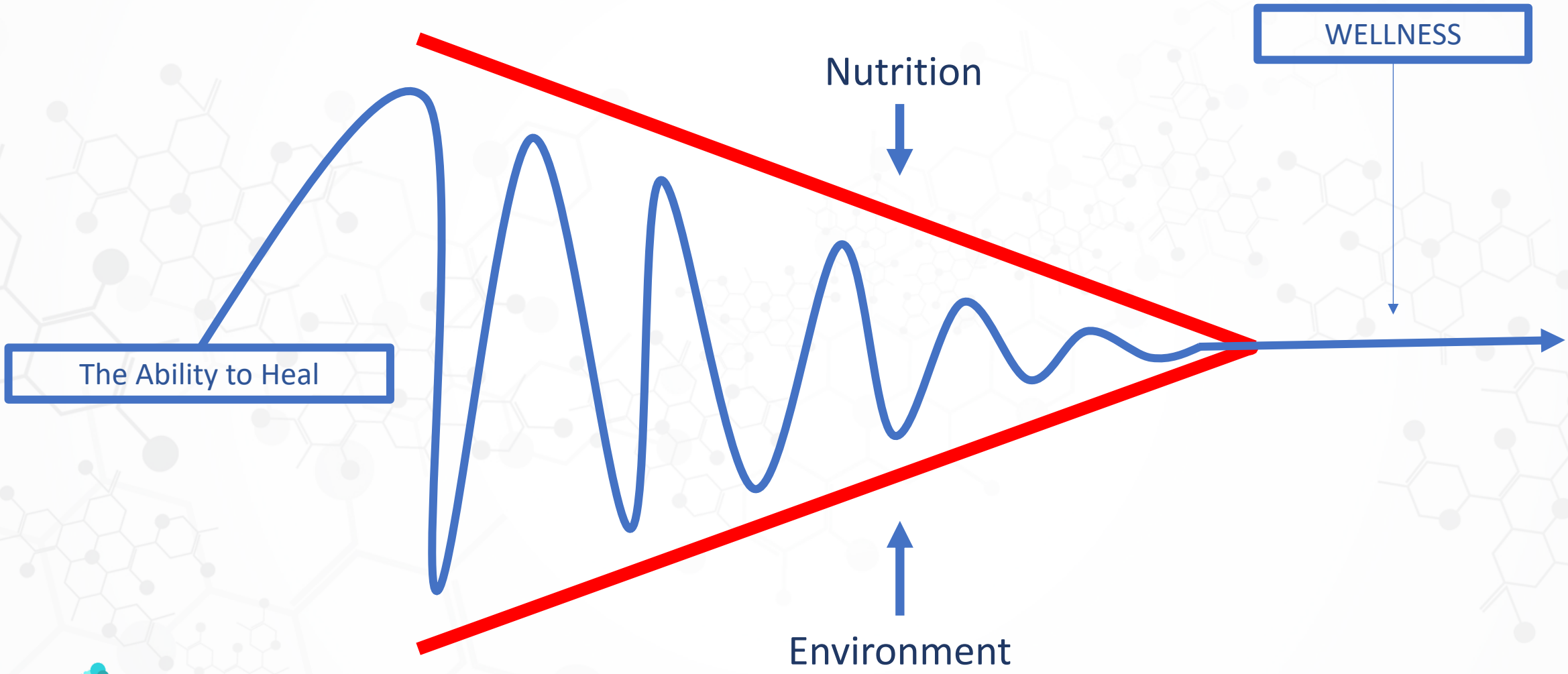
Disclaimer

- *Information in this presentation is not intended, in itself, to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

