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# Brief Report on Fetal Wounds and Extracellular Matrix Proteins

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## Description

The morphologic structure of skin comprises two layers, the epidermis and dermis. The epidermis is the most external layer of the skin and is divided into four or five sub-layers, depending on the region of the body. Among the cells that constitute the epidermis are by far the major cell type, the keratinocytes, as well as melanocytes, langerhans cells, and merkel cells.

## Extracellular Matrix Proteins

The dermis is the layer of connective tissue that supports the epidermis, constituted by extracellular matrix proteins (collagens, elastin, proteoglycans, and glycosaminoglycan) synthesized by fibroblasts. If there is a disruption of one or both layers of the skin, the organism starts the wound healing process to regenerate the injured area, involving cellular, molecular and biochemical mechanisms divided in three healing phases.

Prerequisite for these approaches is a profound understanding and acknowledgement of the underlying pathophysiological processes that lead to disturbed wound repair. Scar formation demarcates the end of the last wound healing phase, the remodeling phase. When it is necessary a greater mechanical stability, dermal substitutes are more suitable than epidermal. Dermagraft and transcyte are cellular commercial dermal substitutes composed by cultured allogeneic neonatal skin fibroblasts seeded in type I collagen/silicone film.

These extracellular matrix analogues and their fibroblasts secrete growth factors and proteins that enhance re-epithelialization by the patient's keratinocytes in partial- and full-thickness wounds. In some injuries, the treatment with epidermal (keratinocyte) or dermal (fibroblast) substitutes alone can result in improper healing. In contrast to fetal wound repair, normal adult wound healing ultimately results in wound closure and replacement of the original tissue with a collagenous scar.

The miracle of perfect, scarless embryonic wound repair is currently poorly understood. The early-gestation fetus can heal skin wounds with regenerative-type repair and without scar formation. Fibroblasts also synthesize compounds of provisional extracellular matrix, including type III collagen, proteoglycans and fibronectin, in order to support cell migration into the area. The restructuring of vascularization at the wound begins immediately after the injury, but has higher activity in the proliferative phase, providing oxygen and nutrients needed for the migration and proliferation of cells and synthesis of extracellular matrix compounds.

## Fetal Wounds

In scar less fetal wounds, the epidermis and dermis are restored to a normal architecture. The collagen dermal matrix pattern is reticular and unchanged from unwounded dermis. The wound hair follicle and sweat gland patterns are normal as well. Previous studies on fetal wound repair in sheep showed that wounds healed with complete skin restoration until the end of the second trimenon. In humans, however, scarring occurs earlier in fetal wound repair. This might be due to the specific response of fetal fibroblasts to the pro-fibrotic mediator TGF-β. Scar formation is the ultimate outcome of wound repair in children and adults.

Cutaneous scars have no epidermal appendages (hair follicles and sebaceous glands) and a collagen pattern that is distinctly different from unwounded skin. New collagen fibers secreted by fibroblasts are present as early as 3 days after wounding. As the collagen matrix forms, densely packed fibers fill the wound site. The ultimate pattern of collagen in scar is one of densely packed fibers and not the reticular pattern found in unwounded dermis. However, there are some disadvantages such as long-time preparation, hyperkeratosis and scar possibility.

Secreted mediators as VEGF and angiopoietins stimulate the proliferation of endothelial cells and restructuring of the vascular system at the wound site. However, a distinct pattern of the neovascularization process can be described forming a circle with an inner ring of circularly organized vessels directly at the wound border followed by radially shaped vessels supplying the inner ones and connecting to the normal, uninjured skin. Disruption in the neovascularization process consecutively leads to wound healing disturbances or chronic ulcers, typically seen in venous insufficiency, arteriosclerotic disease or diabetic foot sores. This pathophysiological phenomenon deserves further attention. Recent research projects focus on blood vessel neoformation and/or delivery to the injury site in order to restore the perfusion and support the healing process.

During the proliferative phase, re-epithelialization occurs to close the epithelial gap and restores the barrier function of the skin. Firstly, the keratinocytes at the border of wounds are stimulated by growth factors, resulting in the proliferation and differentiation of the keratinocytes. The (micro) vasculature

contributes to the initial hemostasis, reduces blood loss and establishes a provisional wound matrix. Blood clot-derived cytokines and growth factors drive the recruitment of pivotal cells that are crucial for the healing process. This provisional wound microenvironment depicts the starting point for new vessel formation and regeneration thereby ensuring the nutritive perfusion of the wound and the delivery of immune cells that remove the cell debris. At first sight, the neovascularization process seems very disordered as the healing wound generates a high density of functional as well as dysfunctional new capillaries. Nonfunctional vessels will regress by time via maturation or apoptotic processes.