PolyMem and Countering Inflammation made easy

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Introduction

Inflammation is the result of the immune system's response to localised damage. Acute inflammation results in local redness, heat, swelling and pain and resolves once the cellular debris and any foreign materials have been removed from the area. This is a normal, healthy response to exogenous cell death or tissue injury that is essential for re-establishing homeostasis and a prerequisite for tissue repair. Conversely, chronic inflammation is an unhealthy and persistent inflammatory response that results in an unabated change in tissue cellular composition and delayed healing. The early detection and treatment of excessive inflammation in wounds of individuals susceptible to uncontrolled or chronic inflammation is therefore important in reducing tissue damage and encouraging progression to healing. This Made Easy explains the causes and impact of inflammation on wounds, describes problems relating to excessive inflammation and outlines the roles of inflammation-managing dressings in containing and controlling a potentially unhealthy inflammatory response. It also specifically describes how the PolyMem range of multifunctional polymeric dressings work and the way in which they reduce and counter inflammation, thereby promoting healing.

Causes of inflammation

Wound healing consists of three overlapping stages: inflammation, proliferation and remodelling. Inflammation is the immediate and critical phase of tissue repair and healing (Gefen, 2018). The inflammatory response causes vasodilation, increasing both blood flow to the damaged area and vascular permeability, resulting in cells, clotting factors and protein-rich exudate leaking into the area around the damaged tissues (Cutting et al, 2015). Enhanced blood flow increases tissue perfusion and the amount of oxygen available for tissue repair processes. The increased osmotic pressure draws more fluid, containing cells and nutrients, to the injury site, resulting in local swelling and pain (Figure 1). The cells and clotting factors limit the spread of microbes, initiate the coagulation cascade, and release signalling molecules called cytokines that recruit immune system cells to remove bacteria and cell debris (Figure 1). As cytokines are released, they also contribute to redness (erythema), swelling, heat and pain (Cutting et al, 2015). Following debris removal, the inflammatory response should usually subside (Gefen, 2018).

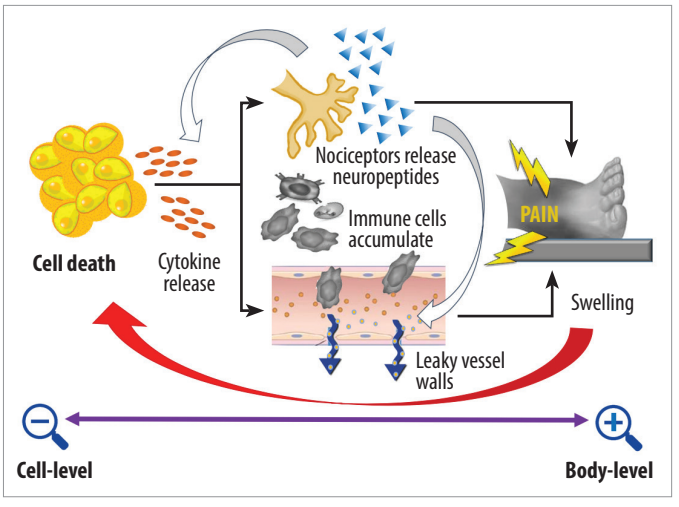

Figure 1: Inflammation from the cell-level to the body-level: Cell death causes release of cytokines that attract immune system cells from the vasculature to the damage site for clearance of cell debris and for resisting any invading pathogens. The released cytokines also stimulate nociceptors which amplify the inflammatory response via release of neuropeptides. While allowing extravasation of immune system cells from nearby blood vessels through relaxation of the vascular walls, cytokines and neuropeptides also increase the permeability of vascular walls which leads to leakage of plasma fluids, oedema and swelling at and near the initial damage site. The swelling further irritates nerve ends and therefore interacts with neuropeptide release, causing pain. The swelling also increases the interstitial pressures in the affected tissues, which potentially causes additional cell death, and so on and so forth

The peripheral nervous system (PNS) is also important in the healing process (Chéret et al, 2013). During early inflammation, free nerve endings (nociceptors) which use many of the same molecular pathways as immune cells, signal to local immune cells to initiate the immune response through the rapid release of signalling molecules called neuropeptides (Chiu et al, 2012). These neuropeptides trigger and disseminate the inflammatory process (see Box 1) causing increased local temperature, sensitivity to stimuli and pain as well as swelling and bruising (Beitz et al, 2004; Chiu et al, 2012; Ashrafi et al, 2016).

