

Biophysical properties of a Hydrofiber® cover dressing

Mike Walker, Sharon Lam, Dave Pritchard, Christine A Cochrane

Abstract

Aims: Good exudate management needs to be maximised for both the wound and the patient to ensure wound healing can take place in a timely manner. The *in vitro* studies in this article evaluate a new Hydrofiber® cover dressing (HCD) for both its exudate management and biophysical properties (e.g. cellular adhesion and contouring to uneven wound surfaces). **Methods:** These included measuring fluid absorption and retention, the conformability of dressings to a simulated wound surface, measurements of fluid lateral spread, dressing bioadhesion and interactions with wound fibroblasts that were set into a collagen gel matrix. **Results:** Although the majority of the foam dressings absorbed more fluid than the corresponding new HCD, when placed under pressure the ability to retain fluid was far greater with the HCD showing fluid retention in excess of 90%. Similar results were shown for the lateral spread of fluid. Not all the dressings showed complete conformability to the simulated wound surface due to the more 'rigid' structure of the foam dressings. The results for the bioadhesion and gel contraction studies showed the importance of combining absorption properties of foams with Hydrofiber® Technology by showing less bioadhesion ($p < 0.001$) and being less detrimental to fibroblast contraction and viability ($p < 0.001$). **Conclusions:** These studies highlight the need to choose the most appropriate wound dressing, which not only provides the best possible exudate management, but also subjects the wound to the least number of interventions that might interfere with the repair process. **Conflict of interest:** Mike Walker, Sharon Lam, and Dave Pritchard are all employees of ConvaTec Ltd.

KEY WORDS

Wound care

Wound healing

Hydrofiber® cover dressing

Hydrofiber® technology

Any breakdown of the protective function of the skin results in the formation of a wound, which not only provides a portal of entry for bacteria, but also creates an exit portal for wound exudate. The amount of exudate produced in any one wound may become a problem to both the patient and the clinician if it is not controlled.

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In a healing wound, exudate is considered to be an aid to the healing process by maintaining a moist environment, promoting cell proliferation and providing essential cell nutrients, as well as stimulating autolysis (the removal of dead or damaged tissue) (World Union of Wound Healing Societies [WUWHS], 2007).

It is important that an appropriate dressing is chosen which not only has the capacity to absorb exudate, but can also be retained within the dressing structure and not become laterally spread onto the surrounding skin.

In chronic wounds, however, exudate is considered to be 'a corrosive biological fluid', due to the many harmful components (e.g. bacteria, enzymes) that are contained within it (Chen et al, 2004). Consequently, it is important to manage exudate 'to maximise the

benefits to the wound and the patient' (WUWHS, 2007). If leakage is allowed to occur there is the potential for maceration and excoriation of the healthy periwound skin, which can lead to further breakdown of this tissue due to the corrosive nature of chronic wound exudate.

Recent studies have shown that the periwound skin area of ischaemic diabetic patients is often compromised (Walker et al, 2008; Vuorisalo et al, 2009). It is important that an appropriate dressing is chosen which not only has the capacity to absorb exudate, but can also be retained within the dressing structure and not become laterally spread onto the surrounding skin.

Equally important is the ability of the dressing to conform to the wound surface, as this leaves no room for dead spaces between the wound and the dressing interface, reducing the possibility of increased bacterial proliferation (Hoeskstra et al, 2002; Jones et al, 2005).

Wound dressings should also be able to 'respond' to the wound environment,

influencing the cellular environment of a healing wound through the maintenance of moisture balance (Bishop et al, 2003).

Some wound care products can become attached to a wound or skin surface, and this can result in trauma to the wound upon removal of the dressing, which may disrupt the newly formed fragile re-epithelial tissue surrounding the wound. This can be painful for the patient. A previous study using an *in vitro* wound fibroblast cell adhesion model has shown that fibroblast adhesion can vary with dressing composition (Cochrane et al, 1999).

Methods

Test materials

The dressings shown in Table 1 were chosen as representative polyurethane-based foam dressings to compare their biophysical properties versus a new gelling foam dressing containing Hydrofiber® Technology.

The range of these *in vitro* laboratory studies to the best of the authors' knowledge has not been completed on such products and provides an overall understanding of each dressing's performance. The results are intended to provide useful information to ensure that the most appropriate wound dressing is chosen.

Fluid absorption and retention

In these studies, all the foams (A-H) as well as GF-N and GF-A were tested in triplicate. A 5x5cm test sample was cut from the centre of each dressing and weighed [w_1]. The samples were then placed onto a stainless steel perforated plate inside a large tray, with the wound contact layer facing downwards.

A solid Perspex plate and an equivalent weight were placed onto the exposed dressing surface to ensure a force equivalent to 40mmHg (Lide, 1991), which is considered to be representative of the pressure applied with a high compression bandage (Thomas, 1997). Solution A (sodium chloride and calcium chloride solution BP) was added to the tray until it reached the top of the stainless steel

Table 1
Dressings tested

Dressing	Reference	Dressing	Reference
Allevyn™ (Smith & Nephew)	Foam A	Mepilex® Border (Mölnlycke)	Foam F
Allevyn™ Plus Adhesive (Smith & Nephew)	Foam B	Tielle™ Borderless (Systagenix)	Foam G
Biatain® (Coloplast)	Foam C	Tielle™ Plus Borderless (Systagenix)	Foam H
Biatain® Adhesive (Coloplast)	Foam D	Versiva® XC® Non-adhesive (ConvaTec)	GF-N
Mepilex® (Mölnlycke)	Foam E	Versiva® XC® Adhesive (ConvaTec)	GF-A

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perforated plate, which was left at a temperature of 20°C for 24 hours. After this time any excess solution A was carefully removed and the test sample was immediately re-weighed using forceps [w_2]. The test sample was then placed onto a double layer of absorbent paper towel, and the Perspex plate and weight applied onto the sample for one minute before re-weighing [w_3].

