

## ***Pseudomonas aeruginosa* burn wound infection in a dedicated paediatric burns unit**



**To the Editor:** We read with much attention and a great deal of interest the findings of the retrospective review by Coetzee *et al.*<sup>[1]</sup> suggesting that many South African paediatric patients develop infections after burn injuries. Furthermore, the authors state that there was a significant resistance to topical therapy, along with some resistance to antimicrobials, in the patients treated. Will healthcare providers be able to use a non-resistant agent to reduce the chances of patients acquiring

infection after a burn injury in the near future? Yes, this may be possible with the help of a novel molecular peptide called SS-31 that specifically acts to assist in the recovery of mitochondrial function, which is severely damaged by burn injuries.<sup>[2]</sup> This is important, since the mitochondria play a vital part in patient recovery and may help to reduce healing time and hence also the risk of infection.

On a molecular level, damaged mitochondria increase oxidative stress and hamper oxidative phosphorylation in skeletal muscles

and other burned areas of the body, which may limit their ability to heal. Peptide SS-31 potentially has the ability to alter post-burn physiology and could be a powerful tool in preventing post-burn infections. Moreover, SS-31 increases adenosine triphosphate synthesis and decreases reactive oxygen species,<sup>[3]</sup> thereby alleviating symptoms and aiding faster recovery from burn trauma. Again, this will decrease healing time and reduce vulnerability to infection. This peptide is still in its experimental phase, but if it is approved it may revolutionise the way burned patients are treated, along with reducing post-burn complications such as infection.

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**Dr Coetze responds:** We thank the above authors for their comments. Burn injury and immobilisation have been shown to be associated with mitochondrial dysfunction in animal models.<sup>[1,2]</sup> Mitochondrial dysfunction and apoptosis in skeletal muscle of burn animal models peaked on days 3 - 7 after the burn injury.<sup>[3]</sup> It is hypothesised that immobilisation mitochondrial injury is due to the production of reactive oxygen species by the mitochondria of the immobilised skeletal muscle.<sup>[1]</sup>

A series of mitochondrial-targeted cytoprotective peptides have been designed by Szeto and Schiler.<sup>[1-3]</sup> Of these 'SS-peptides', SS-31 has been shown to be protective against mitochondrial dysfunction in skeletal muscle after days 3 - 7 in burn animal models, as well as in skeletal muscle of immobilised animal models.<sup>[1,3]</sup> There is great interest in and much research targeted towards finding novel mechanisms to attenuate the effects of burn injury, as well as to improve recovery. The SS-31 peptide is certainly an exciting prospect in this regard. It has, however, only been shown to alter mitochondrial damage in skeletal muscle of burn animal models, and has not been studied in humans. To hypothesise that treatment with peptide SS-31 in the burns patient will reduce infectious complications and improve outcomes is probably premature and certainly not based on any current evidence.

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