



Figure 1. The phases of cutaneous wound healing. (a) Immediately following cutaneous injury, blood elements and vasoactive amines extravasate from locally damaged blood vessels within the dermis. Vascular permeability is temporarily increased to allow neutrophils [polymorphonuclear neutrophils (PMNs)], platelets and plasma proteins to infiltrate the wound. Vasoconstriction follows, in response to factors released by these cells. (b) Coagulation then occurs as platelets aggregate with fibrin, which is deposited in the wound following its conversion from fibrinogen. (c) Platelets release several factors, including platelet-derived growth factor (PDGF) and transforming growth factor β (TGF- β), which attract PMNs to the wound, signalling the beginning of inflammation. (d) After 48 h, macrophages replace PMNs as the principal inflammatory cell. Together, PMNs and macrophages remove debris from the wound, release growth factors, and begin to reorganise the extracellular matrix. (e) The proliferation phase begins at about 72 h as fibroblasts, recruited to the wound by growth factors released by inflammatory cells, begin to synthesise collagen. (f) Although the rate of collagen synthesis slows down after about three weeks, collagen crosslinking and reorganisation occur for months after injury in the remodelling phase of repair (**fig001ktl**).