BOX 1. Role of neuropeptides in inflammation (Chéret et al, 2013)

Inhibit exocrine and endocrine secretion from the nervous system
Stimulate:
- nerve growth factor production and release
- inflammatory cytokine production and release
- vascular permeability and leakage
- local vasodilatation
- anti-inflammatory and anti-nociceptive actions

Promote the differentiation/proliferation/migration of:
- endothelial cells (which line blood vessels)
- fibroblasts (which generate connective tissue and link skin layers)
- keratinocytes (which form the outer skin layer)

Promote the formation of new blood vessels
Remodel granulation tissue

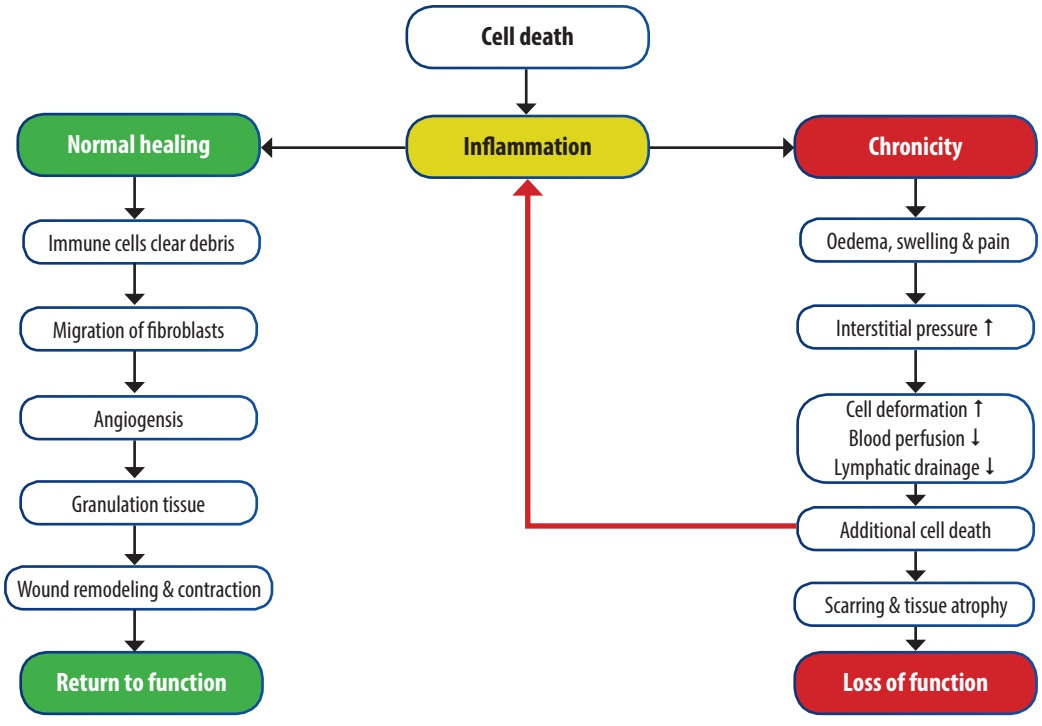

Figure 2: Inflammation is the critical juncture for the post-injury cascade of events: Progression to closure cascade (left) versus a maladaptive cascade, leading to chronicity (right)

Inflammation is the critical juncture of the post-injury cascade of events

Generally, there are two possible responses after injury: normal healing versus a maladaptive response leading to chronicity. Inflammation is the critical juncture where the post-injury cascade of events is determined: that is, whether the wound would progress to normal healing or to chronicity (Figure 2). The nature of the inflammation and associated swelling – including their intensity, spread, timing and time course – are central factors in the wound healing and 'fate' of the wound. The inflammatory response is promoted by the presence of cellular debris and pathogenic or infectious materials, which prevents proliferation, decelerates cell migration and hinders healing (Bell, 2010). Uncontrolled, persistent inflammation augments swelling, causes wider secondary cell death and tissue damage, primarily due to the high interstitial pressures associated with oedema, delays healing and increases scarring and atrophy (Davies and White, 2011) (Figures 1 & 2). Immune system dysfunction results in a chronic low-level inflammatory response with atypically high baseline cytokine levels that prevents the programmed cell death (apoptosis) cycle from stopping, resulting in continued cell death and intensified tissue damage, and a reduced resilience to cell damage and injury (Mason, 2011; Gefen, 2018).