Fluid absorption per unit area was calculated as:

$$(w_2 - w_1) / A, \text{ where } A = \text{area of sample tested in cm}^2$$

Fluid retention was calculated as:

$$(w_3 - w_1) / A, \text{ where } A = \text{area of sample tested in cm}^2$$

In addition to the above, a visual assessment of the ability of the dressings

to retain fluid under compression was conducted by fully saturating a dressing, positioning it onto absorbent paper, and placing a weight equivalent to 40mmHg on top of it. The leakage of fluid through and from the sides of the dressings was assessed visually.

Contouring to an *in vitro* simulated wound tissue model

This method was devised to assess a dressing's ability to follow the contours of an uneven simulated wound surface. A cover dressing or retaining tape was also applied to more closely represent clinical situations. Dressings GF-N, foam A and foam E were tested.

Two saw cuts were made to the edges of the bottom half of a 90mm diameter Petri dish, perpendicular to the base of the dish and approximately 4cm apart. A simulated wound surface (porcine belly tissue) was cut to fill the distance between the two saw

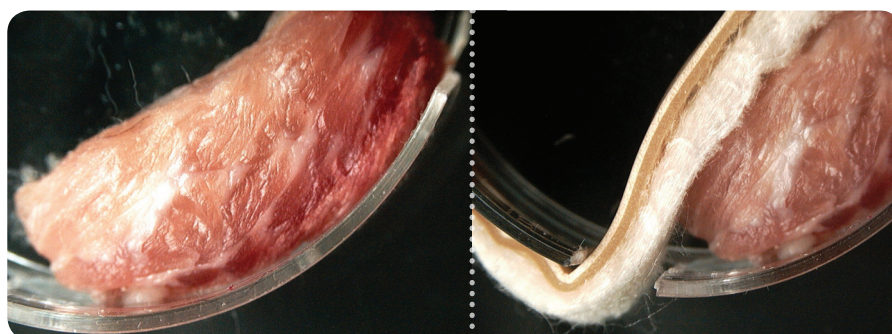


Figure 1. Porcine belly tissue fixed to the inside of the Petri dish (left) and indentation made for dressing (right).

cuts (approximately 15x5mm). This simulated wound surface was stuck to the inside edge of the Petri dish with cyanoacrylate adhesive (Figure 1). A small hole was made in the outside edge of the Petri dish to allow a needle to penetrate through to the surface of the simulated wound bed. Samples of the dressings under test were cut to approximately the same size as the simulated wound tissue and each was placed over the simulated wound surface and secured in place with Micropore® (3M) tape.

A needle was attached to a syringe pump with a piece of flexible tubing and pushed through the hole in the Petri dish until the tip protruded slightly above the surface of the simulated wound bed. A Microjet Crono PCA syringe pump (20ml) (Cane Medical Technology, Italy) was programmed to deliver solution A containing a pink food dye at a rate of 2.0ml/hour. The sample was then placed under a microscope with a camera attached. Initial images were captured using a software system. The syringe pump was activated and a series of images was captured over a period of several minutes as the test dressing hydrated. The food dye was added to provide a clear demonstration of each dressing's absorption capacity and to show their ability to contour to a simulated wound surface.

Lateral spread test

To assess the lateral spread of fluid from individual dressings (e.g. A, B, E, F and GF-N), the following *in vitro* procedures were carried out. A plastic vial with the end cut off was positioned in the centre of the wound contact layer of the test dressing and held in place. The diameter of the vial represented the 'wound' contact area. Following this, 20ml of simulated exudate (equine serum, a solution more akin to wound fluid that contains proteinaceous material and has a viscosity more like wound fluid) (Sera Laboratories International) was injected into the vial, immediately hydrating the dressing over the 'wound' contact area.

Once the full content of the syringe had been expelled, the stop watch was started. When 60 seconds had elapsed, any non-absorbed fluid was removed from the plastic vial with the syringe. In the authors' opinion, 60 seconds was an appropriate length of time as if a wound is exuding the dressing needs to absorb the fluid quickly and lock it away, preventing it from moving along the dressing. The vial was then removed. A ruler was placed underneath the dressing to measure the amount of lateral spread of the fluid along the dressing, and a photograph was taken of both dressing and ruler using a digital camera. Once all the photographs had been taken, the area of lateral fluid spread was measured using image analysis software. Each product was tested in triplicate to allow some form of statistical analysis and the lateral spread expressed as a percentage of the original 'wound' area. The mean percentage increase in lateral spread was calculated as follows:

$$\left[\frac{\text{Lateral spread/vial area}^*}{100} \times 100 \right] - 100$$

*where vial area = 660.6mm²

Bioadhesion studies

The evaluation of cellular adhesion and any subsequent granulation tissue damage in the clinic are likely to be difficult and probably subjective. The development of an *in vitro* model allows for quantitative measurements of cellular adhesion to wound dressings (Cochrane et al, 1999).

