Drawtex: breaking the vicious circle of cellular and molecular imbalances

Annemarie Brown, Mandy Yorke

Annemarie Brown is Independent Consultant, Tissue Viability Solutions; Mandy Yorke is Clinical Manager, Solent NHS Trust Podiatry Team

Email: abrowne61@aol.com

ABSTRACT

Uncontrolled cellular and molecular activity in the inflammatory phase of healing will determine whether a wound becomes chronic. Assessment and interventions designed to remove the barriers to healing are essential in order to break the vicious cycle and to kick-start healing in chronic wounds. This product focus gives an overview of the inflammatory phase of the wound healing continuum; discusses how the imbalance of matrix metalloproteinases/tissue inhibitors of matrix metalloproteinases occurs; how this imbalance manifests itself clinically within the wound; and what health professionals can do in order to tip the balance in favour of healing. It discusses a new wound dressing, Drawtex, which combines three modes of action to maintain a moist wound environment in order to debride, manage exudate and reduce bioburden by locking harmful proteases into its core. Case studies are presented where Drawtex has been used to achieve healing in wounds that were not responding to treatment.

KEY WORDS

- Cellular imbalance
- Chronic/non-healing wounds
- Modulating dressings
- Multiple functions

The NHS needs to save £15–20 billion by the end of 2013/14 to reinvest into frontline services in order to manage the increasing health costs of an ageing population (Gray, 2013). The annual overall cost of wound care in the UK has been estimated at £2–3 billion, or about 3% of the total NHS (Posnett and Franks, 2007), and this may be seen as an area for potential saving. In addition, the Quality, Innovation, Productivity and Prevention (QIPP) agenda introduced by the Department of Health (DH) in 2010 sets out a challenge for clinicians to contribute to these savings by improving productivity and eliminating waste while ensuring clinical quality and placing the patient at the centre of care (DH, 2010a; DH, 2010b).These factors put considerable pressure on health-care professionals (HCPs) to develop wound care formularies based on both cost effectiveness and product efficacy. Using products with multiple functions may be a pragmatic way to reduce dressings costs.

Wound care spend

The highest proportion of the wound care spend is on the management of chronic wounds (Posnett and Franks 2007) since, as a general rule, acute wounds such as skin tears, surgical wounds, lacerations, dog bites etc tend to heal rapidly and uneventfully. Lazarus et al (1997) defined an 'acute' wound as

> ***'one that proceeds through an orderly and timely reparative process, resulting in the restoration of function and skin integrity.'***

Chronic wounds, however, fail to proceed in this manner and have been described by Enoch and Price (2007) as those that 'heal slowly, heal and recur or never heal at all' or those that

> ***'do not respond to treatment or have an underlying aetiology which has not been corrected.'***

Wound healing continuum

The wound healing process is best described as a cascade of events set in motion by a series of physiological responses as a result of an injury or trauma (Bryant, 2000). Although often described in three or four distinct phases for explanatory purposes, in reality these phases often overlap and form a continuum that is referred to as the healing cascade (Martin, 2013).

In order to distinguish between acute and chronic wounds, the normal healing process will now be described with particular emphasis on the inflammatory phase, where abnormal or uncontrolled cellular activity will determine whether a wound heals uneventfully or develops into a chronic wound.

The inflammatory phase in wound healing

The inflammatory phase is initiated as a normal body response to an acute injury (Bryant, 2000), and the goals of this phase are to control cellular injury and blood loss and to establish a clean wound bed in preparation for healing. Tissue injury and the activation of clotting factors to achieve haemostasis stimulate the release of vasoconstrictive substances such as prostaglandins and histamine, causing local vasodilation and increased capillary permeability (Martin, 2013). This can be

seen at the wound site as erythema, oedema, heat and an increase in wound fluid within 10–15 minutes (Bryant, 2000).

Chemical messengers are released from the platelets and mast cells, including platelet-derived growth factor (PDGF), which attracts neutrophils to the damaged tissue and signals them to proliferate (Woods et al, 2010). The white blood cells that were rushing through the blood vessels now slow down and accumulate on the lining of the dilated blood vessels. These white blood cells then squeeze through the porous walls of the blood vessels and migrate into the wound space that has already been filled with a fibrin clot (Davis, 2008). Once within the wound, the neutrophils help fight off potential infection and are responsible for cleaning up the wound by ingesting bacteria and devitalised tissue (Martin, 2013).