Lifestyle + Genetics = Chronic Health IMPROVEMENT

Protocols

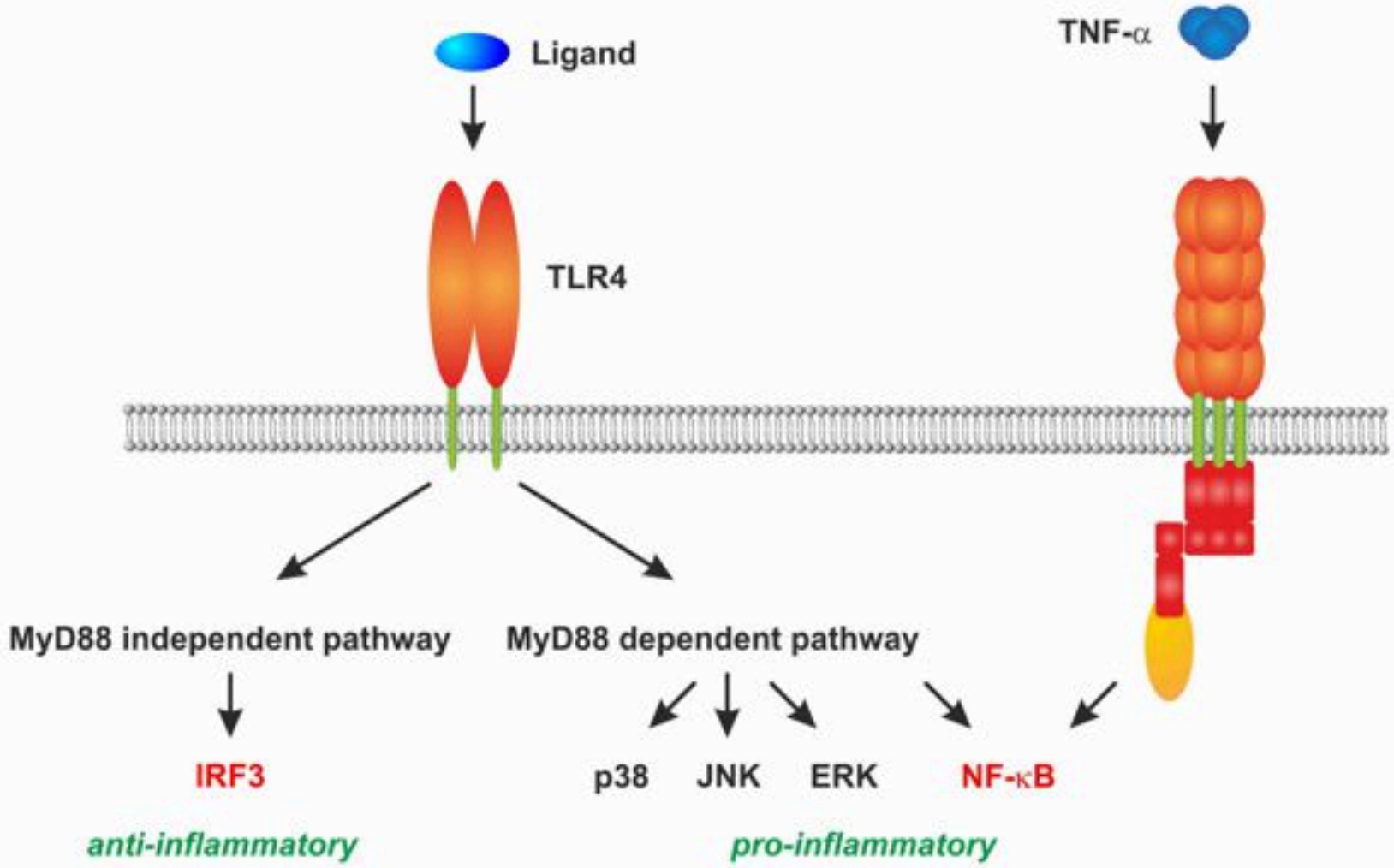


Review

Rheumatoid arthritis: From autoimmunity to synovitis and joint destruction

Marie-Christophe Boissier [a, b], Luca Semerano [a, b], Salima Challal [a, b], Nathalie Saidenberg-Kermanac'h [a, b], Géraldine Fa[illegible] [a, b]

outside the joints. The interactions between genes and environment are crucial in all stages of the disease, involving namely genes from major histocompatibility complex locus, and antigens such as tobacco or microbes (e.g. *Porphyromonas gingivalis*). T and B cells are activated as soon as the earliest phases of the disease, rheumatoid arthritis appearing as a Th1 and Th17 disease. Inflammatory cytokines have a considerable importance in the hierarchy of the processes involved in RA. The joint destruction seen in RA is caused not only by cytokine imbalances, but also by specific effects of the Wnt system and osteoprotegerin on osteoclasts and by matrix production dysregulation responsible for cartilage damage. Both innate and adaptative immunity demonstrated their respective cornerstone position in rheumatoid arthritis, since targeted treatments has been efficiently developed against TNF-α, IL-6 receptor, IL-1β, CD20 B cells and T-cell/Dendritic cell interactions.



http://www.wideralab.org/index.php/2-uncategorised/16-inflammatory-signalling-pathways-in-stem-cells

