# Abstracts of Outstanding Presentations of the 79th Annual Meeting of the Medical Association of Nippon Medical School

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## **Abstracts of Outstanding Presentation (1)**

# Characterization of Wnt Signaling Pathway in Keloid Pathogenesis

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#### Objective

Keloid is a fibroproliferative lesion which develops after wound healing. On the other hand, the wingless (Wnt) signaling pathway plays a key role in various cellular functions including proliferation, differentiation, survival, apoptosis and migration. The aim of this study was to characterize the Wnt signaling pathway in keloid pathogenesis.

#### **Materials and Methods**

Primary fibroblast cultures and tissue samples from keloid and normal appearing dermis were used. The expression of Wnt family members (1, 2, 3, 4, 5a, 6, 7a), frizzled4 receptor, receptor tyrosine kinase-like orphan receptor(ROR)2 and the Wnt signaling downstream target, beta-catenin, were assessed using semi-quantitative RT-PCR, Western blot or immunohistochemical methods.

#### Results

Of the Wnt family members, Wnt5a mRNA was highly expressed (Fig. 1) and Wnt2 mRNA was sporadically expressed in keloid fibroblasts compared to normal fibroblasts. A higher immunohistochemical expression of Wnt5a and its receptor frizzled4 (Fig. 2) as well as the downstream target, beta-catenin was found in keloid fibroblasts. Western blot analysis confirmed these results. Normal dermal fibroblasts showed weak or no reaction.

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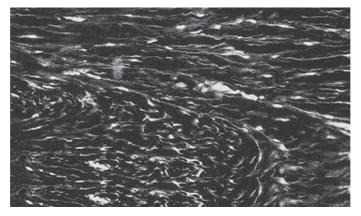


Fig. 1 Immunoflour escence staining for Wnt5a shows strong expression in keloid fibro blasts. Original magnification  $\times 400.$ 

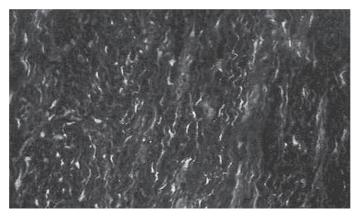


Fig. 2 Immunoflourescence staining for Frizzled4 receptor shows moderate expression in keloid fibroblasts. Original magnification ×400.

## Conclusion

Our results show higher expression of Wnt5a and its receptor frizzled4 in keloid fibroblasts and tissues than in their normal counterparts. These findings highlight a potential role for Wnt5a in the pathogenesis of keloid and warrant further functional analyses to establish it.