Six samples of both GF dressings and all the non-gelling foam dressings (A-H) were tested in these studies. Equine granulation tissue fibroblasts were taken from both healing and non-healing areas of wounds, or from excessive granulation tissue that had been excised from chronic wounds located on a horse's hind limb. Fibroblast cultures were prepared as previously described by Cochrane et al (1999). Briefly, this involved taking tissue samples for fibroblast culture and immediately transferring them to a sterile dish, followed by washing them in Hank's balanced salt solution

(HBSS). Small (3–5mm²) pieces were then placed into 25cm² tissue culture flasks containing media, which consisted of Dulbecco's Modified Eagle Medium (DMEM), supplemented with 10% foetal calf serum (FCS) (Sigma, UK), 20 mM HEPES buffer, 100µg/ml gentamicin and 0.5µg/ml amphotericin B. Cell cultures were incubated at 37°C in a 5% CO₂/95% air environment. Readiness for sub-culturing was determined by the extent of fibroblast cell outgrowth (5–10 days). Cells were farmed successively in a 1:4 split ratio to passage 3–8 experimental use. Fibroblasts were harvested from stock dishes and plated out at 2x10⁵ cells/ml.

A 1cm² piece of each dressing was cut from the central foam padded area and the adhesive border edge. The central areas were applied either as the dry dressing or following hydration (1ml of cell culture medium). All cut dressings were placed onto the monolayer of fibroblasts. After 24 hours the dressings were carefully removed from the surface of the culture, using minimal force to avoid damaging the cells or causing any additional cells to detach from the dressing. The cell numbers on each dressing were determined through trypsinisation and counting the cells manually using a Neubauer chamber.

In vitro fibroblast cell culture studies

Collagen gel studies

Equine granulation tissue fibroblasts were taken from both healing and non-healing areas of wounds, or from excessive granulation tissue that had been excised from chronic wounds located on the horse's hind limb.

Collagen gel contraction model

This is an *in vitro* model that allows fibroblast function (e.g. contraction) to be measured by suspending them in a collagen gel matrix. Contraction of these fibroblast/collagen gels allows the biological mechanisms of wound contraction to be measured (Germain, 1994).

Consequently, this model shows how different dressings may affect the contraction rate of these gels by

the application of individual dressing to the gelled surface. In these studies, equine granulation tissue fibroblasts (which have been shown to be similar to human granulation tissue fibroblasts) were taken from both healing and non-healing areas of wounds, or from excessive granulation tissue that had been excised from chronic wounds located on the horse's hind limb. These fibroblasts were then prepared as described in the previous section (Cochrane et al, 2003).

Following the incubation period, fibroblasts were harvested from stock dishes and plated out into 35mm six-well plates at 1×10^6 cells/ml which were suspended within the collagen at a concentration of 2mg/ml, followed by incubation in a 5% CO₂/95% air environment at 37°C. After one hour, the collagen fibroblast gels had solidified, and the gel surface was washed with 1ml of HBSS. Following this, 1ml of media (M1) (the same media as used in the bioadhesion studies) was added before the application of 0.5g of each individual pre-hydrated dressing section to the gel surface. Gel contraction measurements were made using callipers (mm), at 24-hour intervals up to and including 96 hours. Six measurements were made per product.

Contraction analysis

Contraction of the collagen gels was carried out as follows. Before the application of each dressing a measurement of the diameter of the area of contraction was taken (e.g. 35mm). Subsequent measurements were taken using callipers to determine the amount of contraction that had taken place at 24-hour intervals over a 96-hour period. The calculations represent the mean and standard deviations of six gels for each dressing tested.

Cell viability assessment

The viability of the suspended fibroblasts within the collagen gel was monitored using Trypan blue staining. At the end of the experiment (96 hours), the cells were released from the collagen gel by digestion using 0.2%

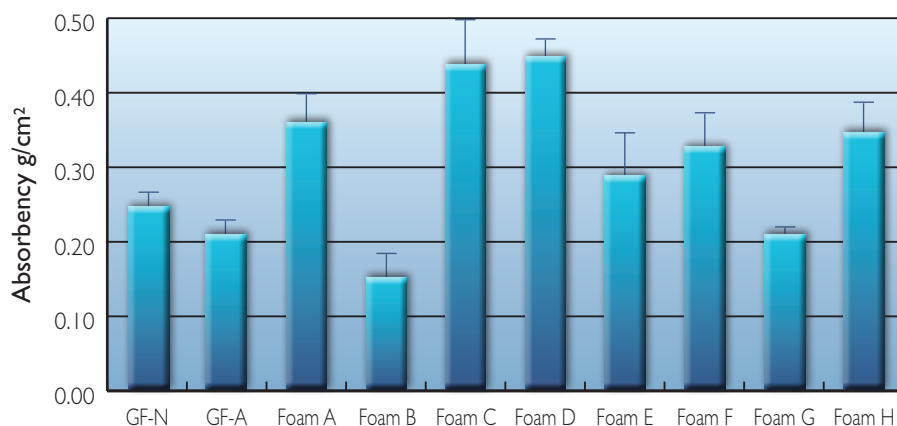


Figure 2. Absorbency for GF dressings and other foam dressings.