During this inflammatory phase of wound repair, neutrophils release free radicals and matrix metalloproteinases (MMPs) into the wound. These have a bactericidal function and promote autolytic debridement to remove the damaged cells and tissue (Gibson et al, 2009). Once they have completed their job, typically after about 3 days, the neutrophils die and can be seen on the wound surface in the form of slough, which is moist, sticky, stringy and typically a yellow/creamy colour (Martin, 2013). The death of the neutrophils signals the arrival of macrophages that remove any remaining neutrophils and continue to release MMPs and inflammatory cytokines that prolong the inflammatory process (Wolcott et al, 2008). The wound cannot progress into the next phase of healing until there is an equal balance between the number of MMPs and tissue inhibitors of matrix metalloproteinases (TIMPs) secreted by the macrophages in the wound in order to replace the fibrin matrix with granulation tissue (Percival and Cochrane, 2010). See *Table 1* for more details.

Table 1. Definition of key regulators in the inflammatory phase

Regulator	Origin	Function
Growth factors e.g. platelet derived growth factor; transforming growth factor beta, basic fibroblast growth factor, keratinocyte growth factor	Macrophages	Proteins that act like hormones binding to specific cell receptors to alter the cell's function. Stimulate proliferation and migration of fibroblasts and keratinocytes
Cytokines, e.g. interleukin–1, tumour necrosis–α, interferons	Macrophages	Substances other than cytokines. Combine with cytokines to 'fine tune' the wound repair process
Matrix metalloproteinases (MMPs)	Neutrophils, macrophages, fibroblasts and endothelial cells in response to cytokines growth factors and TIMPs. MMP-1, 2, 8 and 9 associated with chronic wounds	Enzymatic compounds capable of degrading type 1 and type IV collagen. Break down basement membrane during inflammatory phase and contribute to neoangiogenesis during proliferative phase and remodelling of extracellular matrix in maturation phase
Tissue inhibitors of matrix metalloproteinases (TIMPs)	Produced by cells within the wound and also serum	TIMPs bind to MMPs rendering the MMP inactive. The production of MMPs and TIMPs is coordinated to achieve desired level to achieve wound healing

While they are crucial in all phases of the wound healing continuum, if the MMPs are not down-regulated by TIMPs during the inflammatory phase, the excessively high number will result in the continued degradation of the growth factors and cells necessary for new growth, such as fibroblasts and keratinocytes (Gibson et al, 2009). There is increasing evidence that the levels of MMPs are greatly elevated in chronic, non-healing wounds (Trengrove et al, 1999; Pirila et al, 2007; Moor et al, 2009).

Factors that affect the cellular balance of the wound

While the majority of wounds progress uneventfully through this process, some wounds become stuck in the inflammatory phase as a result of the cellular imbalance. An imbalance of the levels of MMPs and TIMPs can result in increased degradation of fibronectin, various growth factors and the resultant excess breakdown of tissue occurs within the wound (Bryant, 2000). This results in a prolonged inflammatory phase and, ultimately, a chronic, non-healing wound.

Wound exudate

Wound exudate has an important function in normal wound healing as it provides a moist environment, enables diffusion of growth factors, provides essential nutrients and facilitates autolytic debridement of devitalised tissue (Martin 2013). It is, however, altered by wound aetiology and biochemical changes within the wound environment (for example, infection) and, therefore, the composition of exudate from healing and non-healing wounds differs (Wysocki et al, 1993; Trengrove et al, 1999). Wound exudate, particularly from chronic wounds, contains a cocktail of elements (cellular debris and enzymes), which can be very corrosive to the wound bed and intact skin surrounding the wound (Bianchi, 2012).

Trengrove et al (1999) found that, in wound fluid from acute surgical wounds compared with the wound fluid in non-healing wounds of varying aetiologies, MMP levels were 30 times higher in the wound fluid of the chronic wounds (Trengrove et al, 1999). Various studies have found that, as a wound heals, protease levels (which are predictive of healing) fall, exudate levels diminish and the exudate composition changes (Wysocki et al, 1993; Trengrove et al, 1999).

