Does "micro-trauma" of tissue play a role in adhesive dressing-initiated tissue damage?

KEY WORDS
- Adhesive dressing
- Macro-trauma
- Micro-trauma
- Tissue trauma
- Undisturbed healing

Although designed to promote healing by establishing an optimal wound environment, some dressing types damage the wound and periwound tissues, leading to delayed healing, affecting patient quality of life and having severe implications on the cost of care. In this article the authors hypothesise a novel mechanism by which dressings can traumatise wound and periwound tissue, proposing the concepts of dressing-related "macro-trauma" and "micro-trauma". Within this hypothesis they discuss the role of atraumatic dressings in addressing the challenge of minimising tissue damage, which can impact on the quality of life of patients with difficult-to-heal wounds. They also consider the implications for wound care treatment costs.

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A number of different adhesive systems are used to keep wound dressings in place (Rippon et al, 2007). The removal of dressings that adhere to the wound bed or periwound skin is a common cause of tissue trauma (Rippon et al, 2012). Repeated applications and removals potentially result in skin stripping (Rippon et al, 2007; Cutting, 2008; Davies and Rippon, 2008). This trauma is a major concern to both patients and clinicians, as the tissue damage may increase the size of wounds, lead to additional pain, cause inflammatory skin reactions (Cutting, 2008), or delay healing (Solowiej et al, 2009). Inadequate management of chronic wound exudate may result in skin excoriation, irritant dermatitis, and periwound skin maceration, which may also delay healing (Cutting and White, 2002).

The complex wound-healing response is dependent on the ordered progression of healing processes such as inflammation, granulation tissue formation and epithelialisation; perturbation in one or more of these phases is likely to lead to delays in healing (Chen and Rogers, 2007). There is a growing body of evidence that sustained elevation in the inflammatory response is crucial to the establishment and maintenance of chronic wounds in patients with a variety of underlying aetiologies (e.g. chronic venous insufficiency, diabetes; Mustoe et al, 2006; Chen and Rogers, 2007). The prolonged activation of a skin-localised inflammatory response due to underlying pathologies leads to localised dysregulation of inflammation and the release of factors detrimental to tissue integrity (e.g. inflammatory cell-derived protein-degrading enzymes such as elastase, matrix metalloproteinases and oxygen radicals). The inadequate control of the inflammatory response and accumulated damage of the inflammatory cell-derived components ultimately results in ulceration.

The contributions of adhesive-induced damage to "large-scale" tissue traumas, such as epidermal stripping and the destruction of new wound granulation tissue, have been investigated in a number of studies (Rippon et al, 2007; Cutting, 2008; Davies and Rippon, 2008; Waring et al, 2011). The contribution of aberrant inflammatory responses to ulcer formation and maintenance pathology has been supported by tissue biopsy characterisation studies (including wound exudates) as well as histological examination of skin and ulcer biopsies (Rogers et al, 1995; Loots et al, 1998; Yager and Nwomeh, 1999; Moor et al, 2009).

In this article, we present the hypothesis that adhesive dressings traumatise tissue via two separate, but related, mechanisms based on the magnitude of the traumatising event. We also consider the potential routes via which both traditional and modern dressings can contribute to tissue trauma

"Repeated application and removal of adhesive dressings leads to a sustained and cumulative localised tissue micro-trauma events."

and the impact of this trauma on healing, quality of life and treatment cost. Finally, we consider how the use of atraumatic wound dressings can address the challenge of reducing dressing-related tissue trauma and its consequences.

HYPOTHESIS

We hypothesise that adhesive-induced tissue damage consists of two components:

- "Macro-trauma" results in the immediate, adhesive dressing-mediated tissue damage events, such as skin stripping, blistering and tearing, as well as direct wound neo-matrix damage.
- "Micro-trauma" is characterised by adhesive dressing-mediated small-magnitude cell and skin extracellular matrix damage. Repeated application and removal of adhesive dressings leads to sustained and cumulative localised tissue micro-trauma events. The result is tissue damage at the wound site caused by dressings using excessively adherent adhesives.