Damage to the nervous system can also result in immune dysfunction and amplified or chronic inflammation, which includes defects in the phagocyte activity of the immune system cells (Chiu et al, 2012). This is observed in people with neuromuscular conditions, diabetes, brain trauma and spinal cord injuries (Chéret et al, 2013; Gefen, 2018). It also occurs in older people, as the number of nerve endings decreases as the skin ages (Ashrafi et al, 2016). Chronic inflammation activates a class of silent nociceptors in the PNS, causing them to respond to even minor stimulations. Their activity boosts nociceptive transmission in the central nervous system, increasing the perception of pain experienced from the damaged tissues (Mason, 2011).
For example, diabetes reduces nociceptor density and decreases neuropeptide release; without nociceptors to recruit immune cells to damaged tissues, an ulcer can develop that is hard to heal and at high risk of infection (Mason, 2011). The lower levels of various neuropeptides found in people with diabetes and neuropathic conditions have been linked to impairments in nerve growth, cell proliferation, migration and differentiation, granulation tissue remodelling and blood vessel formation in wounds as well as overall reduced immune responses (Chéret et al, 2013). In chronic wounds including pressure ulcers, sustained release of some neuropeptides lowers the threshold at which nociceptors are stimulated, increasing sensitivity and causing greater pain in the surrounding area (Davies and White, 2011; Ashrafi et al, 2016). Pain is a stimulator of nerve end

activities which interact with release of neuropeptides and the intensity and length of the inflammatory phase, and, hence, pain is an important predictor of healing time (Ashrafi et al, 2016). Pain is also known to cause psychological stress, which has been linked to impaired healing (Gouin et al, 2011).

Vasodilation during inflammation brings oxygen, immune cells, glucose and nutrients to the site of damage. Poor blood supply reduces the amount of oxygen available to perform numerous steps involved in the wound-healing cascade (Ashrafi et al, 2016). Poor tissue perfusion due to chronic hypotension, such as in the lower limbs of paraplegic or quadriplegic patients, prevents essential wound-healing components being delivered to the wound (Ashrafi et al, 2016). Circulatory problems therefore impair the healing process, prolong the inflammatory response, result in greater tissue damage, and increase the risk of a wound becoming chronic. Oedema associated with plasma fluids and exudate from acute inflammation stops the wound from drying out, aids cell movement across the wound bed, carries nutrients required for cell development, enables immune and growth factor diffusion, and aids the removal of dead cells and tissues (Cutting, 2003). This exudate is usually light coloured and decreases over time. However, excessive and prolonged oedema causes a considerable rise in interstitial pressures which increases the stiffness of tissues and the mechanical stresses developing in the tissues under bodyweight forces. The rise in tissue pressures further increases cell deformation levels and tissue distortions, particularly in tissues confined between bony structures and a support surface as in a person who is stationary in a bed or chair. The rise in interstitial tissue pressures also obstructs or may potentially even occlude blood and lymphatic vessels, which will further exacerbate the conditions in the swelled tissues (including acidosis, insufficient supply of metabolites and hormones, and deficient clearance of metabolic by-products) (Figure 2). Chronic non-healing wounds with high exudate levels usually present with abnormal inflammatory markers and there is an increased risk of pain, infection and odour (White & Cutting, 2006).

Inflammation and the role of dressings

Wounds have a negative impact on health-related quality of life and are associated with high healthcare costs. They should be assessed regularly, as their status may change over time in relation to fluctuations in inflammation, bacterial load and ischaemia. Dressings have a recognised role in managing inflammation, its associated symptoms (pain, swelling and exudate) and factors that inhibit progression to the next phase of healing (the presence of debris, devitalised tissue and microbes). The most appropriate dressing should be selected following a comprehensive assessment of the patient, and the wound. The identification of any underlying pathology together with factors that may impact on healing should be the primary objective. Failure to correctly evaluate wound progress may result in wound deterioration and/or the consequences of inappropriate treatment.

Wound inflammation and pain are inextricably linked within the physiology of healing (Cutting et al. 2015) and pain reduction is a top priority for many patients (Bell and McCarthy, 2010). Factors contributing to pain include the use of adhesive products, dried-out dressings, wound irrigation and anxiety or fear (Bell and McCarthy, 2010). Patients' pain should be assessed on presentation and the use of pre-emptive analgesia before dressing changes considered. Atraumatic, non-adhesive dressings with the potential to reduce background pain and minimise pain during dressing changes should be selected.