As well as causing problems within a wound, such as breakdown of the extracellular matrix, exposure to MMPs can damage the periwound area as the proteins enveloping

the corneocytes are destroyed, affecting the epidermal barrier function (Langoen and Bianchi, 2013). This can result in a red and 'weeping' skin surface. Additionally, the cytokines in chronic wound fluid cause damage to the stratum corneum, further reducing skin barrier function (Wolcott et al, 2008). Therefore, effective exudate management is key to preventing further wound breakdown and peri-wound maceration.

Bioburden, biofilms and infection

The normal host response to bacterial contamination of a wound is an inflammatory response that allows white cells to infiltrate and clean the wound in an effort to prevent infection. However, if the number of invading pathogens is excessive, wound healing will be delayed and a wound infection may result (World Union of Wound Healing Societies (WUWHS), 2008; Siddiqui and Bernstein, 2010).

Bacteria in chronic wounds form micro colonies, known as biofilms, which are formed from multiple species of bacteria. The bacteria species within biofilms are dynamic, can adapt and alter their structure and are very resistant to normal host responses, killing by inflammatory cells, antibiotics and disinfectants (Costerton et al, 1999; WUWHS, 2008; James et al, 2008; Edwards-Jones and Flanagan, 2013). Recent research suggests that approximately 60% of wounds with delayed healing have bacterial biofilms and that the inflammatory cells activated in response to the biofilm release free radicals and proteases, including MMPs, in an attempt to deactivate and destroy the bacteria. Unfortunately, the proteases also destroy pro-healing factors and extracellular matrix components in the wound bed, disrupting the wound healing process.

As a result, more exudate is produced, resulting in a toxic corrosive 'soup', which results in wound deterioration. Gibson et al (2009) refer to this as a 'vicious circle' and suggest that the clinician needs to break out of the circle by alleviating any environmental, systemic, local and wound-related factors that might contribute to the delay in healing. The overall aim is to tip the balance in favour of the repair processes. They suggest that this can be achieved in the following ways (adapted from Gibson et al, 2009):

- Treating the cause of the bioburden by reducing the inflammation.
- Debriding slough/devitalised tissue or removing pus within the wound since these will encourage multiplication of microorganisms (Edwards-Jones and Flanagan, 2013). Maintenance debridement may be required to prevent accumulation of slough.
- When indicated, wound bioburden can be reduced using hydroconductive debridement dressings (e.g. Drawtex), antimicrobial dressings (e.g. silver- or iodine-based technologies) and antibiotics (WUWHS, 2008). However, antibiotics and antimicrobials are less effective at treating bacteria in a biofilm, and physical removal by debridement is currently the only demonstrated method for removing biofilm burden.
- Managing the consequences of debridement by using a wound product such as Drawtex reduces protease activity, manages excess moisture, reduces bioburden and debrides devitalised tissue.
- Removing proteases, e.g. by absorption of protease-rich wound fluid into dressings; by removal with negative pressure wound therapy or the use of protease-modulating products.

Assessing the wound bed: TIME framework

One of the most popular wound bed assessment frameworks is wound bed preparation (WBP) (Schulz et al, 2003), which is a conceptual framework for managing chronic wounds at a cellular level (Sibbald et al, 2000; Sibbald et al, 2007; Moffatt et al, 2004). Designed as a practical decision-making tool to optimise wound care practice, Schultz et al (2003) further developed the concept into the acronym TIME. The framework is comprised of four components, each of which needs to be considered when assessing wounds (Brown and Flanagan, 2013). The four components are:

- T—tissue, non-viable or deficient
- I—inflammation and infection
- M—moisture balance
- E—epithelial (edge) advancement.

The principle of the framework is that, by working systematically through each individual component, the practitioner will be able to prioritise and implement interventions aimed at removing all potential barriers to healing in chronic wounds (Brown and Flanagan, 2013).

The original WBP model placed the emphasis on the physiological and molecular barriers to healing at an advanced level and attracted some criticism in that it focused practitioners (particularly those inexperienced in wound care) to assess the wound in isolation (Brown and Flanagan 2013).

