The anti-inflammatory effects of glucocorticoids are numerous, and endogenous production is one means of downregulating the inflammatory response when no longer required, in order to prevent unwanted (‘bystander’) damage to normal tissue.

In this context, glucocorticoids are antagonistic to:

● capillary blood flow and vasodilation (and therefore edema formation, and loss of protein and leukocytes from the vasculature into inflamed tissue and subsequent migration of these cells)

● vasoproliferation

● platelet aggregation

● fibrin deposition

● fibroblast proliferation and formation of collagen.

Glucocorticoids suppress the infl ammatory function of leukocytes (specifi cally granulocytes, mast cells and monocyte-macrophages) by stabilizing the membranes of these cells. This prevents release of infl amatory mediators such as histamine and arachidonic acid metabolites of both the cyclo-oxygenase and lipoxygenase pathways. They also inhibit particular metabolic pathways, e.g. production of proinfl ammatory cytokines such as IL-1, IL-6 and TNF-.

Glucocorticoids downregulate expression of Fc receptors on macrophages, thereby decreasing phagocytosis of opsonized particles (e.g. antibody-coated erythrocytes or platelets). The effects of glucocorticoids on neutrophil function are controversial. These drugs are generally considered to induce impairment of chemotaxis, adhesiveness, bacterial killing and lysosomal enzyme secretion. However, a recent study of neutrophil function in dogs administered prednisolone (1 mg/kg) has shown elevated IgG- or C3b-mediated phagocytosis, increased chemotaxis and chemiluminescence, and reduced adherence to nylon wool. Glucocorticoids may induce the characteristic blood leukocyte profile (neutrophilia, lymphopenia, monocytosis and eosinopenia) known as the ‘stress leukogram’.