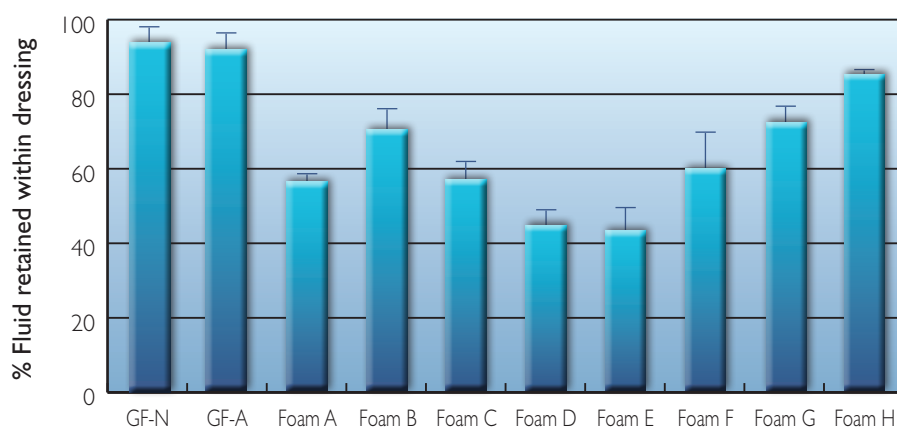


Figure 3. Fluid retention for GF and other foam dressings under a force equivalent to 40mmHg.

collagenase in cell culture medium for ten minutes, re-suspended in DMEM and tested for viability. Viability was expressed as the percentage of viable cells remaining in the cell suspension.

Results

Statistical analyses

Where relevant, two-sample significance tests (e.g. t-tests) were performed using the statistical package Minitab Release 14 for Windows® 2003.

Fluid absorption and retention

The absorption results demonstrate that all the foams, except foams B and G, absorbed significantly more fluid ($P < 0.05$) than those obtained for the GF-N and GF-A (Figure 2).

This result was not unexpected as foam dressings that do not gel have a high fluid capacity due to their more 'open' structure. However, what is of greater importance is a dressing's ability to retain fluid under pressure (i.e. under

compression bandaging). Consequently, the results for fluid retention under pressure demonstrate the dressings that were shown to absorb the most (e.g. foams C and D) subsequently retained only 58% and 45% respectively, whereas the two Hydrofiber® cover dressings (HCDs) (GF-N and GF-A) were both shown to retain >90% fluid under a force equivalent to 40mmHg (Figure 3).

These differences were shown to be statistically significant ($p < 0.05$) for both the adhesive and non-adhesive HCDs compared to the other non-gelling foam dressings tested. Figure 4 shows the foam dressing surfaces.

The results for the visual assessment of fluid leakage under compression are shown in Figure 5. Following the application of pressure, the absorbent paper beneath the GF-N dressing remained dry (Figure 5 centre). In contrast, fluid loss from foams A and E

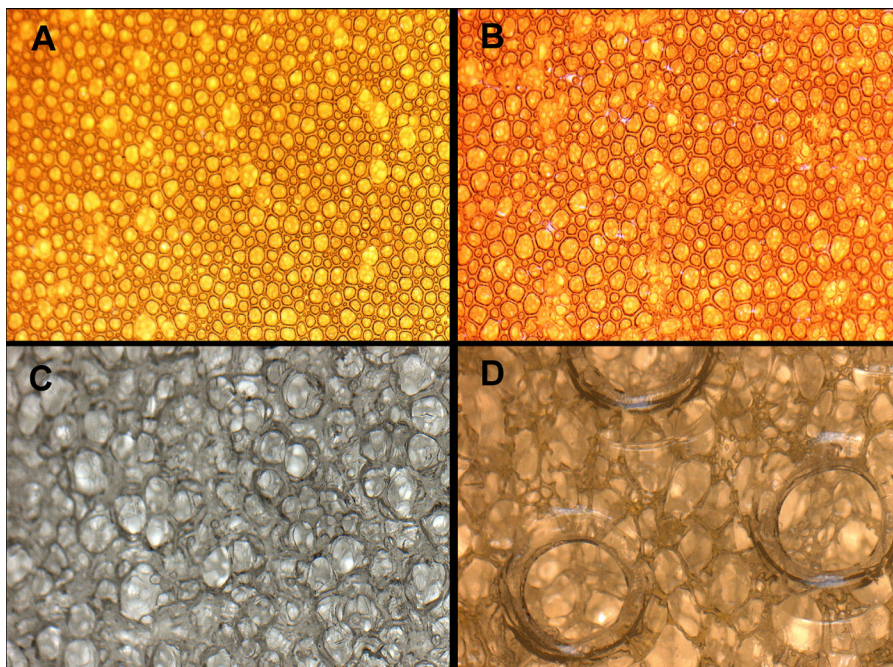


Figure 4. A and B show the wound contact surface for foams C and D respectively, and C and D show the wound contact surface for foams E and F respectively. The differences in the physical appearance of the wound contact layer may help to explain the differences noted in their respective absorption and retention properties.