Excess exudate resulting from the inflammatory process needs to be absorbed and removed from the wound bed and periwound skin to prevent maceration. The presence of moderate or high volume exudate, which can be malodorous, is often distressing for the patient. It necessitates more frequent dressing changes and causes discomfort, as well as requiring increased clinician time to manage. The fluid-handling capacity of the dressing should be considered with the aim of providing a moist wound environment, avoiding strikethrough and minimising dressing changes to reduce the risk of trauma to the wound bed. Foam dressings, gel-forming fibrous dressing/alginates, superabsorbent dressings and negative pressure wound therapy are appropriate for the management of exudate.

When the inflammatory response is impaired, the prolonged presence of debris, eschar, devitalised tissue, callus and microorganisms including biofilm increases the risk of secondary tissue damage and infection. When the circulatory system is compromised, there is also an increased risk of ischaemia, and therefore devitalised or necrotic tissue formation. Debris and non-viable tissue need to be removed and the wound bed cleaned to encourage the development of healthy tissue, reduce the bacterial load and infection risk. Sharp (rapid) or autolytic (gradual) debridement, as appropriate, followed by cleansing is important when the patient first presents with a wound. A surfactant-containing wound cleanser is recommended (Baranoski and Ayello, 2008) and can be used to clean the wound bed at each dressing change. Alternatively, as cleansing is a major source of pain at dressing change and can disrupt the formation of new tissue, a dressing can be selected that aids autolytic debridement, as this process is atraumatic. The use of autolytic debridement with moist wound dressings is effective, for example, in the management of diabetic foot ulcers (Woo et al, 2013).

What is PolyMem and how does it work?

PolyMem dressings are multifunctional polymeric membrane dressings consisting of four components (see Box 2 and Figure 2). The cleanser, moisturiser and superabsorbent starch co-polymer are contained within the hydrophilic polyurethane matrix. This is covered with semi-permeable backing film (which is not included in cavity products). While the components have specific actions as detailed below, they interact synergistically with the others in all aspect of the wound healing process to deliver clinical benefits beyond what each could achieve individually: the outcome is greater than the sum of

the contributions of each individual component. As an example, all the components play both primary and supporting roles in creating the continuous wound cleansing system provided by the PolyMem family of dressings.

BOX 2. Some primary roles of the components within PolyMem dressings (Cutting et al, 2015; White et al, 2015)

Wound cleanser: Surfactant is continuously released into the wound bed after the dressing is applied. It helps loosen the bonds between wound debris and healthy tissue, assisting with the removal of eschar and necrotic tissue and supporting autolytic debridement. This minimises the need for manual wound cleansing.

Moisturiser: Glycerine is released at the same time as the cleanser, moisturising the wound bed and preventing dressing adherence. This reduces pain and trauma to the wound bed and periwound skin at dressing change. Glycerine draws fluid containing nutrition and growth factors from the deeper tissue, stimulating healing in the wound bed.

Superabsorbents: These absorb and bind excess exudate within the dressing, helping balance moisture levels and reducing the risk of maceration. They draw enzymes, nutrients and white blood cells into the wound bed, aiding repair and new tissue development.

Semi-permeable membrane: The thin film backing protects the wound from the environment and allows evaporation of excess exudate, optimising moist wound-healing conditions.

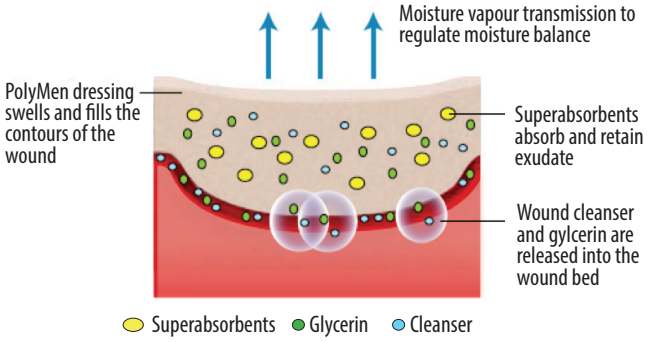

Figure 3: Structure and function of the PolyMem dressing

Indications

PolyMem products offer a single formulation that can be used as primary and/or secondary dressings throughout the wound-healing continuum. The range is indicated for a variety of acute and chronic wounds including, but not limited to, full- and partial-thickness wounds, skin tears, pressure ulcers, leg ulcers, diabetic foot ulcers, fungating wounds, superficial and partial thickness burns and surgical wounds.