MACRO-TRAUMA AND ITS EFFECTS ON WOUND HEALING

Dressing-related macro-trauma is the physical damage of tissue (e.g. periwound skin and wound bed) as a result of the excessive adherence of wound dressings to the tissue with which they are in contact (Rippon et al, 2012). The removal of dressings that adhere too strongly to the tissue surface leads to periwound epidermal stripping, blistering and skin tearing – clear signs of tissue damage (*Table 1*). Bleeding of new granulation tissue in the wound bed at the time of dressing removal is a further indicator of damage.

Macro-trauma due to excessive adhesive forces

Macro-trauma arises when the adhesive forces between the dressing and tissue are greater than the bond strength between tissue structures (e.g. epidermal layers, epidermal and dermal compartments, and blood vessels). The fragile and friable granulation tissue of the wound bed is particularly prone to disruption by mechanical forces (Xu et al, 2009), and macro-trauma episodes such as wound bed bleeding have been reported with increased frequency in association with adhesive dressings (Rippon et al, 2012).

A recent study examining the use of silicone-coated non-woven polyester dressings in a sheep dermal-wound model, found that coated dressings exhibited "minimal stickiness" to the wound bed and an enhanced wound healing response compared with foam or cotton gauze (Losi et al, 2012).

Adhesive interactions between dressing and wound bed can also lead to macro-trauma if a dressing that does not use a self-adhesive system component (e.g. non-adherent gauze, hydrofibre, or alginate) becomes incorporated into the wound bed. This occurs when the dressing is unable to control the moisture balance, allowing the wound to dry out, leading to adhesion of the dressing (Rippon et al, 2012).

Macro-trauma reduces quality of life

Reductions in quality of life due to delays in healing time are exacerbated by elevated pain levels both at and between dressing changes (Upton and Solowiej, 2012). Pain levels may also be elevated as a result of stress (Solowiej et al, 2009; Woo, 2010).

MICRO-TRAUMA AND ITS EFFECT ON WOUND HEALING

Micro-trauma occurs as a result of repeated exposure of skin and wound tissue to applied small-scale physical forces and stresses during the repeated application and removal of adhesive dressings (*Table 1*). Micro-trauma is subversive; the damage to the tissue is not immediately apparent because of the small-scale nature of the insult (Bronneberg et al, 2006). Tissue is damaged via a series of micro-wounding events, leading to damage of the tissue's cellular and extracellular matrix components and inducing localised inflammatory responses (Pedersen and Jemec, 2006; Adair-Kirk and Senior, 2008; Huo et al, 2009). This leads to the sustained release of inflammatory cell factors such as protein-degrading enzymes (Maquart et al, 2005). Time-dependent accumulation of these factors during cycles of application and removal of adhesive dressings leads to increased levels of tissue damage, elevated tissue inflammation resulting in observable skin reddening and overt tissue breakdown (macro-trauma).

There is also the potential for micro-trauma to occur as a result of small-scale shear forces

experienced by patients. These shear forces arise in tissues, acting against the force of friction, when a patient's position is changed (e.g. moving on a bed), resulting in the development of pressure ulcers (Ohura et al, 2005). The use of dressings in positions susceptible to shear stresses and tissue damage may help to dissipate these damaging forces. However, a proportion of these forces is likely to be transmitted to the underlying skin. In severe cases, the shear stresses result in the applied dressing rolling up and/or coming off completely. However, smaller magnitude forces and stresses at the interface between skin and a dressing are likely to result in localised tissue micro-trauma and stimulating localised tissue inflammation.

Micro-trauma due to dressing and tissues interactions

The small-scale mechanical stressing of skin (e.g. during tissue expansion procedures) results in histologically identifiable tissue damage (Huo et al, 2009), and affects skin properties such as barrier function (Pedersen and Jemec, 2006), with little or no outward sign of skin damage. These micro-trauma events stimulate the production of a variety of cell-derived components such as pro-inflammatory cytokines (Bronneberg et al, 2006) and protein-degrading enzymes (Yamamoto et al, 2003), which are crucial in normal wound healing progression. The accumulated effect of small-scale local tissue perturbations plays a role in the onset and maintenance of nonhealing wounds such as venous leg and diabetic foot ulcers (Mustoe et al, 2006; Chen and Rogers, 2007).

