

Approaches for the Electrical Stimulation in Wound Healing Process

Stephen Blister^{*}

Department of Life Sciences, University of Alcala, Madrid, Spain

DESCRIPTION

Cells are frequently subjected to exogenous electrical stimulation in an effort to influence biological processes including death and cell proliferation. The processes of external electrical stimulation during the stages of inflammation, proliferation, and remodelling of wound healing are initially discussed in this section [1].

The inflammatory phase begins with the creation of the wound and includes the coagulation cascade, inflammatory pathways, and immune system activation. Electrical stimulation enhances cellular vascular permeability and vasodilation, allowing more white blood cells, platelets, and other cells to concentrate at the wound site. Macrophages migrate to the anode with very low stimulation, and the rate of directional migration is proportional to electric field strength, resulting in increased phagocytosis of oxalate microspheres, C-type albicans, and apoptotic neutrophils. These findings imply that the electric field activated ERK and P13K pathways, which boosted intracellular Ca2+ influx, including TRPV2-like Ca2+ influx in macrophages, so improving bacterial phagocytosis efficiency [2-5].

Re-epithelialization, fibrogenesis, and angiogenesis occur during the proliferative phase of wound healing. Electrical stimulation increases keratin deposition and the rate of keratinocyte migration, both of which are necessary for re-epithelialization. It also accelerates keratinocyte proliferation and differentiation. When keratinocytes are electrically stimulated, the inflammatory cytokines IL-6 and IL-8 are reduced while the ERK1/2 and p38 MAP kinase pathways are activated. Electrical stimulation alters the migration of Dictyostellium discoideum, the reepithelialization of mouse corneal wounds, and the direction and directed migration of cultured corneal epithelial cells. Electrical stimulation promotes wound healing and decline in inflammatory cytokine expression by suggests a smooth transition from inflammatory to the proliferative phases [6-8].

An *in vivo* study of human wounds treated to monophasic and biphasic electrical stimulation at field strength of 100 mV/mm for 30 minutes each day for 16 days revealed enhanced

granulation tissue ingrowth into the wound centre. On day 16, immunohistochemical examination demonstrated a doubling of epidermal thickness, considerably higher cytokeratin-10 staining, and nearly three times more cytokeratin-10 mRNA expression compared to controls.

Furthermore, electrical stimulation significantly increased the expression of the following proteins in the corresponding *ex vivo* wound model, Proliferating Cell Nuclear Antigen (PCNA), a DNA clamp that acts as a processivity factor for DNA polymerase delta, and Human Double Minute 2 (HDM2), in which HDM2 forms a complex with p53, regulating p53's tumour suppressor functions. These proteins, which are all known to be involved in cell cycle regulation and DNA damage repair, were discovered to respond to the electrical stimuli in distinct ways. Thus, PCNA increased, while HDM2 dropped, and SIVA1 showed no significant change [9,10].

Electrical stimulation has also been shown to increase collagen deposition and speed up migration in Fibroblast Growth Factor (FGF). Electrical stimulation greatly increases FGF-1 and FGF-2 secretion by fibroblasts, readjusting the balance of cell migration, proliferation, and differentiation. Fibrogenesis is necessary for tissue granulation, and fibroblasts must be allowed to move in order to cover the wound. Electrical stimulation aided directional migration of fibroblasts, with localised clustering of integrin and lamellipodia production more likely to occur on the cathode side of the electric field.

Electrical stimulation was also observed to stimulate FGF-1 and FGF-2 production in fibroblasts. Because FGF-1, FGF-2, and Vascular Endothelial Growth Factor (VEGF) are angiogenic factors that must be present for optimal wound healing, these elevated expressions suggest that electrical stimulation has a significant therapeutic potential in wound healing. Electrical stimulation increased the rate of cathodic migration of both types of cells, increased production of the C-X-C Chemokine Receptor Types 4 (CXCR4) and 2 (CXCR2), which are important for the cell migration by causing the mitotic cleavage on both types of the cell perpendicular to the field vector and the random orientation of control.

Correspondence to: Stephen Blister, Department of Life Sciences, University of Alcala, Madrid, Spain, E-mail: blister123@gmail.com

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Even at the end of the wound healing process, electrical stimulation accelerates remodelling by increasing myofibroblast contractility and replacing type-III collagen with type-I collagen via collagen fibre reorganization, enhancing maturity by increasing tensile strength.

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