How PolyMem tackles inflammation

Inflammation may develop rapidly and spread to healthy tissues surrounding the site of initial damage. If persistent, the inflammation and oedema damage cells and tissues in the injury spiral described above (Figure 1 & 2), increasing pain and delaying healing (Cutting et al, 2015). PolyMem manages and contains the inflammatory response at the initial wound site and reduces inflammation in the surrounding tissues (Beitz et al, 2004). This action decreases bruising, swelling and secondary injury, reducing wound sensitivity to touch and manipulation (Cutting et al, 2015; Benskin, 2016).

Wound fluid is drawn into the wound by PolyMem dressings, ensuring adequate hydration of the wound bed and thus supporting healing (Benskin 2016). The removal of excess fluid decreases the impact of oedema, reducing further potential damage associated with sustained cell deformation and tissue distortion, and prolonged obstruction of the vasculature and lymphatic system. The removal of excess fluids also reduces pain and alleviates its psychological effects (e.g. depression), as well as the risk of maceration.

Reducing or easing inflammation disrupts changes in the pain-signalling pathways caused by long-term inflammation. PolyMem dressings diminish nociceptor activity in the skin, reducing various symptoms of inflammation and the potential unnecessary amplification of the inflammatory process (Kahn, 2000; Beitz et al, 2004). Its mode of action supports the repair of cellular damage under intact as well as damaged skin. Specifically, the dressings appear to reduce the chronic inflammation that occurs in neuromuscular conditions and in older people, and to increase the local sensitivity of the immune system (Gefen, 2018). PolyMem may therefore be suitable for preventative use on at-risk sites, such as the sacrum or heel, in vulnerable patients (Gefen, 2018).

Evidence for PolyMem

Evidence from laboratory as well as clinical studies demonstrates that PolyMem dressings focus the inflammatory response at the primary site of tissue damage and dampen nociceptive activity in the epithelium, reducing swelling, pain, itching and burning and importantly, shifting a wound from chronicity to a normal healing path (Figures 1 & 2) (Kahn, 2000; Beitz et al, 2004; Weissman et al, 2013). Significant reductions in the visible effects of inflammation, oedema and bruising have been reported in human and animal studies (Kahn, 2000; Hayden and Cole, 2003; Beitz et al, 2004; Schmid, 2010). PolyMem use resulted in reduced postoperative pain scores and lower increases in skin temperature compared to a standard dressing (Hayden and Cole, 2003).

In addition to antinociceptive properties, PolyMem has an analgesic effect. Even though it is only applied to the skin, PolyMem appears to affect neuropeptide signalling, facilitating better control of inflammation in tissues as deep as the skeletal muscle by decreasing nerve activity in the spinal cord, which reduces nociceptor sensitisation (Beitz et al, 2004). By interacting with the central and PNS to moderate neuropeptide signalling, triggering the clearance of inflammatory mediators, PolyMem modulates the amplification of the inflammatory phase (as described in Figure 1), decreases pain and swelling at the

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Keith Cutting acts as a consultant to multiple wound care companies, including Ferris Mfg. Corp. Professor Amit Gefen acts as a scientific advisor to multiple wound care companies, including to Ferris Mfg. Corp. whose PolyMem® dressing technology is reviewed in this article. This had no influence on the conclusions from the analysis of literature which is presented here.

Table 1. Summary of published studies demonstrating PolyMem's impact on inflammation