Chronic venous insufficiency (venous leg ulceration) and episodes of repeated ischaemia-reperfusion injury (pressure and diabetic foot ulceration) play key roles in ulcer pathology (Mustoe et al, 2006; Chen and Rogers, 2007). Both lead to localised tissue damage particularly in the vascular system of the skin of the lower limbs of affected patients. Inadequate treatment of the underlying aetiology leads to repeated small-scale damage to blood vessels and the stimulation of localised inflammatory activation at the site of the blood vessel damage.

The repeated release of pro-inflammatory cytokines, protein-degrading enzymes (e.g. neutrophil elastase and matrix metalloproteinases [MMPs]) and potent chemical factors such as reactive oxygen metabolites (ROMs) from inflammatory cells during this prolonged activation leads to localised tissue damage (Chen and Rogers, 2007). MMPs and ROMs are elevated in aged skin and are thought to play a role in the breakdown of collagen fibrils of the skin, leading

Table 1. Features of macro- and micro-trauma

MACRO-TRAUMA

Periwound skin stripping characterised by:

- Surface layers are removed from the skin as a result of cells adhering to the adhesive surface.
- The bond between the adhesive dressing and skin surface is stronger than the interaction between skin layers, so when the dressing is removed, the skin layer adhesions fail, leading to stripping of skin away from lower layers of skin.
- Repeated use of adhesive dressings with strong adhesive in the same location results in repeated damage to now compromised skin.

Periwound skin blistering characterised by:

- Interaction between epidermal and dermal layer at the basement membrane zone (epidermal–dermal junction) is disrupted by mechanical forces of dressing removal – the dressing–skin adhesion is not enough to completely remove epidermis.
- Sideways shear forces imparted on uppermost layers of skin by adhesive dressings that are unable to conform easily with the movement of the patient leads to epidermal–dermal junction breakdown and blistering.

Periwound skin tearing characterised by:

- Aggressive adhesive system in combination with friable periwound skin leads to complete failure of skin structures, causing complete breakdown in adhesion within tissue layers.

Wound bed damage characterised by:†

- Delicate and friable newly laid down provisional matrix/granulation tissue is susceptible to mechanical forces imparted due to adhesive interaction between dressing and tissue.
- Newly formed blood vessels damaged as a result of forces of dressing adhesion on removal.
- Wound bed matrix damaged and inhibits wound coverage by epidermal cells.

MICRO-TRAUMA

Matrix damage characterised by:

- Excessive adhesion of dressing to skin/wound leads to significant transference of mechanical forces with tissue layers.
- Mechanical and shear forces imparted on tissue leads to deformations in the interstitial matrix.
- Potential physical micro-disruptions of matrix structures acting as stimulus for tissue responses (e.g. inflammatory activation).

Cell-based damage characterised by:

- Excessive adhesion of dressing to skin/wound leads to significant transference of mechanical forces with tissue layers.
- Physical distortion of skin cells leads to micro-damage of cells and local release of cellular components leading to inflammation stimulation.

Inflammation characterised by:

- Matrix- and cellular-based damage and/or cell activation leads to localised inflammatory responses (see above).
- Localised release of inflammatory mediators (e.g. protein-degrading enzymes, reactive oxygen species).
- Localised tissue damage due to combination of adhesive dressing-generated mechanical forces and sustained inflammatory cell activation.
- Increased susceptibility to further tissue damage due to heightened inflammatory state.

