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The Stretching of Human Keratinocytes May Play a Role in Psoriasis

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Introduction

Psoriasis is an autoimmune disease with cutaneous and systemic manifestations and individuals with psoriasis demonstrate accelerated wound healing in both the involved and uninvolved skin. [1] Psoriasis has a polygenic basis. It is conceivable that one of the genes involved in the pathophysiology of this disease is a gene related to the stretching of cells, *e.g.*, the keratinocyte.

In an attempt to validate the hypothesis that the stretching of keratinocytes plays a role in the pathophysiology of psoriasis and, by extension, wound healing, we examined published data from an experiment in which normal keratinocyte cultures were mechanically stimulated. [2] These data showed that at 30 minutes' nitric oxide synthase-1 was stimulated 35%. The results were not significant ($p = 0.09$, 2-tailed student t-test with differing variances) and the effect was transient, falling to 11% after 6 h. However, the experiment was not designed to test this hypothesis and the significance was limited by using only 2 degrees of freedom. Further, the experiment used keratinocytes that were not derived from a psoriatic individual. Although the stimulation was not statistically significant, the nitric oxide synthase-1 did appear to show sensitivity to the mechanical stimulus. Neither tumor necrosis factor alpha, nor any of the IL-17 genes were stimulated by stretch in these published data.

It has been reported that *in vivo* the concentration of nitric oxide is increased 10-fold over psoriatic plaques compared to uninvolved skin in the same individual. [3] Moreover, nitric oxide stimulates keratinocyte proliferation. [4] Thus, these data may be interpreted to suggest a possible mechanism that might contribute to localization of psoriasis to the extensor surfaces.

The keratinocyte, like the neuron, another cell of ectodermal origin, expresses the n-Methyl-d-Aspartate receptor. [5] In neurons, nitric oxide regulates the intercellular concentration of Ca++ via release of this cation from intracellular stores, i.e., mitochondria. [6] In turn, Ca++ can modulate the kinetics of the n-Methyl-d-Aspartate receptor and thus alter n-Methyl-d-Aspartate

receptor-evoked signals in the neuron. In keratinocytes, this receptor also regulates Ca++ entry and this cation regulates cellular growth and differentiation. Further, the NR2C subunit of the n-Methyl-d-Aspartate receptor is downregulated in both the involved and uninvolved psoriatic skin. [1] Therefore, we hypothesize that dysregulation of Ca++ entry via the n-Methyl-d-Aspartate receptor may contribute to the Increased keratinocyte proliferation seen in psoriasis.

The accelerated wound healing documented in psoriasis, may confer a survival advantage, thereby accounting for the prevalence of this disease in the world-wide population. To date, the physiology of wound healing is poorly understood. In most mammals, wound healing depends on contracture promoted by the panniculus carnosus. By contrast, increased proliferation and migration/movement of keratinocytes appears to play a central role in wound healing in humans. The differences in the wound healing response in humans compared to other animals may explain the absence of psoriasis in animals other than homo sapiens. Moreover, the pathophysiology of psoriasis may relate to wound healing. The extensor surfaces of the human body are prone to injury presumably leading to a wound healing-type response, and keratinocytes that are stretched as they migrate may show an increase in nitric oxide synthase-1 synthesis. Taken together, these data may explain the prevalence of psoriasis on the extensor surfaces of the extremities. This hypothesis could be tested by comparing the synthesis of various proteins in stretched versus non stretched cultured keratinocytes obtained from the skin of individual(s) with psoriasis, as well as, increasing the number of time points when the synthesis of these protein(s) is assayed after stretching the cells.

Conflict of Interest: the authors have no conflicts of interest

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