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[Lab. of Pharmacology]

Active Cutaneous Anaphylaxis (ACA) in the Mouse Ear.

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Active cutaneous anaphylaxis (ACA) was studied in the ear of female BALB/c mice. Mice were immunized with ovalbumin in the presence of aluminium hydroxide gel (alum) or complete Freund's adjuvant (CFA). Two weeks after the immunization, ACA was elicited in the mouse ear by injecting 10 μ l of antigen solution into the ear lobe. ACA was assessed by the amount of extravasated dye, which was given intravenously just after the antigen injection. Antiallergic drugs, antihistamines, beta-stimulants, theophylline and glucocorticoids inhibited the reaction significantly. These drugs inhibited ACA in mice immunized with alum-precipitated antigen and ACA in mice injected with CFA-emulsified antigen similarly. ACA in the mouse ear might be a useful tool for studying drugs for allergy.

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Antitumor Activity of Two Novel Low Immunosuppressive Fluoropyrimidines UK-21 and UK-25.

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Antitumor activities of 2',3',5'-tris-O-[N-(2-n-propyl-n-pentanoyl)glycyl]-5-fluorouridine (UK-21) and 1-{6-[N-(2-n-propyl-n-pentanoyl)glycyl]amino-n-hexylcarbamoyl}-5-fluorouracil (UK-25) were studied. UK-21 given orally showed an antitumor effect against Sarcoma-180, but failed to affect P388, L1210 and Lewis lung carcinoma. In contrast, orally administered UK-25 showed the antitumor effect on all of the tumors employed. These results suggest that UK-21 has the potential for development as a parenterally applicable anticancer drug, and UK-25 has the potential as an oral one.

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Inhibitory Action of Tranilast, an Anti-Allergic Drug, on the Release of Cytokines and PGE₂ from Human Monocytes-Macrophages.

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Tranilast, an anti-allergic drug which inhibits chemical mediator release from mast cells, has been reported to improve keloids and hypertrophic scars. In the present study, we found that tranilast also inhibited the release of transforming growth factor (TGF)- β 1, interleukin (IL)-1 β and prostaglandin (PG) E₂ from human monocytes-macrophages. It was suggested, therefore, that tranilast suppresses collagen synthesis by fibroblasts through inhibiting TGF- β 1 and PGE₂ production and proliferation of fibroblasts through inhibiting IL-1 production by inflammatory cells such as macrophages.