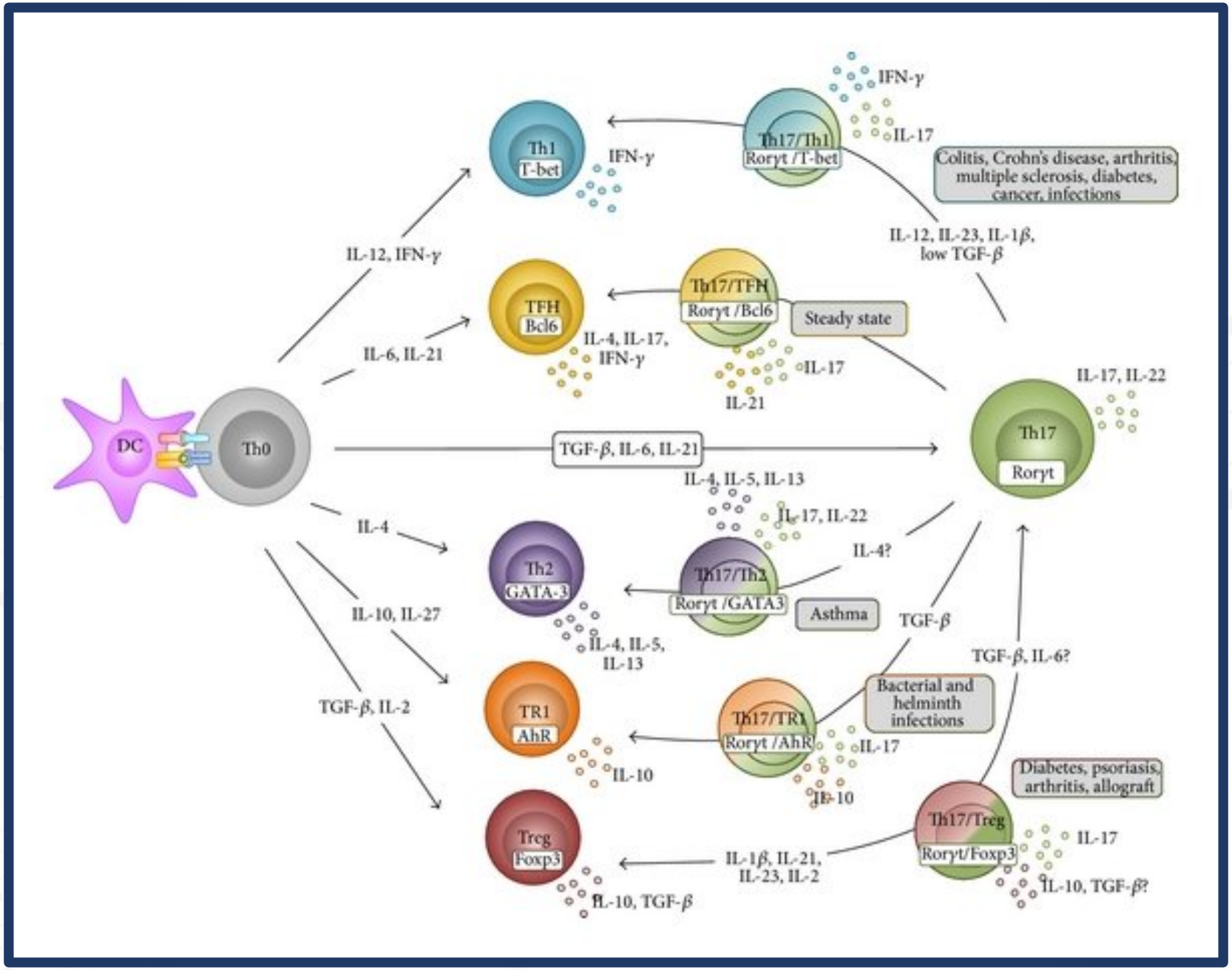
Review > Adv Immunol. 2012;115:45-71. doi: 10.1016/B978-0-12-394299-9.00002-3.

