



Monocytes are white blood cells that mature into macrophages. They are attracted to the inflammation by cytokines, such as IL-1 and TNF- α .



Macrophages, a key driver of inflammation, release large amounts of IL-1, IL-6 and TNF- α , which activate other white blood cells. Macrophages also clean up the inflamed area by ingesting microbes and dead cells.



Helper T cells perform a kind of coordinating command role in the immune system, releasing a series of cytokines that activate other immune-system cells.



B cells produce antibodies. In rheumatoid arthritis they make 'autoantibodies', which mistakenly assail the body itself instead of invading microbes.



Neutrophils are short-lived white blood cells that ingest whatever comes their way, such as the remains of dead cells or bacteria. They also release oxygen radicals and other tissue-damaging substances.

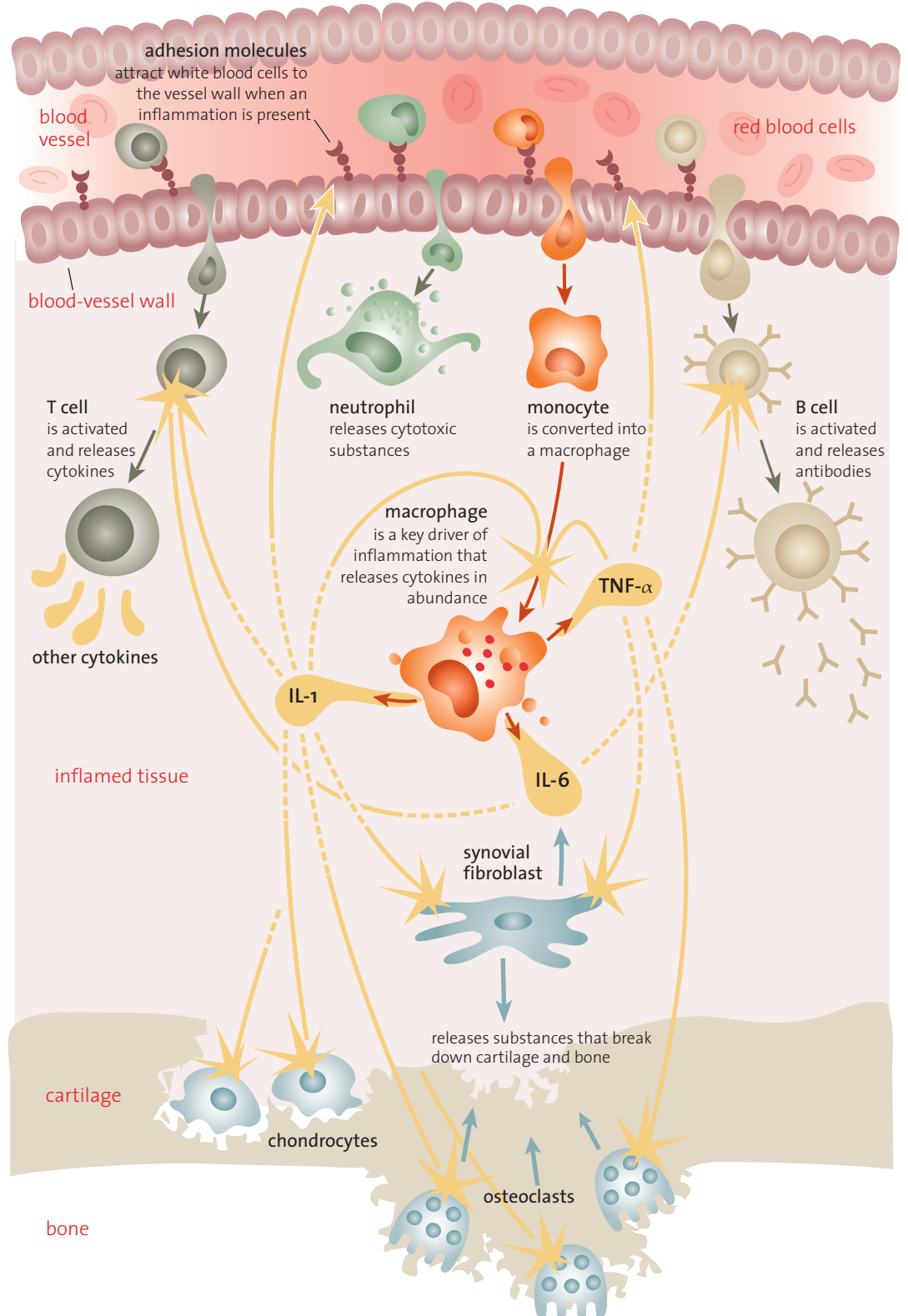


IL-1 and TNF- α attract white blood cells from the blood vessels to the inflamed tissue, and activate a range of cells. In addition, IL-1 raises body temperature.

IL-6 keeps the inflammation going by activating several different types of immune cell, and also contributes to a rise in body temperature. In the liver, IL-6 triggers production of 'acute-phase proteins', one effect of which is to exacerbate the inflammation.

Rheumatoid arthritis

A simplified depiction of the roles of IL-1, IL-6 and TNF in joint inflammation



Synovial fibroblasts are a type of connective-tissue cells that turn aggressive in rheumatoid arthritis. For example, they release IL-6 and substances that break down cartilage and bone.



Chondrocytes are cells that normally promote cartilage growth. IL-1 and TNF- α destroy chondrocytes or pervert them so that they break down cartilage instead.



Osteoclasts are cells that break down and resorb bone tissue.