〔Jpn. J. Allergol., **38**, 493 (1989)〕

Anti-allergic action of glucocorticoids. (II)Effect of glucocorticoids on cell mediated (Type IV) allergic reactions.

HIROICHI NAGAI*, TAMOTSU TAKIZAWA , NAOKI INAGAKI, TATSUO SAKAMOTO, TSUKASA SHIMAZAWA and AKIHIDE KODA.

The effects of three glucocorticoids (steroids; hydrocortisone, prednisolone and dexamethasone) on cell mediated hypersensitivity (type IV) reactions in rats and mice were studied. All the steroids inhibited both the induction and the effector phase of type IV reacrion induced by sheep red blood cells (SBBC) in mouse footpads. The local graft vs host reaction induced by lymphocytes from Brown Norway rats into the footpads of (Lewis×Brown Norway) F_1 rats was also clearly inhibited by steroids. Moreover, the anti type IV reaction mechanisms of glucocorticoids were analyzed *in vivo* and *in vitro*.

〔J. Pharmacobio-Dyn., **12**, 517 (1989)〕

Pharmacological study of Phospholipase A_2-induced histamine release from rat peritoneal mast cells.

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The effect of some drugs on phospholipase A_2 (PLA_2)-induced histamine release from rat peritoneal mast cells was investigated. PLA_2 (*Naja naja*) caused the release of histamine from rat mast cells in a dose related fashion. The release of histamine by PLA_2 was decreased by the removal of each of calcium and glucose from the reaction medium. PLA_2 inhibitors, calcium channel blockers and anti-allergic agents inhibited the release of histamine by PLA_2. When glucocorticoids were administered prior to the harvest of the mast cells, the amount of histamine released from the mast cells by PLA_2 *ex vivo* was decreased.

〔Inflammation, **13**, 401 (1989)〕

Pathological studies on nephrotoxic serum nephritis accelerated with rabbit γ-globulin in mice.

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In order to characterize nephrotoxic serum nephritis accelerated with rabbit γ-globulin in mice, histopathological studies were carried out 15 days after NTS injection, the time when increases in urinary protein and serum cholesterol and a decrease in serum albumin were apparent. As a result, it appears this nephritic model shares a common pathology with human membranoproliferative glomerulonephrilis type I and crescentic glomerulonephritis and can be considered an appropriate model for producing severe nephritis for short periods.