uptake capacity of wound dressings prepared from plant-based wound dressings.

Synthetic polymers have been used widely in the preparation of wound dressings due to their excellent mechanical properties and flexibility. However, their use is limited by their poor biocompatibility and biodegradability. To overcome the aforementioned limitations, they are combined with biopolymers resulting in wound dressings with good features [10]. The loading of antibiotics into wound dressings is a good design approach suitable for the management of chronic wounds [6]. Antibiotics are useful in wound healing. The use of suitable concentrations of antibiotics for the treatment of infections is useful. However, high amounts of antibiotics can result in systematic toxicity [6]. To overcome the systematic toxicity of antibiotics in wound treatment, antibiotics are embedded in wound dressing which exhibit sustained and controlled drug release mechanisms. Some polymer-based wound dressings loaded with antibiotics for the treatment of microbial-infected wounds are hydrogels, foams, beads, dermal patches, films, nanoparticles, hydrocolloids, nanofibers, membranes, and others [7]. This chapter highlights the recent development of polymer-based wound dressings incorporated with antibiotics for the treatment of chronic wounds.

## 2. TYPES OF WOUNDS AND HEALING PHASE

Wounds are classified as chronic and acute wounds. Examples of chronic wounds are pressure ulcers, diabetic ulcers, and vascular ulcers. The common features of chronic wounds are infections, excessive inflammation, and poor capability of the dermal cells to respond to repairs [13]. Chronic wounds are characterized by features such as a high level of proteases resulting in the destruction of extracellular matrix (ECM) thereby preventing the wounds from moving into a proliferative phase and persistent infections [13, 14].

The altered activities of proteases which are common in chronic wounds hinder the wound-healing process. A high level of proteases has been reported in wound exudates of chronic wounds such as pressure ulcers. The high level of proteases results in the degradation of fibronectin, an important protein useful in the remodeling of ECM. The high level of proteases is also responsible for the degradation of selected growth factors [15]. The high levels of cytokines in chronic wounds impede the wound-healing process and causes changes in normal skin fibroblasts [15]. Suppression of the activation of macrophages which is useful for the release of cytokines and growth factors to induce cells such as keratinocytes, endothelial, and fibroblasts is reduced in chronic wounds resulting in a weak inflammatory response [16].

Chronic wounds are characterized by delayed re-epithelization resulting from the failure of migration of keratinocytes and the altered composition of ECM such as fibronectin. The fibroblasts are unable to respond to growth factors and reduce the synthesized amount of collagen [14, 17]. The overexpression of peroxynitrite, a free radical formed from the combination of nitric oxide with hydroxyl-free radicals also delays the healing of chronic wounds [18]. It affects inflammation, vasculature, and collagen deposition [18, 19]. In chronic wounds, slough and necrotic tissues accumulate in the wounds resulting from the altered cellular environment, poor blood supply, debris from dying cells, and others [20]. The slough is composed of pus, dead cells, fibrin, leucocytes, microorganisms, and protein materials [20, 21]. The accumulation of slough and necrotic tissues in chronic wounds enhances the colonization of bacteria which impedes the wound healing by extending the inflammatory response, hindering wound contraction and re-epithelization [20–22].

Acute wound healing involves an organized mechanism which is divided into four phases namely: hemostasis, inflammation, proliferation, and remodeling phase (Fig. 1) [23]. The hemostasis and inflammation phase occurs simultaneously. Hemostasis phase occurs immediately after injury whereby the blood clotting takes place to prevent excessive bleeding. The platelets are activated at the hemostasis phase by type 1 collagen resulting in the release of growth factors and glycoproteins which causes the platelet to aggregate. During the platelet aggregation, clotting factors are released with the deposition of a fibrin clot at the wound site.

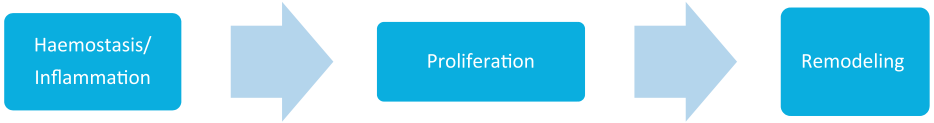


FIG. 1 Wound-healing phases.