Reference	Title	Type	Purpose	Key findings
Weissman O, Hundeshagen G, Harats M (2013) *Burns* 39(6): 1316–20	Custom-fit polymeric membrane dressing masks in the treatment of second-degree facial burns	Case series	Investigate the use of a polymeric membrane face mask in managing second-degree burns (*n*=8) and comparison with a historical cohort of patients with facial burns treated with antibiotic ointment	Inflammation was confined to the actual wound site. Reduced time to full epithelialisation (6.5 days versus 8.5 days). Low pain ratings (2.6 versus 4.7) resulting in pain-free dressing changes.
Kahn A (2000) *Pain Med* 1(2): 187	A superficial cutaneous dressing inhibits inflammation and swelling in deep tissues	Animal study and human case study	Investigate the effect of a superficial cutaneous dressing (PolyMem) versus a control bandage on deep tissue response following mechanical trauma in rabbits (*n*=14)	Significant reductions in the visible effects of inflammation, oedema and bruising (rabbits and human). No pain or limitation of movement following knee surgery and resumption of normal activities 2 days after surgery with PolyMem use in the case study. Evidence suggests PolyMem inhibits nociceptive activity in the epithelium, blocking the central nervous system response that generates swelling, inflammation and pain.
Beitz AJ, Newman A, Kahn AR et al (2004) *J Pain* 5(1): 38–47	A polymeric membrane dressing with antinociceptive properties: analysis with a rodent model of stab wound secondary hyperalgesia	Clinical evaluation	To evaluate rodent pain responses to mechanical and thermal stimuli following the application of polymeric membrane dressing (PMD) versus gauze dressing to stab wounds	Significant reductions in mechanical and thermal sensitisation. No decrease in activity following injury. Reduced spread of inflammation in the deep muscles by 25%, even though only applied to the skin. White blood cells concentrated within the injured area. Reduced spinal cord Fos expression modifying peripheral and central nervous system response, resulting in local analgesia.
Hayden JK, Cole BJ (2003) *Orthopedics*26: 59–63	The effectiveness of a pain wrap compared to a standard dressing on the reduction of post-operative morbidity following routine arthroscopy	Clinical evaluation	Evaluation of a pain wrap dressing in patients (*n*=24) undergoing routine knee arthroscopy to determine its ability to decrease post-operative pain and swelling	Less post-operative swelling. Lower pain ratings (2.2 versus 4.6). Lower increases in skin temperature (0.6C [1.1F) versus 2.1C [3.9F]).

injury site, resulting in in local analgesia, reducing potential secondary inflammatory injury (Figure 2) and helping shift the injury response towards progression to closure. This is a primary feature in the unique mechanism of action of PolyMem since any secondary injury response associated with over-inflammation and oedema – which may delay or even block healing – must be minimised before tissue repair can occur (Beitz et al, 2004; Gefen, 2018).

Observational and clinical studies report that the application of PolyMem results in the swift resolution of open wounds and of damage in intact tissue (Kahn, 2000; Schmid, 2010; Wilson, 2010). Winblad and Harvey (2010) reported that 78% of 103 clinicians surveyed 'strongly agreed' or 'agreed' that wounds heal faster with PolyMem. Its application after knee surgery led to short healing times in a number of case studies (Kahn, 2000; Schmid, 2010). Wilson (2010) reported that 80% of category 1 pressure ulcers resolved within 4 days of PolyMem being applied compared to the typical 2 weeks with standard care protocols. Finally, in patients with moderate to severe ischaemia associated with lower-extremity arterial disease, PolyMem significantly reduced the proportion of deep tissue pressure injuries (DTPI) that opened compared to skin barrier film (45% versus 83.4%) (Henson, 2019). Evidence from clinical studies, case reports and poster presentations demonstrates PolyMem reduces inflammation, relieves pain and facilitates healing.

Tips in practice

- For injured, intact tissue or dry, non-exuding wounds, moisten the wound slightly or moisten the dressing with a few small drops of saline or water prior to application. This will help to activate the dressing components. Do not saturate the dressing. The dressing should cover beyond any inflamed, tender, painful, warm, itchy or otherwise damaged area surrounding the open wound
- Following application, mark the open wound margin on the outside of the dressing so you can monitor exudate absorption and avoid disturbing the wound except at dressing change
- The wound may appear larger at the first few dressing changes as a result of debridement; this is a normal part of healing
- Do not occlude PolyMem with excess tape or bandage, as this will reduce the dressing's ability to draw wound fluid – and therefore nutrients and repair cells – into the wound bed
- It is recommended to read the enclosed *Instructions For Use* of all medical products prior to initial use and to periodically review because they may change over time as more information is learned by the manufacturer

Summary

The inflammatory process is an essential component of tissue repair that is controlled by the immune system and PNS. The release of signalling molecules, cytokines and neuropeptides from immune and nerve cells, respectively, triggers the healing cascade, containing injury and repairing tissues. When this process is maladaptive, a secondary injury may occur resulting in loss of tissue function leading to a high risk of hard-to-heal chronic wounds. In individuals susceptible to maladaptive responses to tissue injury such as the elderly, diabetic, those with a compromised immune system and persons with central nervous system injury, focusing and controlling inflammation with appropriate dressings aid's the body's ability to move towards healing. Evidence shows that PolyMem manages and contains the inflammatory response and dampens nociceptor response, thereby reducing inflammation in tissues surrounding the initial site of injury as well as reducing pain, bruising, swelling and secondary injury, and facilitating healing.

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