†Note: damage also due to incorporation of dried wound exudate within the body structure of the cover dressing. Physical damage on removal of dressing due to intimate interaction (adhesion), though not directly related to dressing's adhesive system

"Tissue is damaged via a series of micro-wounding events leading to damage of the tissue's cellular and extracellular matrix components and inducing localised inflammatory responses."

to a reduction in mechanical strength (Fisher et al, 2009; Ibuki et al, 2012). We hypothesise that the micro-trauma stimulates a small, localised skin inflammation with accumulations of damaging inflammatory mediators. Clinically observable tissue damage and subsequent tissue breakdown (e.g. adhesive-induced macro-trauma) occurs when the tissue is unable to adequately control tissue homeostasis.

Skin maceration and micro-trauma

Skin maceration occurs when dressings are unable to cope with levels of exudate being produced (Cutting and White, 2002). The components of fluid derived from chronic nonhealing wounds differ from acute wound fluid (Thamm et al, 2013). Chronic wound fluids contain a number of components that are damaging to tissue (e.g. MMPs and ROMs). We hypothesise that exposure of tissue (periwound skin and wound-bed granulation tissue) to these detrimental chronic wound exudate components contributes to the tissue damaging nature of skin maceration.

Micro-trauma reduces quality of life

The sustained, dressing-induced micro-trauma episodes may have a systemic impact on a patient's well-being. The presence of a sustained stimulation of a localised inflammation may play a role in affecting the patients' physiology more widely. For example, the physiological consequences of severe trauma (significant blunt trauma, shock initiated as a result of a significant amount of blood loss) are well documented (Xiao et al, 2011; An et al, 2012). The resultant inhibition of the immune system in these patients and the corresponding elevated susceptibility to infection (which can be life-threatening) is a common result of significant tissue trauma (Angele and Chaudry, 2005).

We suggest that there is the potential for any repeated local tissue trauma that leads to sustained levels of stimulated inflammation (e.g. repeated micro-trauma) to have consequences for other physiological processes. However, it may be that these effects are masked by the significantly higher levels of downstream inflammatory processes due to the nonhealing wound.

THE IMPACT OF DRESSINGS AND DRESSING ADHESIVES ON PATIENTS

Wound-related trauma and the pain associated with wounds are major concerns to both patients and clinicians (Solowiej, 2009), impacting the clinical, patient and cost aspects of wound care (Butcher, 2010). The removal of dressings that must adhere to the wound bed/periwound skin, as well as the *in situ* presence of wound dressings whose physical characteristics exacerbate the exposure of tissues to heightened physical stresses such as shear forces, is a common cause of tissue trauma (Waring et al, 2011; Waring and Butcher, 2011). Macro- and micro-trauma can increase the size of wounds, exacerbate pain and delay the healing response (Davies and Rippon, 2010), all of which have a negative impact on the cost of care and quality of life (Rippon et al, 2007).

Clinical impact

Dressings using traditional adhesives as part of the securing mechanism have been associated with higher peel forces and significant skin damage on removal (Dykes et al, 2001; Waring et al, 2008). Dressing-related tissue trauma (periwound skin and wound bed) has been reported in a wide range of wound types, including surgical and traumatic wounds, venous leg ulcers, diabetic foot ulcers and pressure ulcers (Cutting, 2008; Davies et al, 2008).

Adhesive wound dressings have been shown to increase transepidermal water loss, an indicator of the skin barrier function, suggesting skin trauma (Zillmer et al, 2006; Dykes, 2007). Similar findings have been reported in a retrospective review of data from patients with a variety of wound types treated with adhesive dressings (Eager, 2001). A randomised controlled study determining the effect of repeated removal of dressings – including dressings with traditional adhesives – on peri-ulcer skin of patients with venous leg ulcers showed significant dressing-related trauma compared with adjacent, non-treated skin (Zillmer et al, 2006).

Damage caused by dressings puts additional pressures on clinical resources due to the additional time taken to heal these secondary wounds (Gupta et al, 2002; Butcher and White,

2011). Mechanical stresses, such as those applied by overly-adherent wound dressings, have been shown to prolong tissue inflammation (micro-trauma), and contribute to extended wound-healing times (Wong et al, 2011a;b).