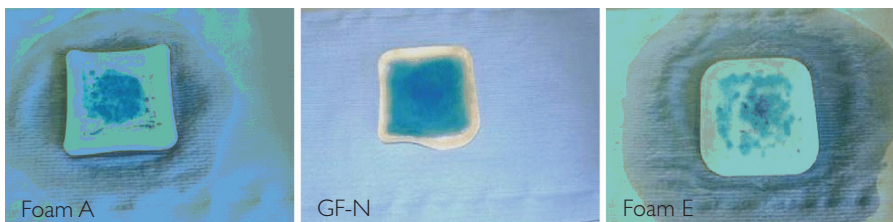


Figure 5. Spread of fluid following the application of a weight equivalent to 40mmHg pressure.

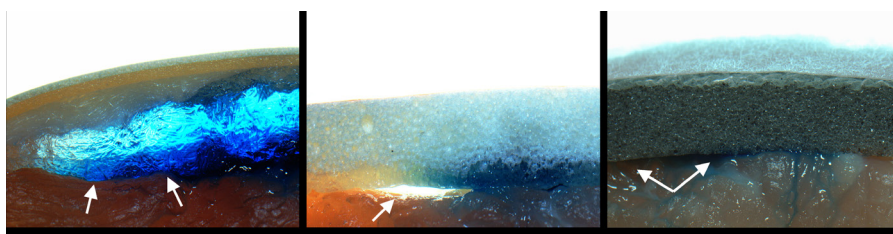


Figure 6. GF dressing fibres swell to form intimate contact with the uneven wound surface. Foams A (middle) and E (right) both show areas of non-contact with the uneven wound surface. GF dressing fibres swell to form intimate contact (arrowed left) with the uneven wound surface. Foams A (middle) and E (right) both show areas of non-contact (arrowed) with the uneven wound surface.

was observed (Figure 5 left and right respectively). In the authors' opinion, these dressings are more prominent in the market and hence were tested in comparison to the new GF dressing. These results provide visual confirmation of those presented in Figure 3.

Contouring to simulated wound tissue

The images obtained using the

simulated wound tissue model demonstrate that not all the dressings tested were able to conform completely to the contours of the simulated wound surface. As the samples were hydrated with the syringe pump, swelling was observed in all of them. However, the GF-N dressing was shown to gel and as a result of the softness and pliability of the gel, was able to contour intimately with the

simulated wound bed/dressing interface (Figure 6). No evidence of pockets or dead spaces filled with fluid was found at the GF-N dressing/simulated wound interface.

In contrast, both foams A and E demonstrate that there was limited contouring at the simulated wound/dressing interface, and there was also evidence of small areas of fluid-filled dead spaces. Similarly, pools of excess fluid were shown to be present (Figure 6) with both foams A and E.

Lateral spread

Results demonstrate that the lateral spread of fluid for the GF-N dressing was significantly less ($p < 0.005$) than for the non-gelling foam dressings tested (Figure 7).

Bioadhesion

The cell count results for the dry central pad area of each non-adhesive dressing demonstrate that the GF-N dressing had significantly less fibroblast adhesion ($p < 0.001$) when compared with all the non-gelling foam dressings tested (e.g. foams A, C, E, G and H). A similar pattern was seen when the central padded area was hydrated, and even though foams G and H demonstrate a marked reduction in fibroblast adhesion, all the non-gelling foam dressings were still shown to have significantly greater cell adhesion when compared to the GF-N dressing ($p < 0.001$) (Figure 8).

The results for the adhesive border area of each dressing demonstrate that the GF-A dressing caused significantly less bioadhesion than foam A ($p < 0.001$), but there was no differences recorded against foams D and F (Figure 9).

Fibroblast contraction cell culture studies

The results presented in Figure 10 demonstrate that while all the dressings were shown to be significantly different ($p < 0.001$) to the control (i.e. no dressing applied), the GF-A dressing was shown to be significantly less detrimental to fibroblast contraction than all the other dressings tested ($p < 0.001$) in this wound model.

Cell viability

These results demonstrate that all the dressings were significantly different to the control ($p < 0.001$). However, the GF-A dressing was $>80\%$ cell viability after 96 hours and this was shown to be significantly less detrimental to the cells in this model when compared to all the other dressings tested ($p < 0.001$) (Figure 11).

Discussion

Healing recalcitrant wounds can take a long time due to many complicating factors, one of which is the necessity for long-term exudate management, which needs to be provided to ensure that patients have an acceptable quality of life (Graham, 2004). Exudate management has historically been linked to the absorptive capacity of the dressing, and while this is an important aspect, absorption alone is not the answer — often there is a need for supplementary exudate management strategies.

In wounds where exudate is considered a problem, it is important that dressings are part of the overall treatment regimen. It is also vital that at each dressing change the wound is re-examined to confirm that the dressing is dealing with the changing environmental conditions of the wound. It is important that dressings address not only the absorption aspects of wound exudate, but also provide an environment that is conducive to healing (Dowsett, 2008). Equally important is the dressing's ability to provide an optimal moisture balance at the wound interface, which is likely to lead to reduced wound bed maceration and also provide protection to the surrounding periwound area (Bishop et al, 2003).

