The skin, mucous membranes and other organs (Montague et al, 2005). Older skin becomes thinner and drier, and its blood supply diminishes (Fig 1). As collagen and elasticity, becoming more vulnerable to cuts and abrasions (see article 11).

Elastin, becoming more vulnerable to cuts and abrasions. The number and quality of neutrophils to kill ingested microbes declines. Macrophagic ability of macrophages declines.

T-lymphocytes and cell-mediated immunity
T-cells leaving the thymus are stimulated to proliferate once they have bound with an antigen. They can differentiate into:
- Cytotoxic T-cells, which mediate the direct cellular killing of target cells;
- Helper T-cells, which help other cells, usually via the production of cytokines; supressor T-cells, which work by controlling the immune system;
- Memory T-cells, which store encounters with antigens.

Ageing affects the functions of T-cells. As the thymus atrophies with age, fewer T-cells are produced and the naive T-cell population is not replenished. Naïve T-cells are important T-cells that become active when exposed to foreign antigens, and then convert into dormant memory T-cells which reactivate on re-exposure to the same antigen. Older people have many memory T-cells but almost no naive T-cells, and cannot respond to new antigens as well as younger people.

T-lymphocyte cytokoty and proliferative response also decreases significantly with age. This is partly attributed to interleukin 2 (IL2), which is produced and secreted by T-cells to induce their proliferation and long-term growth. As T-cells age, they lose their capacity to produce and respond to IL2. Helper T-cells from older people also show altered profiles of cytokine secretion; they show a decreased responsiveness and helper function for B cell antibody production (Swain et al, 2005). Also, memory T-cells generated at a young age respond well to antigens over time compared with those derived in old age.

B-lymphocytes and antibody mediated (humoral) immunity
B-lymphocytes originate and mature in the bone marrow. They are activated by antigens, then become antibody-secreting plasma cells. These antibodies are known as immunoglobulins. They neutralise antigens and assist other components of the immune system in destroying non-self antigens. Some mature B cells become memory B cells, capable of ‘remembering’ specific antigen that triggered antibody production. The quality of the humoral (antibody) response declines with age, with older people having fewer B cells in the peripheral blood. They also produce less antibody in response to most foreign antigens. However, as most B-lymphocyte functions are regulated by T-lymphocytes and their cytokines, it is difficult to separate age-related decreases in B-lymphocytes from those due to T-lymphocyte defects. The clinical implication of decreased antibody response is significant when considering preventative healthcare such as vaccines.

AUTOMUNE CONDITIONS
Although the overall immune response to antigens decreases with age, immune reactivity increases. The percentage of auto-reactive T-lymphocytes and auto-antibodies increases with age, indicating a loss of tolerance to self. This is associated with common age-associated autoimmune conditions, such as rheumatoid arthritis.

REFERENCES