Journal watch

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Wolcott R, Sanford N, Gabrilska R, Oates JL, Wilkinson JE, Rumbaugh KP. Microbiota is a primary cause of pathogenesis of chronic wounds. Journal of Wound Care 2016;25:S33–S43. doi: 10.12968/jowc.2016.25.Sup10.S33

Conjecture exists regarding the role and process through which microorganisms on a wound surface contribute to wound chronicity and infection. In the paper by Wolcott and colleagues (2016), theories regarding the role of microorganisms in wounds are described and an investigation of whether wound microbiota actively spreads in other host environments is presented. Wound microbiota is defined as the collective of microorganisms on wound surfaces. Specifically, this study sought to determine wound microbiota viability, dispersal and infection capability when an animal model was inoculated with microorganisms from chronic human wounds.

The trial obtained wound exudate (using filter paper and Adaptic Touch Dressing) and wound slough (biopsy) from 25 human wounds, and from another 25 human wounds it obtained wound slough alone. Wounds were of any aetiology of ≥ four weeks that were highly exudating. Mice were administered a full-thickness surgical excision wound that was, with the exception of controls, inoculated with the human samples.

The majority (81%) of mice generated polymicrobial communities within the wound. Ninety-three per cent of the microbes from humans were propagated in the mouse model although the relative percentages of species did differ, and 91% presented with a clinical appearance associated with a chronic wound. Control mice did not develop microbiota during the four-day follow-up period. No difference between control and intervention mice was observed with respect to wound healing.

The authors concluded that wound microbiota sourced from wound exudate and slough is capable of propagating on new host tissues with a comparable microbial profile. Clinical implications for risks of contamination were specifically highlighted and the results were used to postulate, on theoretical grounds, that wound microbiota is a main cause underlying the pathogenesis of chronic wounds.

A key limitation of the study was the absence of human samples without clinical signs of chronicity or infection. However, the detrimental impact of obtaining a biopsy from these wounds is a recognised and reasonable barrier in the field. The paper does, however, also highlight the value of developing reliable, accessible, and non-invasive approaches to tissue sampling, such as the use of the filter paper, to enable ongoing investigations to further explain the role and process of microbiota on wound chronicity and infection.

Park E, Long SA, Seth AK, Geringer M, Xu W, Chavez-Munoz C, Leung K, Hong SJ, Galiano RD, Mustoe TA. The use of desiccation to treat *Staphylococcus aureus* biofilm-infected wounds. Wound Repair & Regeneration 2016;24(2):394–401. doi:10.1111/wrr.12379

Moist wound healing is a cornerstone of contemporary wound management. The investigation outlined in the paper by Park and colleagues (2016) therefore presents a controversial perspective regarding the role of moisture and biofilm management. The research built upon the authors' prior observations that biofilm colonised wounds left to open air desiccation (dehydration) healed more rapidly than those wounds that were kept moist. This *in vitro* investigation cultured *Staphylococcus aureus* biofilms on the ventral surface of 10 rabbit ears. Treatment conditions included wounds left to open air desiccation, 10 µL saline treatment, and a comparison of Manuka honey UMF®5+, Comvita, New Zealand, and molasses (proposed to be a comparable consistency to honey but without the antimicrobial properties). Clean wounds without biofilms were also considered for some comparisons. Wounds in the open air desiccation arms were left uncovered and the remaining wounds were dressed with Tegaderm. Treatments were applied 6, 8, and 10 days postoperative and rabbits were euthanised 12 days postoperative. Tissue samples were obtained from both the scab of the wound and the wound bed using biopsy.

Wound beds tended to yield smaller viable bacterial counts of *S. aureus* than scab samples. The wound bed of saline-treated wounds yielded the most bacteria (1.82×10^7 CFU) compared to open air (2.25×10^2 CFU), molasses (8.10×10^4 CFU), and honey (3.75×10^4 CFU). Wounds treated with open air desiccation, molasses, honey, and clean wounds all had significantly less up-regulation of inflammatory markers of TNFα and IL-1β. Honey was found to inhibit *S. aureus* growth.

The authors subsequently acknowledged that the *S. aureus* growth was performed using planktonic-phase bacteria rather than biofilm phase bacteria, for which resistance to the treatment conditions may vary considerably. The authors also cautioned against the incorporation of desiccation

in chronic wound care until further research has been conducted. Not explicitly addressed was the performance of the molasses-treated wounds compared to saline, although the authors did reference the evidence regarding agents with high sugar content (hyperosmolar agents) in wound healing. Despite these limitations, this study raises questions about the impact of moisture level on biofilm development and current approaches to biofilm management that warrant further investigation.

Woo KY. Physicians' knowledge and attitudes in the management of wound infection. International Wound Journal 2016;13(5):600–604. doi:10.1111/iwj.12290

The paper by Woo (2016) reports survey results of physicians working in a variety of Canadian provinces and territories. The aim of the research was to explore physician self-rated knowledge and learning needs with particular reference to wound infection and biofilms. A customised survey instrument was developed that was reviewed by interprofessional educators and wound specialist clinicians. The physician population was sampled from registered users of an online continuing medical education platform. Of the 250 clinicians invited by email to participate, 88 completed the survey (a 35% response rate), the majority of whom were family physicians (77%).

Wound aetiology seen in practice varied but included diabetes-related foot ulcers, traumatic wounds, burns and post-surgical wounds. All participants stated prior experience assessing and managing wound infection; 80% of physicians reported encountering a presentation of wound infection at least weekly. Current knowledge was consistently lower than desired knowledge with biofilm-associated areas of knowledge rated as the least well understood amongst physicians. The areas that physicians nominated as learning needs included wound debridement and cleansing, when to use antibiotics, when to start and stop treatment, topical antimicrobials, and barriers to wound healing. The author concluded that the research confirmed a knowledge gap and a desire to know more amongst physicians with respect to wound infection. The frequency of physician-reported encounters with wound infection is also noteworthy.

The author acknowledges that a limitation of the study is that the sample may not be representative of all physicians. Reliance on self-rated wound infection knowledge is also a limitation. The presentation of survey results is challenging to consistently interpret due to the varied use of percentages and means. The survey, nevertheless, was successful at engaging medical practitioners that can be difficult to involve in research especially about a clinical field that is frequently underestimated for its importance and relevance.