However, there are many variables involved in exudate management and these need to be addressed in order to meet clinicians' needs and improve clinical practice. For example, to help reduce levels of maceration, it is important to understand how different dressings deal with fluid absorbency, wicking characteristics, fluid retention and 'breathing off' fluid (although this may lead to dressings

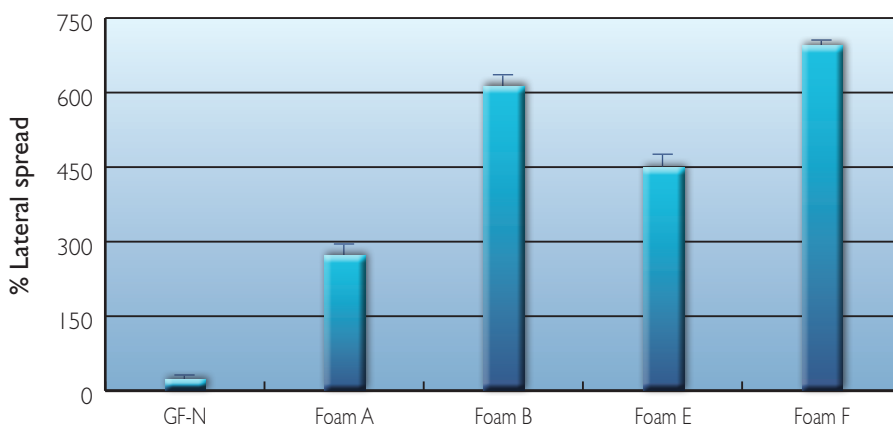


Figure 7. Percentage lateral spread for GF-N and other foam dressings.

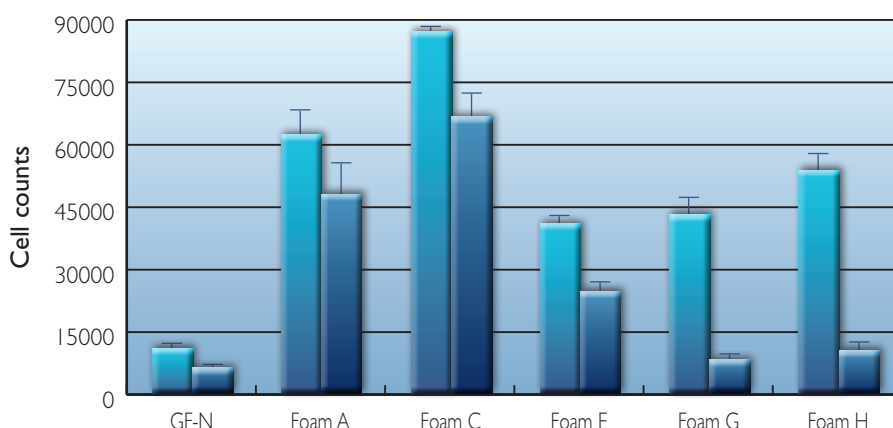


Figure 8. Bioadhesion for central padded area, both dry (light blue) and hydrated (darker blue).

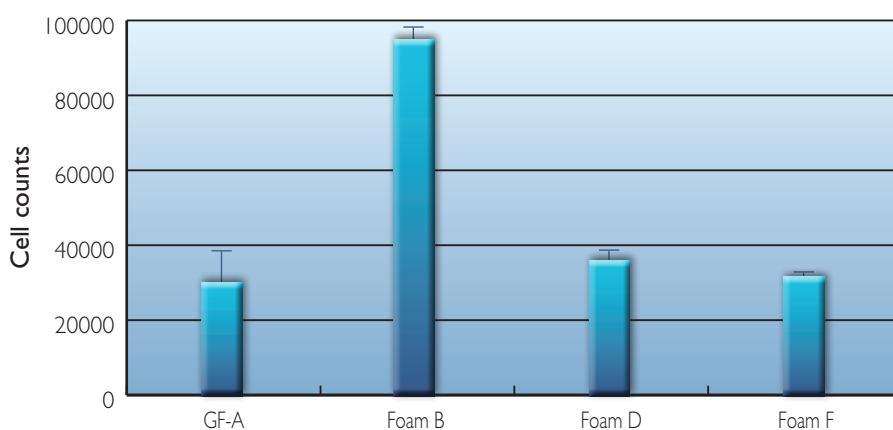


Figure 9. Bioadhesion for adhesive area.

drying out if too much moisture is lost from the wound). Dressings containing Hydrofiber® Technology have been shown to provide fluid retentive properties (Parsons et al, 2005), which ensures that harmful components often found in exudate such as bacteria and proteolytic enzymes become locked within the gelling structure (Newman et al, 2006; Beldon, 2008). Dressings that

have these properties are also likely to reduce the chance of fluid leakage, which can affect immediate periwound tissue and lead to further breakdown in the integrity of the surrounding epidermal tissue (Figure 12) (Walker et al, 2008).

These *in vitro* studies have attempted to address some of these

issues and suggest that HCDs are an important new addition to the modern wound dressing formulary, combining the properties of other foam dressings (e.g. absorption) with Hydrofiber® Technology. These dressings not only provide sufficient absorption of exudate, but can also help maintain moisture balance via three mechanisms:

- ▶▶ The transition of fibres in the contact layer into a moist gel
- ▶▶ The intimate conformability of the gel to the wound bed
- ▶▶ The ability of the dressing to retain fluid under pressure.