Since then, the importance of addressing patient-centred factors such as lifestyle, adherence to treatment and the pyschosocial needs of patients has been incorporated into the model (Inlow et al, 2000; Falanga, 2004; Harding et al, 2008; Dowsett, 2009). These factors are important since there is a large body of evidence to demonstrate that living with a chronic wound has a major impact on patients in terms of reduced quality of life (Price et al, 2004; Maddox, 2012), poor body image and self-esteem (Mudge et al, 2006), depression (Jones et al, 2006), feelings of hopelessness and powerless (Salome et al, 2013) and their sense of wellbeing (Wounds International, 2012). All these patient factors will need to be addressed in order to achieve a concordant relationship.

The TIME framework is now widely acknowledged to help rationalise the implementation of advanced treatment modalities and monitor progression to healing (Moffatt et al, 2004; Dowsett and Newton, 2005; Mulder, 2009; Ousey and McIntosh, 2010). In addition, it has been used successfully as a teaching tool in wound care education (Dowsett, 2009). It should be noted that one dressing in isolation may not address all the aforementioned factors. Accurate assessment, together with an understanding of the healing process, is required to enable clinicians to choose products wisely. Drawtex, however, is a product that is

suitable to address all of the barriers to healing that have been identified in the TIME framework.

Drawtex: clinical indications and mode of action

This article now describes the Drawtex product, which is a hydroconductive debridement wound dressing combining hydroconductive action, electrostatic action and capillary action.

Hydroconductive action

Hydroconductive action is controlled by Darcy's law (Darcy, 1856), which defines the ability of a fluid to flow through porous media. Fluid can move from wetter to drier, even against gravity. This explains how water can be transported from the roots of a tree to the leaves. The Drawtex LevaFiber technology allows the dressing to lift, hold and transfer the wound exudate both vertically and horizontally using hydroconductive action (Smith et al, 2013).

Electrostatic action

Electrostatic action occurs when the negatively charged Drawtex wound dressing comes into contact with the wound exudate. Ions from the exudate form a mobile layer of the opposite charge, known as the electric double layer, effectively reversing the charge on the surface of the dressing to become positive. This allows the dressing to draw out a large amount of exudate, wound debris, bacteria and harmful MMPs (Spruce 2012; Smith et al, 2013).

Capillary action

Capillary action gives Drawtex its ability to move exudate, wound debris, bacteria and MMPs into the porous material of the dressing. With the small pores acting as capillaries, intermolecular attractive forces between the exudate and solid surfaces of the wound dressing allow the exudate to be drawn upward against the force of gravity (Smith et al, 2013).

The combination of these three types of action results in hydroconductive debridement. This process is indicated for wounds with moderate-to-heavy exudate such as venous leg ulcers, pressure ulcers, diabetic foot ulcers, complex surgical wounds, amputation sites, cavity wounds, post-operative wounds and other difficult-to-heal or non-healing wounds. Two clinical case studies (Wolcott and Cox, 2012; Ochs et al, 2012), a prospective randomised single-centre pilot study (Wendelken et al, 2012) and a study using a burns wound infection model (Ortiz et al, 2012) have found that the combination of three types of activity allow Drawtex to move exudate, wound debris, bacteria and harmful MMPs into the dressing, then disperse them both vertically and horizontally, evenly locking them into the Drawtex fibres In this way, Drawtex can help HCPs create an environment for optimal wound bed preparation (Spruce, 2012; Smith et al, 2013).

The following case studies describe the work of the Solent NHS Trust Podiatry service on three patients with diabetic foot ulcers (notoriously difficult to heal), which demonstrate the efficacy of Drawtex in achieving autolytic debridement combined with effective exudate management while containing harmful proteases within the dressing to reduce further tissue damage.

Case studies

Patient 1

Patient 1 is a 77-year-old male who was due for bypass surgery to help heal the wound on his foot which had been present for 22 weeks. The necrotic plaque was initially hydrated with the very closely monitored use of a thin hydrocolloid dressing for 1 week, which was changed twice in that time. Drawtex was then used on the wound, which measured 1.5 cm × 1.0 cm and had a depth of 0.7 cm (*Figure 1*). It contained 100% slough, which was well adhered to the wound bed. One week later, the wound size had reduced to 0.9 cm × 0.3 cm with the depth reduced to 0.2 cm. 50% of the wound had epithelialised, and the other half was dry slough. The surgeon reviewed the patient and cancelled the bypass surgery as the wound was healing well by that stage (*Figure 2* and *3*).