Autoimmune arthritis: the interface between the immune system and joints

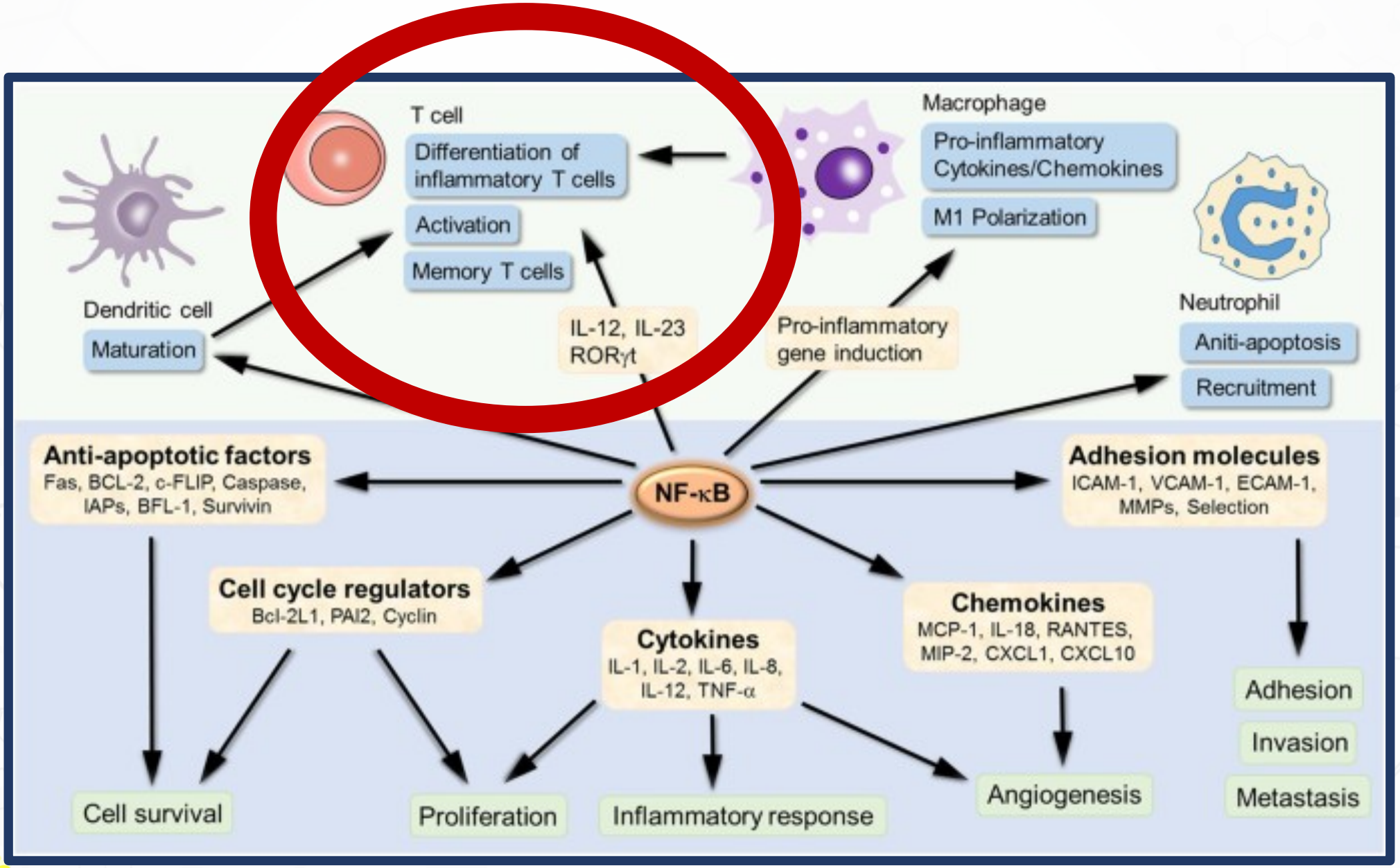
Noriko Komatsu [1], Hiroshi Takayanagi

Affiliations + expand

research has shown that CD4(+) T cells, especially IL-17 producing helper T (Th17) cells, play an important role in RA development. However, it still remains to be clarified how the systemic immune response results in the local joint disorders. Studies on animal models of RA have shed light on the importance of the interaction between immune cells and joint-specific mesenchymal cells. In particular, joint-specific mesenchymal cells contribute to the Th17-mediated augmentation of the inflammatory phase in RA by promoting the migration of Th17 cells to the inflammatory joint and then homeostatic proliferation with increase in IL-17 production. In addition, recent progress in osteoimmunology has provided new insights into the pathogenesis of the bone destruction phase in RA. Of note, Th17 cells have been shown to enhance the differentiation of osteoclasts via joint-specific mesenchymal cells. Thus, the interaction of CD4(+) T cells and nonhematopoietic mesenchymal cells in joints plays a key role in RA pathogenesis during both the inflammatory and bone destruction phases. Focusing on this interaction will lead to a better understanding of the mechanism by which the systemic immune response results in local joint disorders and also helps



https://www.researchgate.net/publication/283965918/figure/fig2/AS:329918396289024@1455670083081/Th17-cell-plasticity-T-helper-cells-differentiate-from-naive-T-cells-Th17-cells-are.png



