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Pulsed electromagnetic fields activate mechanosensitive developmental programs in the absence of mechanical input

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Ulsed electromagnetic fields (PEMFs) in the extremely low frequency (ELF) range have been shown to promote the healing of chronic bone fractures (non-unions) in humans and animal models. Despite clinical successes with PEMFs, however, *in vitro* studies often yield contradictory results, limiting their usefulness in tissue engineering applications. We have recently shown that PEMFs instigate mechanosensitive tissue regeneration at the level of the principal transduction process, by vicariously, yet specifically opening TRPC1 cation channels. PEMF induced calcium entry (via TRPC1) then stimulates stem cell proliferation in preparation for tissue regeneration. Given the high incidence of TRPC1 in skeletal muscle my oblasts we examined the effects of PEMFs on skeletal myogenesis in tissue culture. Brief (10 minute) pulses of PEMF are optimal at increasing myoblast number; shorter, or longer; exposures times are less effective at promoting proliferation. The most effective field strengths at promoting myoblast proliferation are within the 100s of mT range, field strength greater than 1.5 mT instead inhibit proliferation. Accordingly, cell cycle progression is sped up after PEMF exposure. As there are now clear indications that changes in the macroscopic mechanical environment as well as mechanical dysfunction on molecular level interfere with the ability of aged stem cells to effectively regenerate mechanosensitive tissues, PEMF-based therapies offer the unique possibility to activate mechanically-regulated developmental programs that would be otherwise muted in the elderly and infirm. The development of PEMF-based technologies will thus have important clinical implication for stimulating muscle regeneration in clinical scenarios characterized by mechanically dysfunction or immobilization.

Biography

Dr. Franco-Obregón received his PhD in the Neurosciences at the University of California at San Francisco, where he studied the contribution of calcium entry via mechanically-gated channels in muscular dystrophy. His main scientific focus continues to be the understanding of the biophysical mechanisms influencing skeletal muscle cell survival and development. He is currently in the Biomedical Engineering Program of the Swiss Federal Institute of Technology, the ETH where he administered the Flow Cytometry Laboratory of the University and ETH Zürich. He is currently on the editorial board of the Journal of Biosensors and Bioelectronics.