Patient impact

Pain is a significant problem for patients with all types of wounds (White, 2008). For wounds such as nonhealing ulcers, where the duration of the wound can be measured in months or years, wound-associated pain is a major contributor to the patient's quality of life (Heinen et al, 2004). Psychological stress before and during dressing changes has been shown to influence the pain experienced by patients; and pain levels increase when an individual is stressed, anxious or depressed (Solowiej et al, 2009; Woo, 2010). The pain experienced during dressing changes can be one of the most painful aspects of care (Price et al, 2008), aside from the wound itself. The use of traditional dressings that use aggressive adhesive systems to secure them is a major contributor to patients' poor experiences with dressing changes.

Cost impact

The clinical and patient consequences of using dressings incorporating aggressive adhesive system have extenuating costs. Cost-effectiveness modelling studies have suggested that the most expensive aspect of the care of patients with ulcers is clinician labour, including the time taken for nurses to change dressings (Meaume and Gemmen, 2002). Any dressing-associated tissue trauma (macro- or micro-trauma) that extends healing time (or complications such as infection) will add significantly to the cost of wound care. Increased levels of tissue macro- and micro-trauma, therefore, lead to increases in the overall cost of treatment. For cases where hospitalisation is required (or discharge is delayed), care costs can escalate. As well as costs directly associated with dressing-related tissue trauma, additional costs that are not immediately obvious (e.g. extra prescriptions and clinical consultations) can lead to a cascade of additional expense (Butcher and White, 2011).

Butcher and White (2011) have developed a cost equation for the issue of pain at dressing change and highlight that the issue is multi-dimensional and environment specific. It is clear that the cost implications of applying an appropriate dressing to a wound can be wide-reaching. Appropriate dressing selection is increasingly being seen as a way to not only improve healing in difficult-to-heal ulcers and acute wounds (and reduce cost because of improved healing rates), but also to improve cost-effectiveness by minimising additional costs due to dressing-related tissue trauma (Guest et al, 2012).

ADDRESSING THE IMPACT OF MACRO- AND MICRO-TRAUMA WITH ATRAUMATIC WOUND DRESSINGS

In the development of adhesive wound dressings, the aim is to achieve an adhesive that is strong enough to keep the dressing in place during the patient's day-to-day movements, but also to have a low enough level of adhesion so that the dressing does not cause trauma to the skin during repeated application and removal cycles. Studies suggest that, in the majority of cases, the current technology in dressing adhesive systems is not adequate to find the ideal balance (Davies and Rippon, 2008; Rippon et al, 2012).

Thomas (2003) has coined the phrase "atraumatic dressings" for a category of dressings which do not cause trauma to newly formed tissue or to the periwound skin on removal. This category of dressing is based around soft-silicone adhesive technology which, when coated onto dressing materials, allows interaction between the dressing and wound/periwound skin (Davies and Rippon, 2008). This soft silicone interface layer allows a more efficient interaction between tissue and dressing, providing a greater area of contact with the skin and a better level of adhesion without tissue damage. Tissue trauma is minimised during the repeated application and removal of these dressings. When the dressing is removed, the tissue–dressing adhesive contact fails before the tissue–tissue bond is compromised, so the dressing pulls free from the tissue.

Clinical studies in a variety of chronic wounds show that dressings using soft silicone are associated with less traumatic injury (macro- and micro-trauma) related to the dressing than those with

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REFERENCES

Adair-Kirk TL, Senior RM (2008) *Int J Biochem Cell Biol* 40(6–7):1101–10

An G, Nieman G, Vodovotz Y (2012) *Int J Burns Trauma* 2(1):1–10

Angele MK, Chaudry IH (2005) *Langenbecks Arch Surg* 390(4):333–41

Bronneberg D et al (2006) *Ann Biomed Eng* 34(3):506–14

Bugmann P et al (1998) *Burns* 24(7):609–12

Butcher M (2010) A Delphi study of the financial impact of pain at dressing change. Presentation. Wounds UK Annual Conference, Harrogate

