Synopsis of Original Research Paper

Study the extracellular matrix, especially versican involvement of wound healing by use of RNAi

Hajime Tomita

Department of Dermatology, and Allergology, Nagasaki University Hospital

Traumatic injury leaves disfiguring scar and pigmentation, which provide significant reduction of QOL. Adequate skin wound healing leads to beautiful scar. Therefore, there is a need for a better treatment approach. Skin wound healing is a complex biological event as a result of the interplay of epidermis and dermis. Formed good dermis wound bed performs an essential function for epidermal cell migration and re-epithelization. Although, extracellular matrix existing dermis has relations with cell adhesion, cell migration, cell differentiation, and cell proliferation, little is known about a role of extracellular matrix in wound healing. To clarify a role of extracellular matrix in wound healing, we investigated mouse fibroblast cell using suppression of versican, which is a hyaluronan-binding, extracellular chondroitin sulfate proteoglycan.

The levels of versican expression were measured over time after mouse cutaneous wounding. On day 4 and 5, mRNA level of versican were significantly increased in day 4 and 5 relative to day 1 to 3. By contrast, mRNA level of versican were significantly decreased in day 6 and 7. Therefore, versican may regulate the wound healing process.

The mRNA expression of Srpx2, Ccl6, and Smoc2 was increased in versican suppressed mouse embryonic fibroblast. Previous reports showed that these factors regulated the inflammatory cell infiltration and angiogenesis. These results suggest that versican may regulate the wound healing through Srpx2, Ccl6 and Smoc2.

Blockade of versican changed the various factors in mouse embryonic fibroblast. These effects of versican blockade may occur in this wound study on day 1 to 3, 6, and 7. Taken together, it is possible that versican regulate the wound healing by the Srpx2, Ccl6 and Smoc2 at some stated periods.