Critical colonisation: Clinical reality or myth?

All wounds healing by secondary intention, in particular the 'chronic' wounds, contain micro-organisms (MOs). The Wound Infection Continuum (WIC) (Kingsley et al, 2004) identifies various stages of wound colonisation extending from 'colonisation' through 'critical colonisation (CC)' to 'spreading infection'. These wounds will heal in the colonised state. However, CC has been equated with delayed healing, and, as such, has been termed local infection by some. This has given rise to a vigorous debate on the terms used in the WIC and their clinical relevance.

Recently, wound microbiology studies have shed new light on the influence of colonisation and infection on healing. The publication of criteria for wound infection (Cutting and Harding, 1994) has similarly helped diagnosis. However, these included 'delayed healing' as an infection criterion, provoking debate on the pathophysiology of this situation, and on the clinical relevance of the microbiological state of CC. It is important that all involved in wound management appreciate the need to recognise infection and delayed healing, in order that appropriate treatment may be implemented. While CC has yet to be scientifically proven, or clearly defined beyond reasonable doubt, this state is becoming accepted as a clinically distinct situation (Jorgensen et al, 2005). Here, Keith Cutting and Andrew Kingsley give their opinions on CC. RW

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Is critical colonisation (CC) a concept for academic discussion rather than a clinically meaningful state?

KC: The concept of CC is rooted in practice. Davis (996) theorised that delayed healing in venous leg ulcers was related to an imbalance between the bacterial load and the local defences, but without the host response that usually results in signs of infection. This concept is currently being subjected to initial empirical scrutiny but requires prospective/longitudinal investigation. Until this occurs, the significance of CC both clinically and scientifically will remain in a tenuous state.

AK: There is no doubt in my mind that it is both things. It represents a situation that occurs in many chronic wounds. There are many reasons for chronicity, but I believe that bioburden is a significant contributor. These static wounds exhibit clinical signs which are the clues that will help to unlock chronicity. It is these clinical cues that need academic debate because clinicians use the presence of different signs, of varying severity, and in different combinations to prompt them into treatment action. Debate can lead to greater consensus in the wound care community and, in turn, lead to simplification of education for generalist practitioners, who in reality, do the vast majority of wound care.

Does CC have any scientific foundations in microbiology, biochemistry or physiology?

KC: Any proposal/discovery/development has to be viewed within a temporal context, therefore the foundations on which CC is currently built will undoubtedly undergo revision as we learn more. Currently, a circumspective approach is most likely to be held by those involved in wound healing until foundations are firmly established. Bacteria produce matrix metalloproteases which augment chronicity by lysing protein and thus delaying healing. Similarly, bacterial virulence factors can cause host tissue damage. It has yet to be confirmed that biofilm presence (bacterial communities) is related to CC (delayed healing). These communities exist within a self-generated exopolysaccharide matrix, which affords them protection from host defences and could account for unconventional host response. Other virulence determinants, such as quorum sensing and bacterial synergy, could be integral to CC. We should not forget that pathogens can initiate damage across a spectrum of immune responses.

AK: I think so, although I accept cause and effect evidence is limited. CC only applies to wounds healing by secondary intention, because closed wounds either heal or generate an acute infection evidenced by cellulitis and pus. For the open wound, we know that there is an inevitable and progressing contamination. I use the word inevitable despite growing talk of prophylaxis with antiseptic agents, since it is unlikely to exclude all bacteria from a wound, but may have beneficial effects in reducing quantity or controlling pathogenic species. Again, hard clinical research evidence for this is limited, but it is not an unreasonable conclusion at present. Contamination progresses to colonisation which is not always detrimental since organisms can be cultured from healing wounds. There is

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some evidence to show that the more species of bacteria cultured from the surface fluids of a wound, the lesser the chance of healing. Additionally, some *in vitro* work on anaerobes suggests mechanisms by which they are thought to hinder healing *in vivo* (Stephens et al, 2003). Clinically, this work on anaerobes is supported by the often good response seen to static wounds when anti-anaerobe antibiotics are added to ineffective aerobe-only regimens. I accept there is a problem with the 'seeing is believing' style of observational evidence, but it remains reasonable in the absence of a better guide to clinical practice. To my knowledge there is no reliable specific biochemical or cellular marker that can be used as a diagnostic test for CC, but that does not disprove it. Evidence supporting the theory of CC is mostly indirect and is applied microbiology rather than direct evidence gathered from open wounds in a state of delayed healing.

Is a linguistic obfuscation, complicating a simple situation of local wound infection, detrimental to diagnosis and treatment?

KC: Obfuscation or clarification – only time will tell. Undoubtedly, CC is a local, non-spreading infection. Whether it shares all of the characteristics of local infection (Cutting and Harding, 1994) remains to be seen. If there is no difference (microbiologically, histologically and immunologically) between CC and local infection, as currently defined, then the term CC should be abandoned.

AK: I would choose clarification rather than obfuscation, and enlightening over detrimental because when I have used the term at educational events many delegates have expressed new found understanding of clinical problems they regularly face. I endorse the term CC over local infection precisely because it does not use the word infection, because infection is inextricably linked in the current clinical mind with the need to prescribe an antibiotic. Due to resistance there is a global imperative to reduce the use of these drugs, so education leading to early diagnosis of a bioburden state and treatment with topical antiseptic dressings is beneficial. Local infection is one step on along the infection continuum from CC, and is a highly valuable term defining a restricted, localised, non-spreading cellulitic reaction. If we define and reserve this term for this situation that is distinct from CC, then researchers could investigate whether antiseptics alone are effective treatment, or, if it genuinely needs antibiotics. In addition, it might be possible to establish if the combined use of antiseptics and antibiotics achieves faster resolution of the problem. This would be ground-breaking work.

Do you believe that the use of the term CC aids or hinders clinical decision-making?

KC: It is said that beauty is in the eye of the beholder. The beauty of CC (if it exists) is that it may elicit signs of its presence with which we have yet to come to terms. Epithets such as glazed, slimy, and foamy have been used. Until definitive characterisation has been established, clinicians can proceed with only suspicion as to its presence.

AK: Whether it is right or wrong, the term has certainly changed my decision making, allowing me to use the range of antimicrobial products now available. Without the term and the underpinning concept, I would have been reluctant to return to the use of antiseptics which were previously denounced in nursing circles as agents of pain and cell cytotoxicity in their previous formulations.

Have you observed the use of the term in clinical situations and what was your impression of its impact?

KC: I have not observed its use clinically, beyond that of my nursing colleagues involved in tissue viability. When I mention the possibility of delayed healing and a relationship with CC in the presence of non-nursing colleagues, eyebrows are raised.

AK: Yes, it is certainly developing into common language in my area among nurses in community settings. This is valuable because common understandings can shorten the time to suggesting possible solutions. The continued use of the term in everyday nursing language suggests it has some sort of clinical resonance. **WUK**

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