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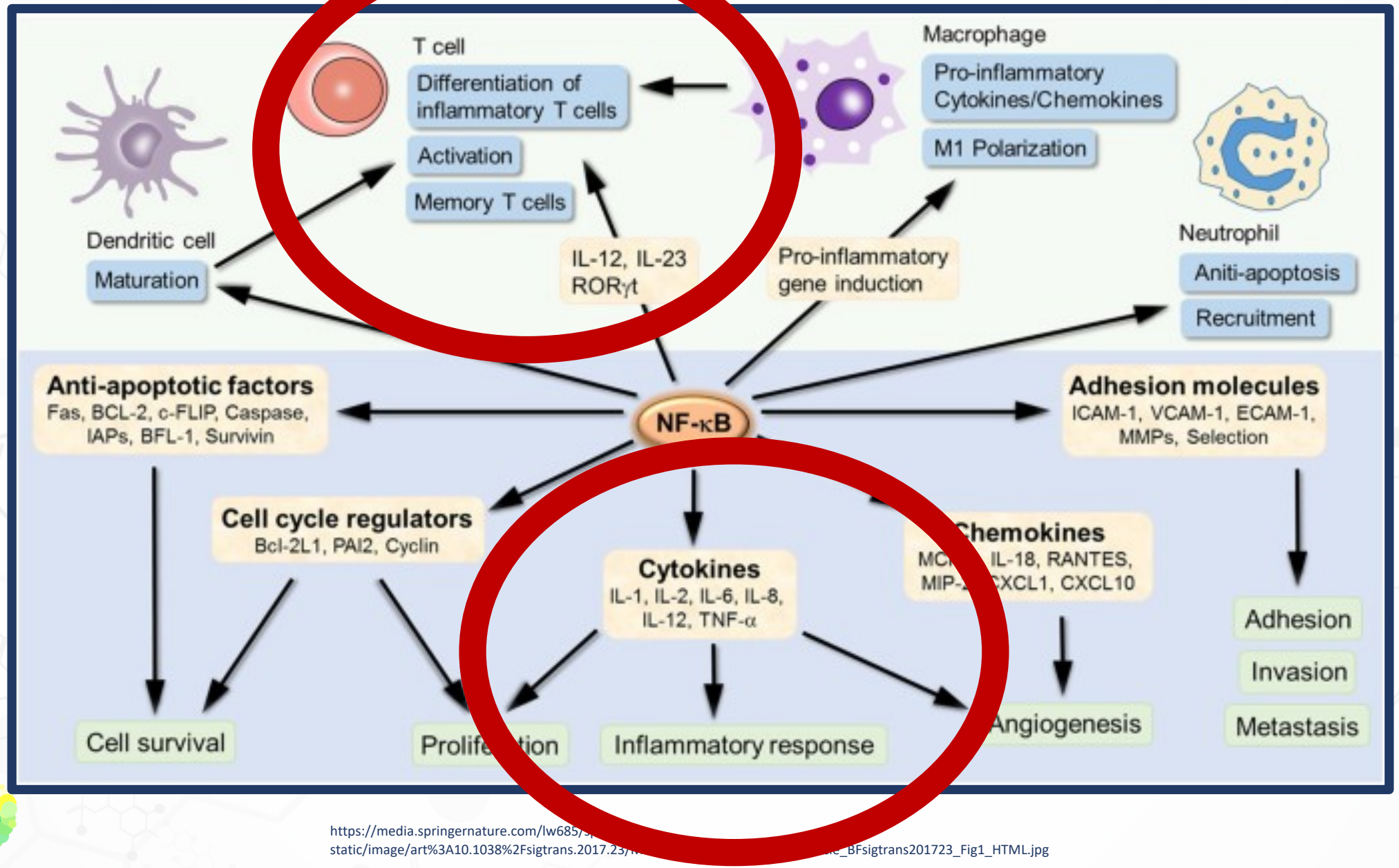

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Two Types of Inflammation

- NF-kb
  - Nuclear signaling molecule
    - Triggered by TNF-a and IL-1
      - Rapid Release upon Injury and/or infection. Indication of tissue damage.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2882124/

