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Wound healing mechanism at bone dislocation site

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Opinion Article

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DESCRIPTION

Bones, cartilage, ligaments and tendons are examples of biological tissues also known as orthopedic tissues that made up the musculoskeletal system. Osteoarthritis a condition that causes joint stiffness and decreased range of motion can be exacerbated by cartilage loss and fracture. Normal bone healing is a difficult process that eventually gives the traumatized area its original form and function.

However, clinical situations including nonunion, abnormalities of a significant size, systemic bone disease, and fusion surgeries have sparked an interest in finding strategies to speed up this natural healing process. A significant portion of this search involves biologics, many of which are now being used in clinical settings. These biologics include bone marrow aspirate concentrate, demineralized bone matrix, platelet-rich plasma, bone morphogenic proteins, and platelet-derived growth factor. Depending on the outcomes of preclinical and clinical research, numerous others such as mesenchymal stem cells, parathyroid hormone and Nel-like molecule-1 (NELL-1), will probably be utilized in the future.

Orthopedic materials in particular possess specific material qualities that enable them to withstand damage and fracture for an extended amount of time. The biological materials can degrade due to sudden harm or constant wear throughout a lifetime of usage. The design of durable synthetic materials that could help with joint replacements can be inspired by the study of bone and cartilage. Analyzing soft material and polymer fracture, in a similar manner, could help us comprehend biological material fracture.

Bones hierarchical structure gives it both strength and hardness or the capacity to withstand inelastic deformation as well as crack initiation, propagation, and fracture. Early research into the characteristics of bone material, particularly its resistance to crack formation, focused on determining a single value for the critical stress-intensity factor Kc, and the critical strain-energy release rate, Gc. While the resistance curve provided insight into crack development, this approach provided crucial insights into bone behavior.

Intrinsic and extrinsic processes are the two categories of mechanisms that might prevent crack spread and increase hardness. Extrinsic mechanisms produce resistance in the fracture's trail behind the crack tip, while intrinsic mechanisms produce resistance in front of the crack. According to some theories, extrinsic mechanisms aid in crack-tip shielding, which lessens the cracks exposure to local stress of a high intensity.

Because intrinsic hardening mechanisms function on a shorter time scale than extrinsic mechanisms (about 1 m), they are less well understood than extrinsic mechanisms. Although bone can deform plastically, "soft" materials like polymers and cartilage are typically linked with plasticity. Fibrils (length scale 10's nm) sliding against one another, stretching, deforming, and/or breaking are an example of an extrinsic mechanism.

Extrinsic mechanisms for reinforcement are better elucidated than intrinsic mechanisms. Extrinsic mechanisms have a length scale along the micron/micrometer scale, whereas intrinsic mechanisms have a length scale in the nanometers. Imaging of extrinsic mechanisms, such as crack bridging (by collagen fibres, or by uncracked "ligaments"), crack deflection, and micro-cracking, has been made possible by Scanning Electron Microscopy (SEM) pictures of bone. The key factors in fracture-shielding are crack deflection and crack bridging by ligaments that are not cracked, while collagen fibres and micro cracking play a small role.