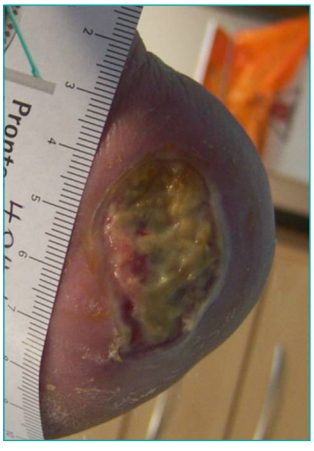

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Figure 1. Patient 1 at start of Drawtex trial.

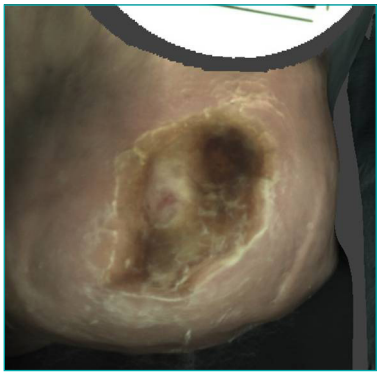

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Figure 2. Patient 1 midway through Drawtex trial.

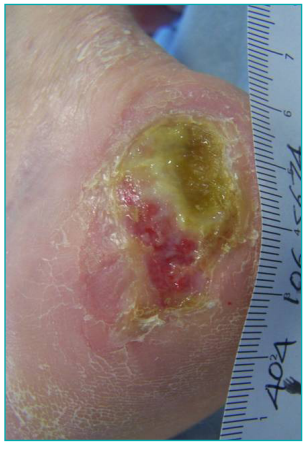

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Figure 3. Patient 1 at end of Drawtex trial.

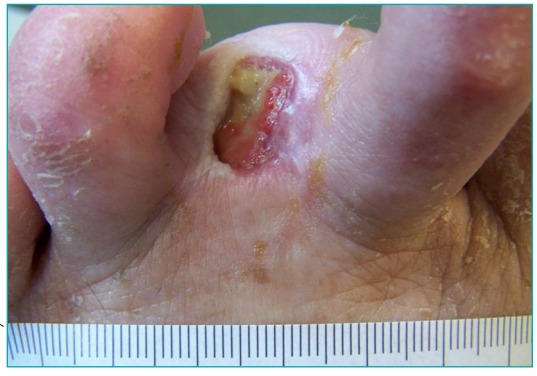
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Figure 4. Patient 2 at start of Drawtex trial.

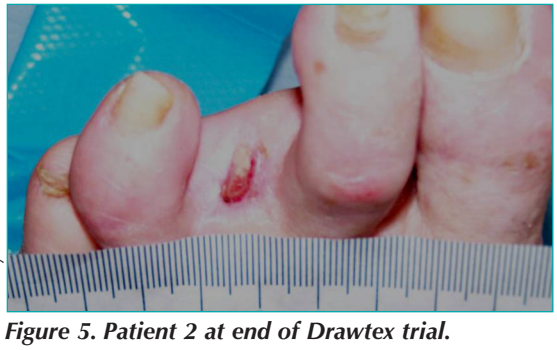
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Figure 5. Patient 2 at end of Drawtex trial.

Patient 2

Patient 2 is a 70-year-old female who had neuropathy and received podiatry services for a third toe amputation site due to ulceration that had been present for 11 weeks. The wound initially measured 1.5 cm × 1.1 cm and was 0.1 cm deep (*Figure 4*). The periwound skin was macerated, and the wound exuded a moderate amount of exudate. The wound bed contained 50% slough and 50% granulation at the start, but after 6 weeks' use of Drawtex, there was 100% epithelial tissue and the wound had healed (*Figure 5*).

Patient 3

Patient 3 is a 58-year-old male with neuropathy. He was treated by the podiatry service for fourth metatarsophalangeal joint ulceration with probing bone, following recent hospitalisation for spreading infection for 15 weeks. The wound initially measured 1.9 cm × 1.5 cm and was 0.1 cm deep (*Figure 6*). The periwound skin was macerated and the exudate level was high. He was treated with Drawtex to prepare the wound bed, where the dressing was used within the cavity. It was observed to manage wound exudate, reduce maceration and remove sloughy tissue from the wound bed (*Figure 7*). The wound size had increased slightly due to the debridement of callous and slough, but a healthy wound bed is now present (*Figure 8*).