Butcher M, White R (2011) Quantifying the financial impact of pain at dressing change. In: Upton D (ed) *Psychological Impact of Pain in Patients With Wounds*. Wounds UK, London

Chen WY, Rogers AA (2007) *Wound Repair Regen* 15(4):434–49

Cutting KF, White RJ (2002) *J Wound Care* 11(7):275–8

Cutting KF (2008) *IJ Wound Care* 17(4):157–62

Davies P, Rippon M (2008) *J Wound Care* 17(11 Suppl):3–31

Davies P, Rippon M (2010) *World Wide Wounds* Available at: http://bit.ly/lyYaMG (accessed 14.10.2013)

Dealey C, Posnett J, Walker A (2012) *J Wound Care* 21(6):261–6

Dykes PJ (2007) *J Wound Care* 16(3):97–100

Dykes PJ, Heggie R, Hill SA (2001) *J Wound Care* 10(2):7–10

Eager CA (2001) Comparison of two foams through the measurement of healing time, frequency of dressing changes and periwound status. Presentation. 14th Annual Symposium on Advanced Wound Care and Medical Research Forum on Wound Repair, Orlando, FL

Fisher GJ et al (2009) *Am J Pathol* 174(1):101–14

Gotschall CS et al (1998) *J Burn Care Rehabil* 19(4):279–83

Guest J F et al (2012) *J Wound Care* 21(8):389–98

Gupta SK et al (2002) *J Wound Care* 11(7):271–3

Heinen M M et al (2004) *J Clin Nurs* 13(3):355–66

Huo R et al (2009) *Dermatol Surg* 35(1):72–9

Ibuki A et al (2012) *Exp Dermatol* 21(3):178–83

Lapioli-Zufelt A, Morris EJ (1998) *J Wound Ostomy Continence Nurs* 25(6):314–6

Loots MA et al (1998) *J Invest Dermatol* 111(5):850–7

Losi P et al (2012) *J Mater Sci Mater Med* 23(9):2235–43

Maquart FX et al (2005) *Biochimie* 87(3–4):353–60

Meaume S, Gemmen E (2002) *J Wound Care* 11(6):219–24

Meaume S et al (2003) *Ostomy Wound Manage* 49(9):44–51

Moor A N et al (2009) *Wound Repair Regen* 17(6):832–9

Morris C et al (2009) *Paediatr Nurs* 21(3):38–43

Mustoe TA et al (2006) *Plast Reconstr Surg* 117(7 Suppl):35–41S

O'Donovan DA et al (1999) *J Hand Surg Br* 24(6):727–30

Ohura N et al (2005) *J Wound Care* 14(9):401–4

Pedersen L, Jemec GB (2006) *Acta Derm Venereol* 86(4):308–11

Price PE et al (2008) *Int Wound J* 5(2):159–71

Rippon M et al (2012) *J Wound Care* 21(8):359–68

Rippon M et al (2007) *Wounds UK* 3(4):76–86

Rogers AA et al (1995) *Wound Repair Regen* 3(3):273–83

Solowiej K et al (2009) *J Wound Care* 18(9):357–66

Thamm OC et al (2013) *Int Wound J* Mar 21 [Epub ahead of print]

Thomas S (2003) *World Wide Wounds*. Available at: http://bit.ly/hENIc4 (accessed 14.10.2013)