All three aspects have been demonstrated in the *in vitro* studies presented here.

Non-gelling foams are considered simple absorptive dressings, which have the capacity to absorb fluid into so-called 'open' spaces within their structure (WUWHS, 2007; Dowsett, 2008). Any fluid that is contained in these spaces is considered free fluid, and therefore unbound to the dressing. Consequently, when these dressings have pressure applied to them, the fluid is released from these spaces and may leak from the dressing. Failure to sufficiently manage exudate can lead to skin maceration and excoriation (Figure 12), which in turn increases the risk of infection and adds to the distress of the patient (Beldon, 2008). It is therefore important that wound dressings have the capability and capacity to retain fluid, particularly under pressure.

The data presented here demonstrates the properties of HCDs to provide free fluid absorption (Figure 2). They also lock away fluid so that when pressure is applied the majority of that fluid is retained within the dressing (Figure 3) rather than being released, as was demonstrated in foams A and E (Figure 5).

The authors suggest that if applied pressure was reduced or removed, these HCDs would continue to absorb fluid until their free swell capacity was reached. This behaviour is a characteristic of all Hydrofiber-based dressings, and is the direct result of the transition of dry fibres from a

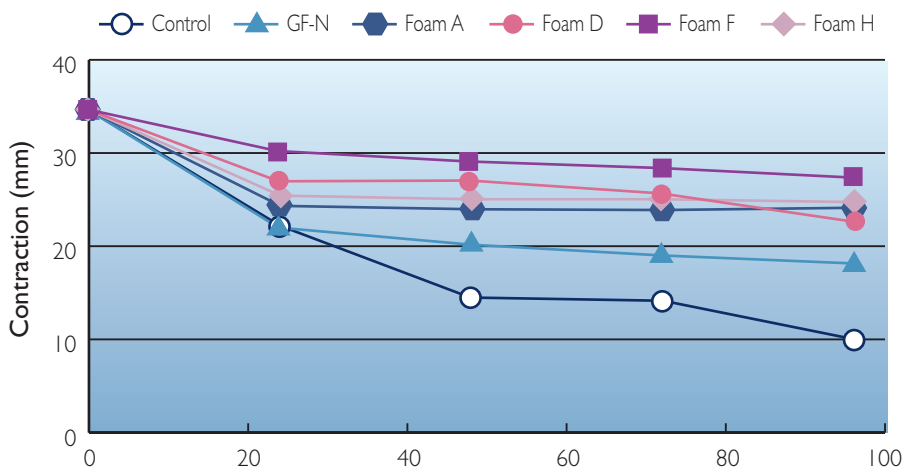


Figure 10. Measurements of fibroblast gel contraction.

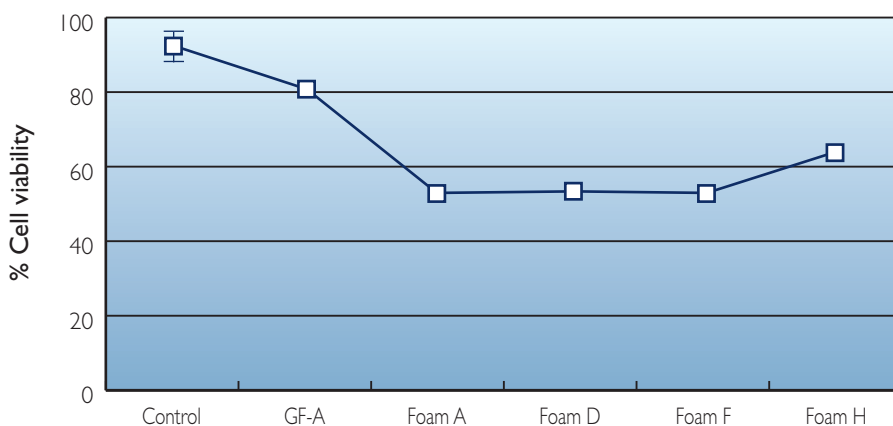


Figure 11. Percentage cell viability.



Figure 12. Exudate management with a typical foam (left) and a gelling foam dressing (right). Left photograph reproduced with kind permission of Arther Newton, Dermatology Liaison Nurse, Southern General Hospital Glasgow. Right photograph reproduced with kind permission of Esperanza Manzanero, Head of Nursing and Salomé Fernández, Registered Nurse, both at Centros Socio Sanitarios Medinaceli, Madrid, Spain.

solid to a gelled state. A previous study has shown that the dry fibres expand approximately ten-fold as fluid is absorbed (Newman et al, 2006). This fluid is absorbed and retained within the gelled cohesive structure as opposed to remaining free — free

fluid is found in the more open-spaced structure of non-gelling foam dressings. Consequently, with these type of non-gelling dressings, it is likely that a reduced amount of fluid will be retained when pressure is applied (Figure 5).

The ability of Hydrofiber-based dressings to lock in wound exudate means that harmful components such as bacteria (Walker et al, 2003) and proteolytic enzymes (Walker et al, 2007), that may be present in the fluid, are removed from the wound bed environment. This has important clinical implications, as chronic wound exudate is considered a 'corrosive' biological fluid (Chen et al, 2004). Thus, not only is the bulk of the exudate and its harmful components locked inside the dressing, but the gelled surface is also in intimate contact with the wound bed (Figure 6), ensuring that it remains moist.