Sizes and application

Under normal circumstances, Drawtex dressings are changed on a daily basis. However, on a healthy wound bed, Drawtex can be left on for up to 7 days. The state of the dressing dictates when it needs to be changed—i.e. when the dressing becomes saturated. Drawtex can be cut to fit any size or shape of wound. In addition, as many layers of Drawtex as necessary can be placed on the wound to ensure it will handle high volumes of exudate. Drawtex maintains its integrity and does not break down or lose fibre when fully saturated. It is not a solid dressing, so it has air permeability when dry. Drawtex is contraindicated in wounds with arterial bleeding.

Conclusion

Drawtex is indicated for all chronic wound types where debridement, reduction of bioburden, modulation of MMPs and effective exudate management are the primary aims of treatment in order to break the vicious circle of cellular and molecular imbalances that occur in the inflammatory phase of wound healing. It is very cost effective, as it combines all these properties within one product, reducing the need for multiple dressings and, as can be seen from the case studies, results in

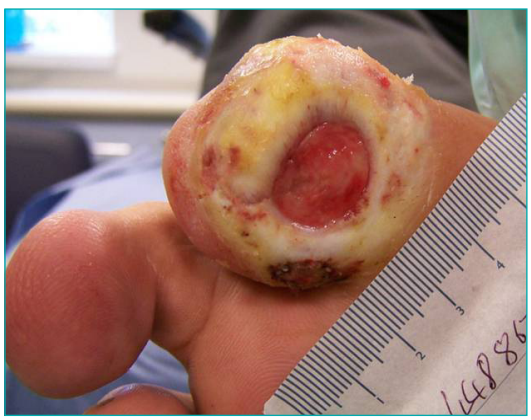
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Figure 6. Patient 3 at start of Drawtex trial.

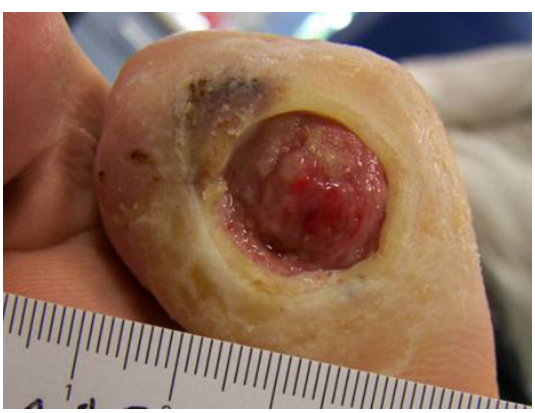
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Figure 7. Patient 3 midway through Drawtex trial.

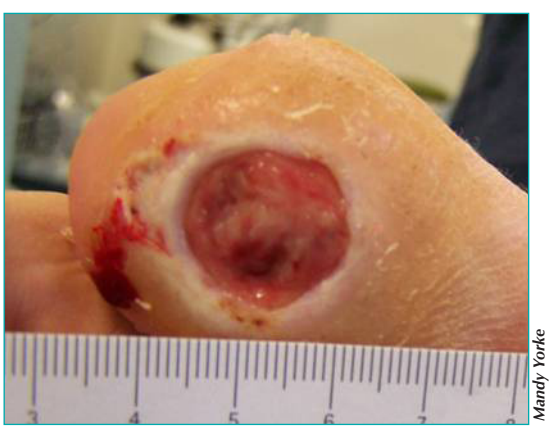
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Figure 8. Patient 3 at end of Drawtex trial.

shorter healing times,. In one case (patient 1), it achieved healing of a problematic wound without the need for surgical intervention, demonstrating a significant saving to the NHS and a beneficial outcome for the patient.

Achieving effective outcomes in the management of chronic wounds, for both patients and HCPs, in a culture of limited resources can be a huge challenge. Drawtex is an innovative product that can help HCPs to meet that challenge. **BJCN**

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KEY POINTS

- An imbalance of matrix metalloproteinases in the inflammatory phase of wound healing may result in a chronic or non-healing wound
- Accurate and timely assessment is necessary to identify and remove potential barriers to healing
- Appropriate use of wound care products will help to remove these barriers
- Choosing a product which has multiple modes of action will result in cost-effective wound management