



SOD B[®] & Skin Repair

Impaired healing wounds affect about 3 to 6 million people in the United States. Aged people (65 years old and older) account for 85% of these events. Wound healing is achieved through 3 precisely and highly programmed phases: inflammation, proliferation, and remodeling. Many factors can interfere with one or more phases of this process, causing impaired wound healing. An over-release of Reactive Oxygen Species (ROS), and an abnormal prolonged inflammatory reaction are involved in the delayed healing process. By providing both antioxidant and anti-inflammatory properties, Superoxide Dismutase (SOD) appears to represent an efficient solution for improving skin healing.

Delayed healing & free radicals release

Following tissue injuries, an excess production of Reactive Oxygen Species (ROS) is observed¹. Once activated, inflammatory cells produce large amounts of ROS, especially superoxide anions (O₂⁻) as part of their defence mechanism^{2,3}. Although this process is beneficial in the absence of substantial wound contamination, increased levels of ROS, during an excessive contamination, inhibit cell migration and proliferation and cause severe tissue damages⁴.

Skin healing alterations and inflammation

During normal wound healing, inflammation processes are stopped once the filling is ensured. However, in several pathological cases, the infiltration by inflammatory cells is permanent, leading to the generation of continuous mediators such as ROS growth factors and cytokines (Figure 1)⁵.

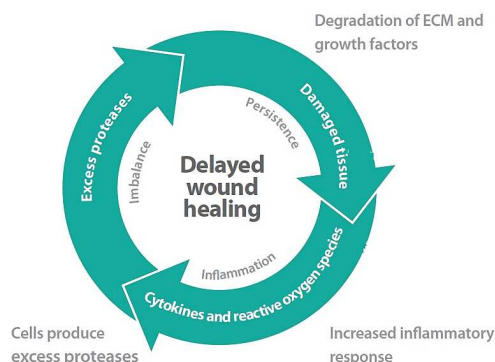


Figure 1: The vicious circle of delayed wound healing.

This provokes fibroblast proliferation and differentiation into myofibroblasts, improving the inflammatory response until a fibrosis state⁶. This chronic activation is due to the generation of mediators, such as ROS, growth factors or cytokines (TGF-β1) which lead to the inhibition of apoptosis^{7,8}.

Impaired skin healing & modern life

Many factors interfere with one or more phases of the healing process, causing impaired tissue repair. This includes age⁹, stress¹⁶, obesity¹⁰, alcoholism¹¹, smoking¹², and UV exposure¹³. These factors act by increasing the inflammatory process and the release of ROS in the wound site. Interestingly, a daily exposure to these exogenous factors is linked to lower endogenous SOD levels¹⁴⁻¹⁷. This could explain the impaired skin healing process.

An optimal wound management consists in preventing the ROS production, reducing the inflammatory response, and preserving skin from aggravating exogenous factors (Figure 2).

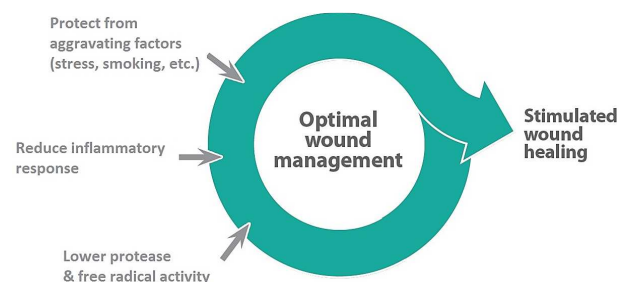


Figure 2: Optimal wound management.



SOD inhibits aggravating factors

An SOD oral supplementation represents an efficient approach to inhibit the impact of stress on the impairment of the wound healing process. Two major clinical studies have already reported a significant reduction of perceived stress (-21.7%) in people orally supplemented with a bioactive and natural melon SOD (SOD B® at 140 IU SOD) (Figure 3)^{18, 19}.

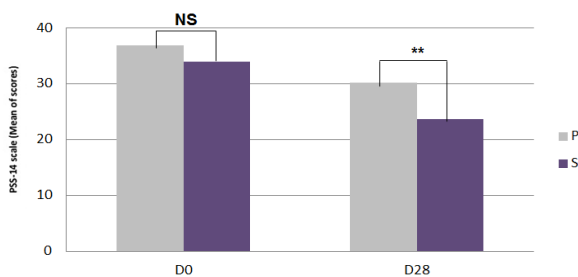


Figure 3: Effect of an oral SOD supplementation (S) on perceived stress compared to placebo (P)¹⁸.

SOD reduces wound size

SOD has been reported to be efficient in reducing the size of post-burn wounds. Topically applied, SOD reduces the size of post-burn wounds. At 24 h following trauma, the lesion sizes of the untreated animals extended to 165% and of those treated with SOD expanded less to 102%, highlighting a significant lower dilatation of lesions^{20, 21}.

SOD accelerates re-epithelialization

SOD applied topically on mice for 3 weeks has been shown to accelerate reepithelialization after skin burn. SOD acts by stimulating the expression of Vascular Endothelial Growth Factor (VEGF), essential for the synthesis of ExtraCellular Matrix (ECM)²².

SOD reduces skin swelling

SOD is efficient in reducing skin swelling after trauma. After 24h, the skin thickness of animals topically treated with SOD (human recombinant Cu,Zn-SOD) was 15% less in comparison with controls untreated animals. During the three days of observation, the resolution of the skin swelling was 45% in SOD applied animals (n=10) compared to only 15% in control animals (n=10)²⁰.

SOD inhibits collagen over-deposition

Anti-fibrosis properties of SOD have been studied, with more than 130 scientific studies published. SOD has been used as a powerful anti-fibrotic therapeutic agent to resorb radiotherapy-induced fibrosis²³. The anti-fibrotic properties of SOD imply the reduction of the fibrotic block. SOD affects the homeostasis of myofibroblasts by inducing the phenotypic reversion of myofibroblasts into normal fibroblasts. These authors showed that this phenotypic reversion is correlated with the inhibition of TGF-β1 (Figure 4)⁸.

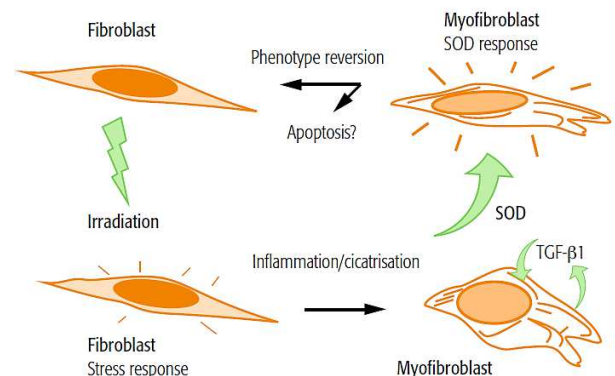


Figure 4: The anti-fibrotic action of SOD⁸.

Conclusion

As the first line of antioxidant defenses, SOD has been demonstrated to:

- Relieve aggravating factor such as stress
- Inhibit fibrosis & collagen overproduction
- Accelerate re-epithelialization
- Reduce post-burn skin swelling

According to its unique way of action, SOD represents an efficient solution for improving skin repair.

Bibliography

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