into structures. Over a period of 6 days, interstitial flow dramatically synergized with VEGF to drive endothelial cell organization into complex structures. Since fibrin is a temporary matrix that quickly degrades, we incorporated type I collagen into the fibrin in varying ratios and found that this mixed matrix produced extensively branched, lumen-containing endothelial structures. Thus, the synergy seen in fibrin matrices between interstitial fluid flow and matrix-bound VEGF can be broadened in the presence of collagen and over longer time periods. This work suggests that interstitial fluid flow may be necessary for matrix-bound growth factor utilization to be optimized for morphogenetic processes in vivo, and provides a method for tissue engineering capillary structures in vitro.

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P11

MICROMECHANICAL FORCES AS A POTENT STIMULATOR OF WOUND HEALING

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In vitro studies have shown that cells divide and proliferate in response mechanical stimuli. Using this knowledge, we hypothesized that micromechanical forces (MMFs), both cyclical and static, are capable of enhancing wound healing through stimulation of both angiogenesis and cellular proliferation. Method. The ears of Wistar rats ($n = 15$) were stretched using elastic connectors to apply 55 g of continuous tension for 4 days. Cyclical tension was applied for 2 h with 1-h rest intervals. Vessel size and morphology were monitored using intravital microscopy. After stimulation, ears were harvested for histology and VEGF immunohistochemistry. Both cyclical and static tension produced significant increases in vessel size. Rat ears subjected to static tension underwent a 110% increase in mean vessel diameters over their tension-free counterparts ($p < .001$). When cycled periodically, significant changes were measurable after only a few hours (25% vs. 3%, $p < .001$), rather than 1–2 days. Stretched vessels demonstrated greater VEGF expression. Preliminary stretched vessel casts also appeared to be larger. MMFs are capable of inducing significant vascular changes when applied cyclically or statically, with cyclical tension being more potent. MMFs in the rat ear model result in increases in vessel diameters and likely upregulate VEGF and increase tissue perfusion. While the mechanisms have yet to be elucidated, these results suggest the crucial role of MMFs in regulating cellular growth and differentiation, as well as their potential as a novel approach for enhancing wound healing.

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P12

METABOLIC VASODILATION IS SENSITIVE TO FLOW IN MOUSE CREMASTER ARTERIOLES

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NO and KATP channels contribute to a metabolically coupled vasodilation in mouse cremaster arterioles. We sought to determine if the NO and KATP channel components of the dilation are sensitive to flow. In the cremaster muscle of anesthetized mice (74 mg/kg IP), electrical field stimulation (15 s, 30 Hz, 0.2 ms, 5–10 V) was used to produce a mean metabolic vasodilation 17.7 ± 1.4 μm from a baseline of 7.1 ± 2.4 μm in control mice (max diameter 28.7 ± 2.3 μm, $n = 6$). Selective block of NOS with 100 μM nitro-L-arginine (LNA) attenuated the immediate poststimulation dilation; 14.7 ± 3.0 μm (max diameter 29.7 ± 2.3 μm, $n = 4$). LNA also caused a delay in the peak change in diameter by approximately 30 s. Selective block of KATP channels with 10 μM glibenclamide did not attenuate the peak dilation; 20.6 ± 3.4 μm (max diameter 33.9 ± 1.1 μm, $n = 3$). Muscle contraction in normoxic ischemic arterioles produced a metabolic dilation of 8.7 ± 2.5 μm in control vessels (from a baseline of 10.6 ± 2.8 μm, $n = 3$), which was significantly less than the metabolic dilation in free flow ($p < .001$). With 100 μM LNA, the immediate poststimulation dilation was significantly less (4.0 ± 1.8 μm from a baseline of 2.9 ± 0.5 μm, $p < .001$, $n = 3$), and the peak response was again delayed. With 10 μM glibenclamide, the dilation was significantly attenuated from free flow dilation (4.9 ± 1.8 μm, $n = 3$ from a baseline of 4.2 ± 1.3 μm, $p < .001$, $n = 3$). We conclude that metabolic vasodilation is sensitive to flow; NO contributes to the early phase of the dilation and the role of KATP channels is greater in ischemia. Supported by NIH HL 76414.

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P13

IS DERMAL BLOOD FLOW IN A NONWOUNDED SKIN MODEL ALTERED BY TOPICAL NEGATIVE PRESSURE?

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Topical negative pressure (TNP) is a method of managing both acute and chronic wounds. It involves the application of a controlled subatmospheric pressure across a wound using an open cell foam dressing. Experimental evidence (in a porcine wound model) has suggested that skin blood flow is modified when subatmospheric pressure is applied across a TNP dressing. However, its effect on nonwounded skin in humans has not previously been investigated. We investigated whether in nonwounded skin, the application of a subatmospheric pressure across a standard TNP dressing results in changes in dermal blood flow. Ten volunteers ($n = 10$) took part in the study.