A main concern for clinicians prior to applying a topical agent on an open wound is safety. Agents that are cytotoxic or cause delay in wound healing are used with reservation. The strongest argument against the use of antiseptics on wounds is that antiseptics have been found, primarily using *in-vitro* models, to be cytotoxic to cells essential to the wound healing process, such as fibroblasts, keratinocytes, and leukocytes.[29-31] However, this cytotoxicity appears to be concentration dependent, as several antiseptics in low concentrations are not cytotoxic, although they retain their antibacterial activity *in vitro.*[27] Since the *in-vitro* results are not always predictive of what may happen *in vivo,* numerous studies have been conducted on animal and human models. The results of these studies are conflicting and will be presented later in the article.

**Cadexomer iodine.** The efficacy of cadexomer iodine has been shown in both animal and human models (Table 2). Mertz, et al.,[48] examined the effect of cadexomer iodine dressing on partial-thickness wounds in specific pathogen-free pigs contaminated with or without methicillin-resistant *Staphylococcus aureus* (MRSA). Applied daily, cadexomer iodine was found to significantly reduce MRSA and total bacteria in the wounds in comparison to no treatment control and vehicle (cadexomer) at all time points studied (1, 2, and 3 days after inoculation). The reduction was most pronounced at day 3.

In an uncontrolled small study series (*n* = 19), Danielsen, et al.,[49] used cadexomer iodine in ulcers colonized with *Pseudomonas aeruginosa* and found negative cultures in 65 percent and 75 percent of patients after 1 and 12 weeks of treatment, respectively.

### Table 3. Hydrogen peroxide

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Author** | **Wound Type** | **Species** | **Number of Wounds Treated** | **Control** | **Effect on Healing** | **Effect on Infection** |
| Bennett, et al.[107] | Partial thickness | Pigs | 48 (8 wounds for each group) | Mafenide acetate; sodium hypochlorite; povidone iodine; acetic acid; no treatment | No effect (69% reepithelialization after 4 days vs. 69% for the no treatment group) | Decreased bacterial counts, (<105 vs. >105 at the control group, statistical difference not calculated) |
| Gruber, et al.[52] | Partial and full thickness | Rats | 40 (20 for each group) | Saline | Increase (10.2 and 8.4 days mean healing time vs. 12.4 and 9.5 for saline in partial-thickness wounds in rats and humans; 17 vs. 19.5 in full-thickness wounds in rats, *p* < 0.05 for the animal study, nonsignificant difference for the human study) | N/A |
| Humans | 20 (10 for each group) |
| Lau, et al.[81] | Appendectomy | Humans | 217 (109 HP, 108 control) | No treatment | N/A | No effect (21 pt developed infection vs. 26 of the control group) |
| Leyden, et al.[82] | Blister wounds (contaminated) | Humans | 144 (24 volunteers with 3 wounds per forearm) | No treatment, triple antibiotic | No effect (14.3 days for healing vs. 13.2 and 9.4 in the no treatment and triple antibiotic group) | No effect (6.7 log *S. aureus* counts vs. 7.1 and 0.4 in the no treatment and triple antibiotic group) |
| Tur, et al.[80] | Ischemic ulcers | Guinea pigs | 34 | Placebo cream | No effect (visual evaluation of percent nonnecrotic wound surface showed no difference between groups); increased blood flow (vascular perfusion measured with laser Doppler velocimeter was increased, *p* < 0.01) | N/A |

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