

# A Different Perspective on Healing and Restoration of the Skin is offered by Dermal Telocytes

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## Introduction

Given that the skin is the largest human organ, completely covering the human body, and that it serves at least a few important functions (mechanical protection, physical barrier, etc.), it is natural to think of it as vulnerable to various injuries. These injuries disrupt the barrier function and can be acute or chronic, appearing suddenly at times but also in the context of some dermatological pathologies. Another function of the skin (often greatly enhanced) is its social/aesthetic function, because healthy and presentable skin boosts many people's self-esteem, contributing to a better social life. Currently, millions of people are suffering from either acute or chronic wounds that must be treated therapeutically. Iatrogenic wounds (e.g., surgical injuries) are examples of acute wounds or unintentional skin injuries of varying severity. Aside from these, chronic skin conditions can be physiological (e.g., face skin wrinkles of varying severity or atrophic skin) or wound-related (e.g., associated with diabetes, venous insufficiency, obesity, etc.) In this context, the estimated wound care products market grows every year, with various sources indicating a market value of billions of Euros, and is further inflated during times of widespread accidents, wars, and so on [1]. Finding better ways to treat these wounds would thus benefit patient care systems. Developing new cell-based therapeutic protocols for skin wound repair could significantly reduce the financial and economic burden of skin wound care issues while also increasing social impact. Further skin-repairing protocols should follow suit. With new technological developments and/or breakthroughs in cellular and molecular medicine (especially concerning tissue engineering and regenerative medicine). This should be the policy of the future in skin wound healing and antiaging dermatology. However, more research into the cellular and molecular mechanisms underlying skin wound recovery and repair/regeneration following trauma is required. Wound care science could look into this series of interconnected biological events that occur serially as the wound progresses, all of which would require a better understanding of the wound microenvironment and the progression chronology of the cellular and molecular response sequences after injury. Developing new protocols in response to new technological advancements and/or developments in cellular and molecular medicine (particularly in tissue engineering and regenerative medicine) should become the future policy in skin wound healing management and antiaging dermatology [2].

## Description

The quantitative and qualitative presence of TCs is influenced by various skin conditions, both normal and pathophysiological. Several diseases

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recognised cellularity changes, and these studies revealed various (ultra) structural changes of TCs and their spatiality with different interstitium components. The progressive accumulation of collagen type II and III packages within the dermis (either papillary or reticular), mucoid edema, and panniculitis in systemic sclerosis leads to disruptions of typical TCs arrangements and normal tissue architecture. The density of TCs is decreasing (in the initial stages, more prominent in the papillary dermis, and after that, in the reticular dermis). Their changes occur in tandem with the alteration of the extracellular matrix, further disrupting the TCs interstitial network. The deterioration of TC integrity and interstitial as the fibrotic process progresses, so does localization. TCs have hypoxic changes in their ultrastructure, with vacuoles within their cytoplasm containing swollen mitochondria and lipofuscin bodies.

However, there is still some debate about whether they are the cause or the result of these changes. The cellular pathological perspectives of these new TCs, as well as their integration into the pathophysiological microenvironment, can dramatically alter structural considerations about these cells. Dermal sclerosis TCs, on the other hand, have a reduced global intradermic distribution and are more prominent around skin adnexa, blood vessels, or nerve endings. It will be interesting to see if the ultrastructural changes in TCs are caused by the ischemia and if TCs are more affected than other interstitial cells. In contrast, another theory holds that Those initial ultrastructural changes in TCs may cause dermal deposition of extra collagen fibers. Psoriasis, on the other hand, is characterised by changes in TCs ultrastructure (keratinization secondary to dermal inflammation, with immunologic determinants and genetic background, with consequences over epidermal turnover) [3].

