

Letter to the Editor

Dear Sirs

We read with great interest the review by Michael Ng (*International Wound Journal*, 2010;7(1):55–61) focused on the role of mast cells in the early stages of wound healing (i.e. inflammation and proliferative phase). Interestingly, the author emphasises the importance of regulating mast cell function to optimise wound healing.

Few years ago, we extensively reported on the pivotal role of mast cells in wound healing (1). Not only the evidence on the 'homeostatic orchestrative' role of mast cells was described, but the potential detrimental effect of an excessive and uncontrolled mediator release (hyperdegranulation) was also reported in our review article (1).

In the paper by Ng, drugs that can optimise wound healing through the inhibition of mast cell function, such as ketotifen, sodium cromoglicate, chlorphenamine and cimetidine, are discussed. In our opinion, an interesting and new approach is missing from this scenario, that is, the downmodulation of mast cell degranulation by the so-called aliamides (2). Aliamides are a family of fatty acid amides whose name derives from the autacoid local injury antagonism mechanism, through which they exert their effects, mainly by downmodulating mast cell function (2,3). Particularly, the aliamide adelmidrol has been shown to reduce skin mast cell degranulation, when topically applied on experimentally induced skin wounds (4). Furthermore, an increase in the percentage of re-epithelialisation and a higher density of elastic fibres were also observed (5). Finally, the ultrasonographic assessment of experimental wounds showed that the topical application of the aliamide adelmidrol significantly decreased the wound volume, thus improving wound healing over time (6). The wound healing-promoting effect of adelmidrol is believed to depend upon the proved ability to downmodulate mast cell degranulation, thus fine-tuning the well-known succeeding and partially overlapping phases of wound healing. This effect was recently confirmed in a model of chronic inflammation, that is, the carrageenin-granuloma model (7). Histological evidence and measurement of

the amount of mediator released by mast cells confirmed the ability of adelmidrol to downmodulate mast cell function (7). In addition, granulomatous tissue formation, neoangiogenesis and leucocyte infiltration were also decreased by the treatment (7). Taken together, these results argue that the aliamide adelmidrol may represent as useful tool in wound healing management.

Respectfully submitted,
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