Upton D, Solowiej K (2012) *J Wound Care* 21(5):209–15

Waring M, Butcher M (2011) *Wounds UK* 7(3):14–24

Waring M et al (2011) *J Wound Care* 20(9):412–22

Waring M et al (2012) *Wounds UK* 8(2):60–7

Waring M et al (2008) *Wounds UK* 4(3):35–47

White R (2008) *Wounds UK* 4(1):14–22

Wong V W et al (2011a) *J Invest Dermatol* 131(11):2186–96

Wong V W et al (2011b) *FASEB J* 25(12):4498–510

Woo K Y et al (2009) *Adv Skin Wound Care* 22(7):304–10

Woo K (2010) *Wounds UK* 6(4):92–8

Xiao W et al (2011) *J Exp Med* 208(13):2581–90

Xu X et al (2009) *Am J Emerg Med* 27(6):729–33

Yager DR, Nwomeh BC (1999) *Wound Repair Regen* 7(6):433–41

Yamamoto K et al (2003) *Curr Vasc Pharmacol* 1(3):315–9

Zillmer R et al (2006) *J Wound Care* 15(5):187–91

traditional adhesives (Meaume et al, 2003; Zillmer et al, 2006; Waring et al, 2012). Atraumatic wound dressings are also associated with significantly less wound-associated pain than traditional adhesive dressings (Meaume et al, 2003; White, 2008; Woo et al, 2009). In patients with acute wounds similar experiences have been reported – reduced levels of periwound skin reactions (micro-trauma) and reduced pain severity scores as well as reduced patient discomfort (O'Donovan et al, 1999; Morris et al, 2009). Significantly lower patient stress levels have also been reported in patients treated with atraumatic wound dressings (Lapioli-Zufelt and Morris, 1998; O'Donovan et al, 1999).

Atraumatic wound dressings have been associated with increased healing rates and reduced levels of dressing-related tissue trauma compared with traditional adhesive dressings (Davies and Rippon, 2008). Because time to healing and additional healing complications are both significant drivers of increased wound care costs, the use of atraumatic dressings are a significant tool in driving cost-effective wound care (Bugmann et al, 1998; Gotschall et al, 1998). Lower levels of adhesion of dressings to underlying tissues have been shown to result in quicker dressing changes (Gotschall et al, 1998). As nursing time is a significant cost in the total wound care costs (Dealey et al, 2012), reducing nursing time at dressing changes is another significant driver of cost-effectiveness.

Studies of the relationship between pain, stress/anxiety and wound healing suggest that the use of atraumatic wound dressings, for example, those that use soft silicone, reduce the level of pain experienced at dressing changes, decrease the stress and anxiety experienced by these patients and is likely to have a positive impact on healing times (Davies and Rippon, 2008; Rippon et al, 2012). The association of increased stress and increased sensitivity to pain suggests also that the use of atraumatic wound dressings will have a positive reinforcing effect for patients – the regular use of these dressings will reduce patients' expectations of having a painful dressing change, and they will feel less anxious.

CONCLUSION

The interaction of wound dressings with the wound bed and periwound skin is complex. Dressings are expected to do more than just cover a wound and offer protection from the outside environment, so how they interact with the wound bed and surrounding skin is becoming more intimate. A more intimate contact between wound and dressing consequently enhances the opportunity for detrimental as well as positive effects on wound healing. Self-adhesive wound dressings use a variety of adhesive technologies to ensure that dressings remain in position for optimising the benefits of the dressings to promote healing. We have proposed that, as well as the large-scale tissue damage (macro-trauma; e.g. skin stripping, blistering and tearing) seen clinically as a result of the repeated application and removal of adhesive wound dressings, adhesive dressing can also inflict smaller-scale (micro-trauma) damage on the wound and surrounding structures that can accumulate and lead to significant tissue damage.

Our hypothesis is testable. Clinical studies examining the use and effectiveness of wound dressings in the care of patients with a variety of nonhealing wounds can be designed to include the collection of data on the subtle skin changes proposed to arise as a result of micro-trauma. Dressing-derived tissue trauma has a significant impact on the overall wound care, delaying healing, resulting in additional complications that require treatment and significantly elevating the costs associated with treatment. When skin is subjected to repeated traumatic insults, such as those experienced with the repeated application and removal of aggressively adhesive wound dressings, it becomes damaged. The use of atraumatic wound dressings reduces dressing-related tissue trauma, results in pain-free dressing changes, improves patients' quality of life through reduced psychological anxiety and stress, improves healing rates and contributes significant cost of care savings. WUK

DECLARATION OF INTEREST

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