〔Japan. J. Pharmacol., **60**, 91-96 (1992)〕 [Lab. of Pharmacology]

The Mechanism Involved in the Inhibitory Action of Tranilast on Collagen Biosynthesis of Keloid Fibroblasts.

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Tranilast is reported to inhibit collagen synthesis of fibroblasts derived from keloid tissues. In the present study, we investigated its inhibitory mechanism on collagen synthesis of fibroblasts from keloid and hypertrophic scar tissues of humans. Collagen synthesis of fibroblasts from keloid and hypertrophic scar tissues is greater than that from healthy skin. Tranilast did not inhibit prolyl hydroxylase activity. Tranilast suppressed the collagen synthesis of the fibroblasts from keloid and hypertrophic scar tissues but not the healthy skin fibroblasts. The agent inhibited the release of transforming growth factor-β1 (TGF-β1) from keloid fibroblasts. Therefore, tranilast is suggested to inhibit collagen synthesis through suppressing the release of TGF-β1.

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Effects of Suplatast Tosilate (IPD-1151T) on Antibody Formation in Mice.

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The effects of suplatast tosilate (IPD-1151T) on antibody formation in mice were investigated. IPD-1151T clearly increased the production of IgM and IgG hemolytic plaque forming cells (PFC) in mice immunized with sheep red blood cells (SRBC). Impaired PFC production in old mice was recovered by the administration of IPD-1151T. The agent clearly suppressed the anti-dinitrophenyl (DNP)-IgE antibody production without affecting anti-DNP-IgM and IgG antibody productions. IPD-1151T did not affect the induction phase of the cellular immune reaction including picryl chloride-induced contact dermatitis and SRBC-induced footpad reaction. These results suggest that IPD-1151T has a class-specific suppressive effect on the production of IgE antibody, but does not suppress the other immune responses.

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Effect of Suplatast Tosilate (IPD-1151T) on Types I-IV Allergic Reactions.

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The effects of suplatast tosilate (IPD-1151T) on Types I-IV allergic reactions were investigated. IPD-1151T dose-dependently inhibited homologous passive cutaneous anaphylaxis (PCA) in rats. The inhibition was observed when given orally 0.5-2 hr prior to the antigenic challenge. IPD-1151T also suppressed the antigen-induced degranulation of mesenteric mast cells and histamine release from peritoneal exudate cells of rats. High dose of IPD-1151T inhibited reversed cutaneous anaphylaxis in rats, whereas the agent did not affect Forssman shock in guinea pigs. IPD-1151T neither affected the Arthus reaction in rabbits nor the picryl chloride-induced contact dermatitis and sheep erythrocytes-induced footpad reaction in mice. These results indicate that IPD-1151T shows a relatively specific suppression of the type I allergic reaction.