2 Types of Inflammation: Repetitive Use – 1 fire.

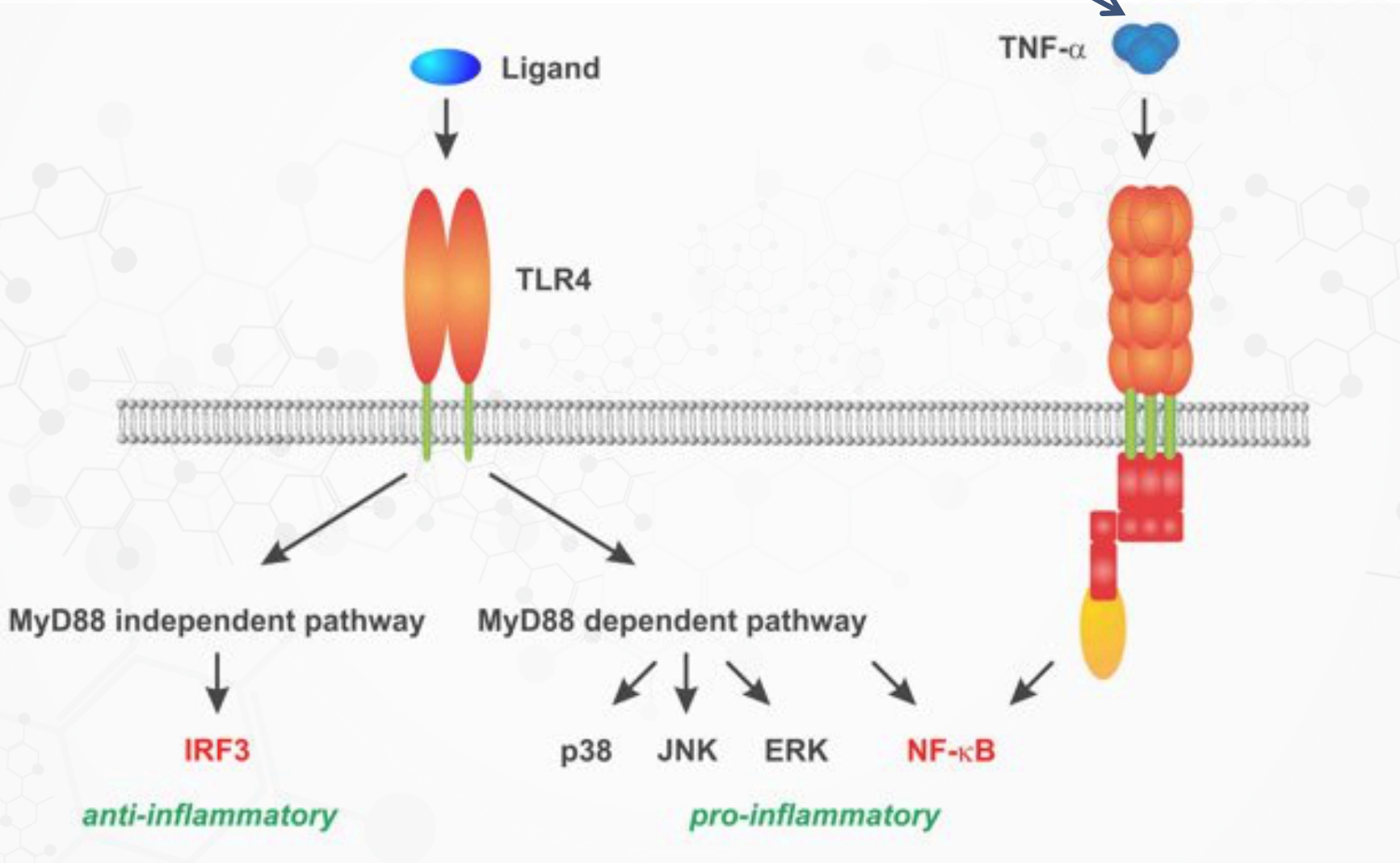
Standard American Lifestyle– All the fires.

Two Types of Inflammation

- LPS activation of NF-kb
  - Toll-like microbial pattern recognition receptors (TLR's)
    - TLR's are a non-self recognition system hardwired to trigger inflammation in the absence of injury or infection.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2882124/

LPS activates TLR’s, triggers TNF-a release from macrophage



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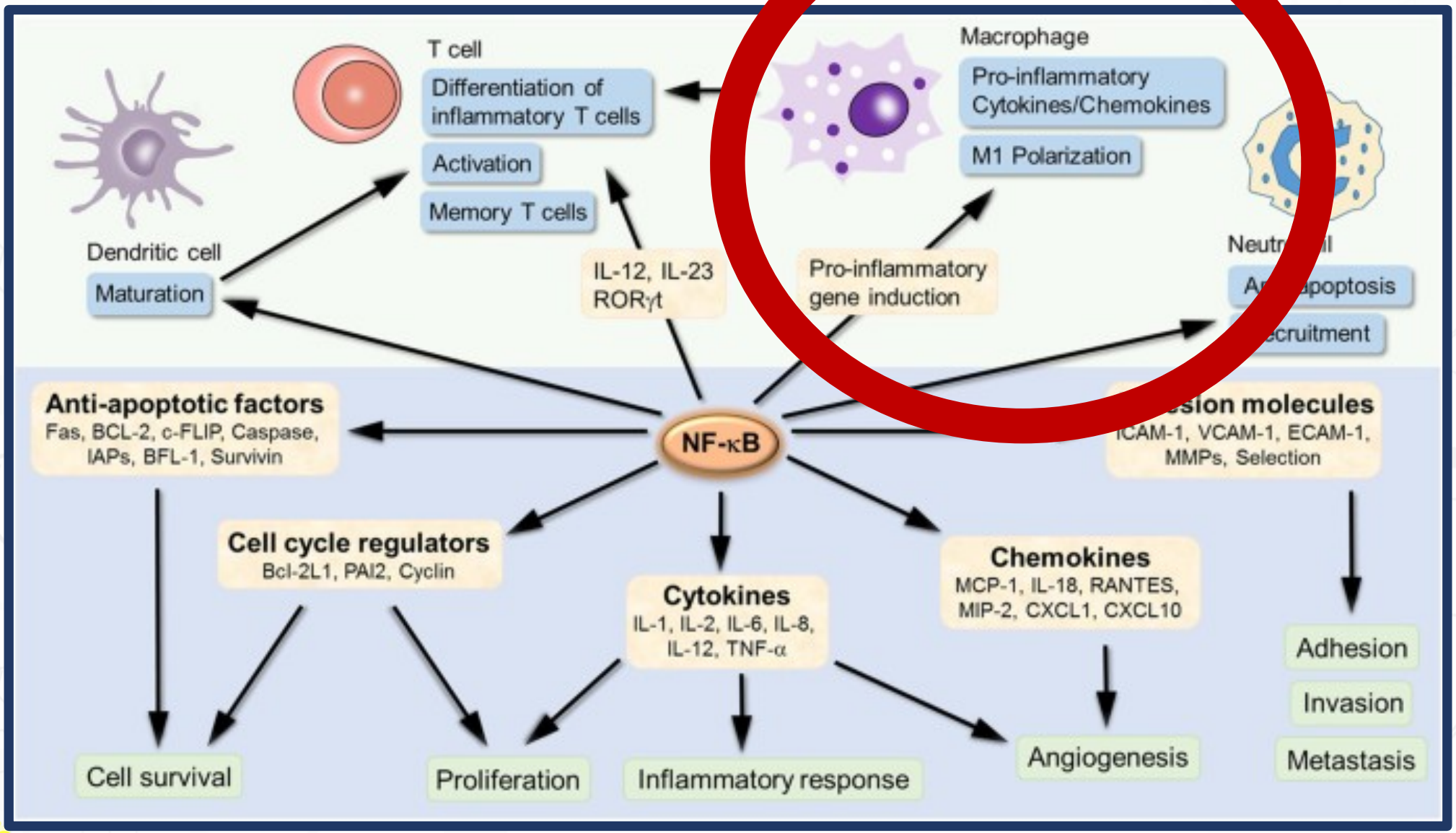

