8. FROM OXIDATIVE STRESS TO BONE HEALING: A BIOCHEMICAL INSIGHT INTO THE FRACTURE RECOVERY PROCESS

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Introduction. Within bone tissue, osteoclasts release oxidative stress (OS) compounds, vital for calcified tissue breakdown and bone healing after fractures. However, imbalances between oxidant compounds like reactive oxygen species (ROS) and antioxidant defenses, may result in bone loss and osteoporosis. Understanding the molecular intricacies of OS in bone tissue provides valuable insights into potential therapeutic approaches aimed at improving fracture recovery process and preserving overall bone health.

Aim of study. To explore the biochemical pathways involved in OS induced damage in bone tissue, impairing fracture healing and enhancing fracture risk.

Methods and materials. The groundwork of this scientific review is based upon a conscientious analysis of 20 publications from established databases like Science Direct, Springer Link, PubMed. All the publications were selected from a period spanning the last five years. Keywords used: oxidative stress, bone health, fracture healing.

Results. ROS, acting as signaling agents, can hinder osteogenic derivation, influencing the dynamic relationship between osteoblasts (OBs), responsible for synthesizing crucial organic and inorganic compounds (collagen, osteocalcin, osteopontin, hydroxyapatite) and osteoclasts (OCs) in bone tissue. Superoxide, as a member of the ROS family, has both physiological (redox signaling) and pathological (pro-apoptotic cascade, cellular necrosis) influence on bone tissue. Its reaction with nitric oxide (NO) produces peroxynitrite, which is highly reactive towards DNA and proteins. Given that OCs constitutively produce NO for their normal function, elevated superoxide levels directly affect the OB/OC ratio, thus affecting bone homeostasis. Impaired fracture healing due to OS can be reversed by means of specialized molecules like superoxide dismutase (SOD), glutathione peroxidase (GPx), vitamin C (ascorbic acid), vitamin E (α-tocopherol) and carotenoids (β-carotene). The mitochondrial protein SIRT3, essential for OC and OB differentiation, proves noteworthy in the inflammatory and ischemic microenvironment during the initial stages of fracture healing, diminishing OS and favoring bone formation. Post-fracture damage to tissue generates a conspicuous amount of free radicals following the ischemia-reperfusion process, which emphasize the importance of SIRT3's OS decreasing properties, and suggest its relevance in mitigating the harmful effects of oxidative damage in the aftermath of a fracture.

Conclusion. Exploring the biochemical intricacies of OS in the context of bone healing offers valuable insights into the mechanisms underlying fracture recovery. Increased levels of plasma biomarkers of oxidant status, like malondialdehyde, marks the need for lifestyle/dietary changes and/or antioxidant supplementation, as to prevent fracture damage directly or indirectly (diseases like osteoporosis, diabetes mellitus, age-related hormonal modifications).