In contrast, the more 'rigid' and inflexible foam dressings demonstrate a reduced degree of conformability. A previous *in vitro* study has shown that where there are areas of dressing non-conformability, the likelihood of bacterial accumulation and proliferation is increased (Jones et al, 2005).

Foams are thought to have many acceptable characteristics, such as ease of use, provision of thermal insulation, non-adherence, versatility and effective absorption of large amounts of fluid (Thomas, 1993). This latter characteristic has been shown in the *in vitro* studies featured in this article (Figure 3). However, in the management of complex chronic wounds, it is important that other aspects of exudate management should not be overlooked. Foams are also effective in clinical areas such as comfort, protection and pain reduction (Gottrup et al, 2008).

Pain is often associated with dressing removal and this may be related to the dressing's interaction with cellular components in the wound bed (i.e. fibroblasts). When applied to wound tissue in the early stages of healing, wound dressings are likely to be in intimate contact with cells involved in the healing process. Consequently, these cells can become attached to the dressing and re-injury to the newly formed granulation tissue and the surrounding epithelial tissue may occur when the dressing is removed.

Cells can also become adhered to the dressing through interaction with extracellular matrix proteins (e.g. fibronectin) or via a dressing's component substrates (Cochrane et al, 1999). Alternatively, this may be due simply to passive cellular adhesion, which can occur if the applied dressing is allowed to dry out. One possible reason for this may be increased protein concentration at the wound/dressing interface, which may lead to the formation of what has been called

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a 'viscous glue', resulting in the dressing adhering to the wound/cell surface (Cochrane et al, 1999). It is possible that either or both of these adhesion mechanisms could occur at dressing application.

Limitations

A bioadhesion *in vitro* model has been used in these studies to assess cell adhesion to dressings (Cochrane et al, 1999). It is acknowledged that any *in vitro* model will have limitations and it is important that these are recognised and accepted in data evaluation. The method described here simply used a single-cell culture approach that does not take into account the numerous other cellular and adhesion-associated events that take place in a wound environment.

There are also many clinical factors to consider — the act of physically applying a wound dressing, the use of secondary dressings, the length of time a dressing may be on a wound and levels of wound exudate are all recognised as important parameters.

These dressings were only in place for 24 hours, which may not truly reflect clinical wear-time, but for comparative purposes each dressing was applied for the same length of time. However, few of these factors, if any, can be simulated by *in vitro* methods, although this model does allow specific cell and dressing interactions to be investigated in detail in a controlled environment.

The bioadhesion results demonstrate that some foams, particularly when hydrated, have low cell adhesion (Figure 8), which may substantiate the association of foams with pain reduction. However, it is also clear from the data in this model that the non-adhesive HCD demonstrated significantly less bioadhesion ($p < 0.001$) when compared to the other foams tested, regardless of whether the dressings were dry or hydrated.

In the case of the hydrated dressings, this may be due to the slow dehydration rate, which helps to maintain a moist cell-dressing interface and minimises passive adhesion. In the dry dressings, one possible explanation may be the lower contact surface area between the dressing and the cell layer, caused by the fibrous nature of the dressing contact layer.

In a recent clinical study, the ease of application and removal of the adhesive HCD was rated as excellent or good in over 90% of the patients studied (Parish et al, 2008). Similar results were seen in a second clinical study, where, in comparison with pre-study dressings, the application of a non-adhesive HCD resulted in significant reductions ($p < 0.001$) in patients' leg ulcer pain, both during the wear time of the dressing and upon removal (Vanscheidt et al, 2007).

Under normal skin conditions, the *stratum corneum* provides a distinct barrier between the viable epidermis and the external environment. The process of re-epithelialisation is an integral part of the wound healing process, but this can be compromised through the application of inappropriate dressing regimens, which

may lead to skin maceration and/or excoriation. As a result, periwound skin may have compromised barrier integrity (Walker et al, 1997). A recent study has shown that the epidermal barrier integrity of peri-ulcerated tissue has been shown to have an approximate five-fold impaired barrier function (Walker et al, 2008).

From a clinical perspective, newly-formed epidermal tissue is often mistaken for maceration as it appears opaque or white. This is because it has not had time to fully develop its more natural pink appearance (Bolton et al, 2000). Therefore, some wounds may have newly-forming epidermal tissue removed through the mistaken fear on the part of clinicians that the tissue has become macerated. Consequently, in an attempt to reduce the perceived risk of further maceration, some clinicians will mistakenly remove moisture-retentive dressings in favour of more conventional dressings (Bolton et al, 2000).

Conclusion

The importance of good exudate management cannot be overstated. It has recently been said that: 'Exudate needs to be managed to maximise its benefits to the wound and the patient' (WUWHS, 2007).

Equally important is the choice of the most appropriate wound dressing. This should provide the best possible wound healing environment. **WUK**

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Key points

- ▶▶ Hydrofiber® Technology products provide gel blocking properties.
- ▶▶ New Hydrofiber® cover dressings (HCD) demonstrated high fluid absorption capacity.
- ▶▶ HCDs provide fluid retention under pressure (e.g. >90%).
- ▶▶ HCDs are easy to apply and provide non-traumatic dressing removal, as shown by both *in vitro* and *in vivo* studies.

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