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Lipopolysaccharides

- 50% of the US population has full blown metabolic endotoxemia.
- LPS inherent in gram-negative gut bacteria (commensal).
- Endotoxemia from Non-genetic causes/Non-injury related causes.
- Lifestyle related!

https://www.ncbi.nlm.nih.gov/pubmed/26133659

https://www.dynamicchiropractic.com/mpacms/dc/article.php?id=58132

Fatty Acids and LPS Toxicity

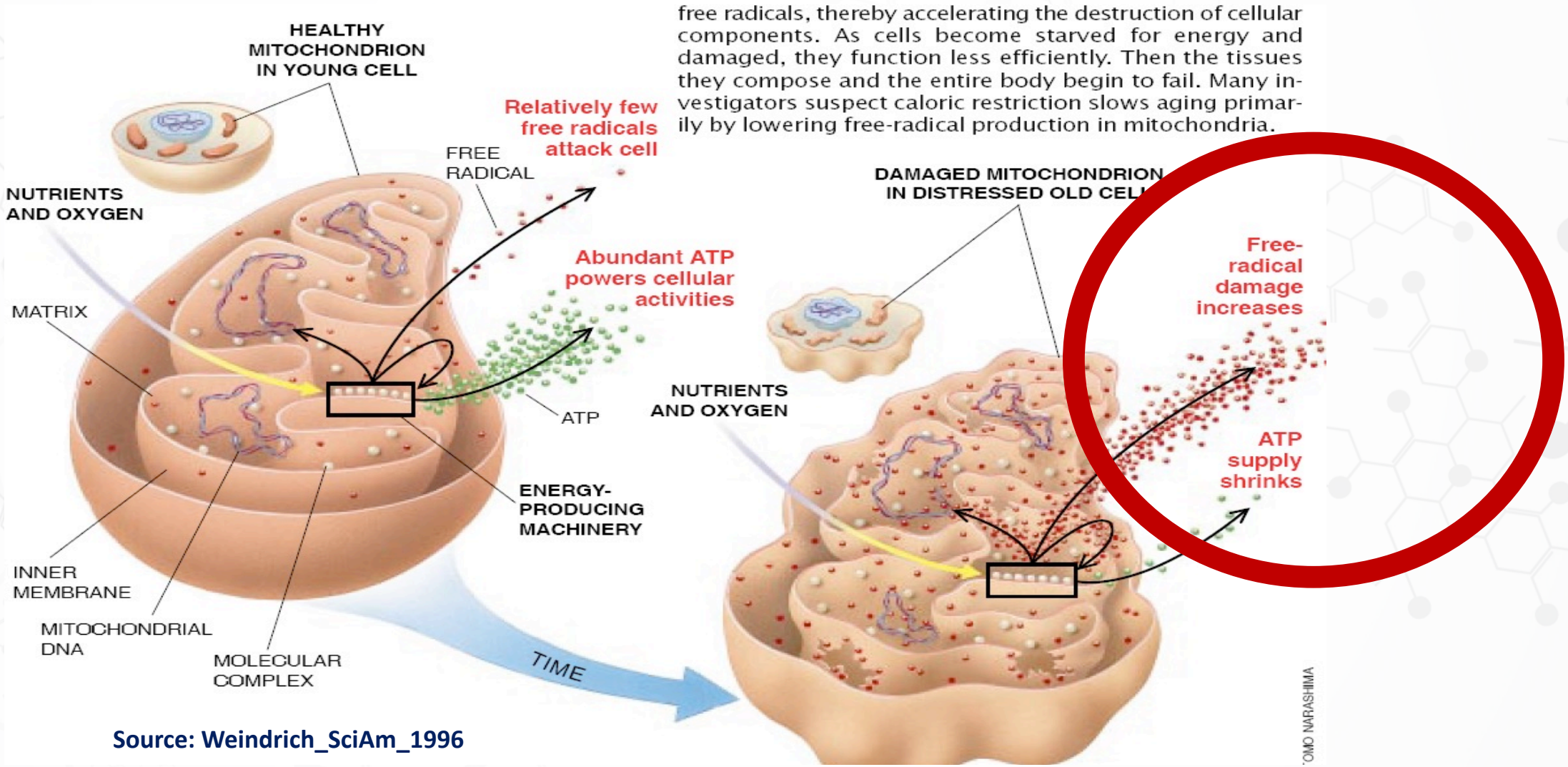
- When commensal bacteria use saturated fatty acids to form their outer membranes, they create a more toxic form of LPS.