Disruption of cellular integrity (involving the cellular bodies, fragmentation of the cytoplasm and cell membrane, nuclear exclusion) and loss of Tps integrity with fragmentation of their continuum at different levels are among the dystrophic changes. Surprisingly, in microscopical studies, dendritic cells were frequently found in contact with the extruded nuclei of disintegrated TCs. This aspect suggests that the destruction of TCs causes a chain of immune reactions. These ultrastructural changes in TCs occur at the same time as the density of dermal dendritic cells increases and Langerhans cells migrate from the epidermis to the dermis. Furthermore, psoriasis is characterised by several vascular changes, including TCs becoming less dense in their proximity and smooth muscle cells losing their contractile phenotype. According to published data, increased density of TCs (positive for PDGFR) aids in the evolution of chronic skin wounds, reconfirming previous findings regarding myocardial regeneration/repairing processes. Skin carcinomas, whether basal cell carcinoma or squamous cell carcinoma, are composed of affected TCs that are less involved in heterocellular junctions than TCs in normal tissue.

TCs have inner plaques of dense electron microscopic material at these junctions. Furthermore, their plasma membranes may be fused (a plasma membrane particularity frequently found in tumour cells). PDGF is a dimeric glycoprotein with multiple sources. Following the interaction of PDGF and its receptor, c-fos and c-myc are activated (two protooncogenes). PDGF is primarily synthesised and stored in thrombocyte alpha granules, and it can be released by activating platelets. Other cells, such as endothelial cells, macrophages, and smooth muscle cells, could also produce PDGF. Aside from mitogenic activity, it may be chemotactic for mesenchymal cells and vasoconstrictors, increase the number of LDL receptors, increase prostaglandin secretion, induce changes in cellular shape, and play an important role(s) in (neo) angiogenesis [4].

Previous research has shown that in experimental acute myocardial

infarction, the border zone of the lesion (metabolically and immunologically, the most important) At each time point, the active area is dominated by different types of cells: inflammatory cells, myofibroblasts, fibroblasts, and TCs. However, 30 days after a myocardial infarction, TCs have mastered the local cellular scenery, primarily through close spatial interactions with blood vessels (including the new-formed blood vessels). Thus, more research is needed to determine the role of skin TCs in reparatory/regeneration processes following an acute skin injury or a chronic skin defect, particularly after administration of growth factors at the lesion site (like PRP). Such growth factors may aid tissue regeneration by increasing the presence, distribution, density, and activity of skin TCs. Furthermore, such findings could be extrapolated and investigated further [5].

## Conclusion

A large body of evidence suggests that at least a few documented skin pathologies are characterised, among other things, by a disruption in the regular tissue distribution of dermal TCs. Furthermore, the clinical and/or structural recovery of these dermatological conditions is characterised by (or even determined by) the rehabilitation of dermal TCs in their previous distribution. Thus, one question is whether TCs are involved in the repairing process or if their recovery is simply a result of the tissue repair complex cellular orchestration. However, given the presence of TCs in skin stem cell niches, as well as previously published data showing TCs' involvement in myocardial regeneration (and their potential in nursing cardiac progenitor cells)

and liver regeneration, it is reasonable to conclude that TCs play a role in these processes. It is tempting to speculate that they may play similar roles in skin regeneration/repair processes. There have been no studies on the dynamic cellular and molecular changes in TCs after such treatment. The skin is one of the most accessible organs that could provide proof-of-concept for the involvement of TCs in diseased or injured tissue, potentially leading to research insights into other diseases and potential new therapeutic approaches with multiple medical, social, economic, and social benefits.

## References

1. Dai, Christina, Shawn Shih and Amor Khachemoune. "Skin substitutes for acute and chronic wound healing: An updated review." *J Dermatol Treat* 31 (2020): 639-648.
2. Tottoli, Erika Maria, Rossella Dorati, Ida Genta and Bice Conti et al. "Skin wound healing process and new emerging technologies for skin wound care and regeneration." *Pharmaceutics* 12 (2020): 735.
3. Yeganeh, Pegah Moharrami, Safa Tahmasebi and Abdolreza Esmaeilzadeh. "Cellular and biological factors involved in healing wounds and burns and treatment options in tissue engineering." *Regen Med* 0 (2022).
4. Popescu, L. M., M. E. Hinescu, N. Ionescu and Carmen Ardeleanu et al. "Interstitial cells of Cajal in pancreas." *J Cell Mol Med* 9 (2005): 169-190.
5. Wang, Fei, Yang Song, Yihua Bei and Changqing Yang et al. "Telocytes in liver regeneration: Possible roles." *J Cell Mol Med* 18 (2014): 1720-1726.

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