Regenerative Support Goals

1. Create a local environment for therapeutic progress.
2. Decrease LPS.
3. Support healthy tissue genesis:
    - Enhance Collagen synthesis
    - Enhance Osteogenic activity
    - Support healthy synovial fluid
    - Support cell membrane health
4. Therapeutic maintenance.

Biogenetix Regenerative Support Kit.

Mitochondria Deteriorate with Stress



Source: Weindrich_SciAm_1996

Review > Joint Bone Spine. 2005 Mar;72(2):124-8. doi: 10.1016/j.jbspin.2004.01.007.

Musculoskeletal manifestations of scurvy

Olivier Fain [1]

purpura, bleeding, and gum abnormalities are the main symptoms. In 80% of cases, the manifestations of scurvy include musculoskeletal symptoms consisting of arthralgia, myalgia, hemarthrosis, and muscular hematomas. Vitamin C depletion is responsible for structural collagen alterations, defective osteoid matrix formation, and increased bone resorption. Imaging studies may show osteolysis, joint space loss, osteonecrosis, osteopenia, and/or periosteal proliferation.

> Cell Tissue Res. 2008 Oct;334(1):111-20. doi: 10.1007/s00441-008-0666-9. Epub 2008 Aug 5.

Regeneration of static-load-degenerated articular cartilage extracellular matrix by vitamin C supplementation

Garima Sharma [1], R K Saxena, Prashant Mishra

Affiliations + expand

PMID: 18679720 DOI: 10.1007/s00441-008-0666-9

cells were seen. Abundant pericellular and collagen fibrils were seen in this group of chondrocytes as compared with all other groups and the control. The results thus show that, in vitro, vitamin C supplementation of chondrocytes after static loading has the potential to reduce the morphological and biochemical degeneration of chondrocytes caused by static loading, thereby improving the cellular health and functioning of articular cartilage.

> Arthritis Res Ther. 2009;11(2):R43. doi: 10.1186/ar2651. Epub 2009 Mar 18.

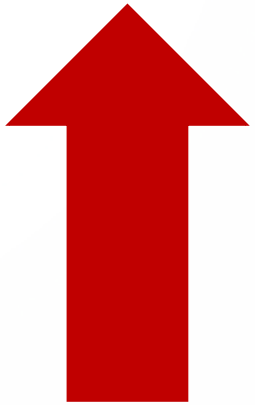
Oral phosphatidylcholine pretreatment alleviates the signs of experimental rheumatoid arthritis

Gabor Eros [1], Saleh Ibrahim, Nikolai Siebert, Mihály Boros, Brigitte Vollmar

Conclusions: Phosphatidylcholine-enriched food as pretreatment, but not as therapy, appears to exert beneficial effects on the morphological, functional and microcirculatory characteristics of chronic arthritis. We propose that oral phosphatidylcholine may be a preventive approach in ameliorating experimental rheumatoid arthritis-induced joint damage.

Standard American Lifestyle– All the fires.

Lifestyle + Genetics = Chronic Disease (RA)



2-Pronged Approach in this style of a case:

Root:
21-Day MCP
Fruit:
Regen Support Kit – Building Blocks.
UltraBiotix
Microbiocide GI
Binder Pro