[J. Cell Biol., **131**, 1387-1401 (1995)] [Lab. of Molecular Biology]

Anterograde and Retrograde Traffic between the Rough Endoplasmic Reticulum and the Golgi Complex

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The transfer of newly synthesised membrane proteins moving from the RER to the Golgi complex has been studied with chimaeric proteins, which consist of a reporter enzyme HRP, anchored to the transmembrane domains of two integral membrane proteins, the transferrin receptor and sialyltransferase. The chimaeras are distributed throughout the nuclear envelope, RER, VTCs and a network of tubules in the cis-Golgi area. The temperature shift experiments demonstrate that these chimaeras are transported from the RER to the cis-Golgi in free vesicles (40-60-nm) at 37 C°, and that the retrograde traffic back to the RER is probably mediated by vesicles with a similar morphology but which, in cells expressing membrane-anchored chimaeras, lack detectable reaction product.

[Planta Med., **61**, 45-49 (1995)] [Lab. of Microbiol.]

Principle of the bark of *Phellodendron amurense* to suppress the cellular immune response: effect of phellodendrine on cellular and humoral immune responses.

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Phellodendrine from Phellodendri Cortex suppressed local semisyngeneic graft-versus-host (GvH) reactions and systemic allogeneic GvH reactions in X-ray irradiated recipient mice, and it also suppressed the induction phase of SRBC-induced delayed type hypersensitivity in mice and tuberculin-induced delayed type hypersensitivity in guinea pigs, but not the effector phase of these reactions. Surprisingly, phellodendrine did not affect antibody production in mice to SRBC, and was expected to be a valuable new type of immunosuppressor against the cellular immune response.

[Jpn. J. Pharmacol., **67**, 279-289 (1995)] [Lab. of Microbiol.]

Suppressive effects of tranilast on pulmonary fibrosis and activation of alveolar macrophages in mice treated with bleomycin: role of alveolar macrophages in the fibrosis.

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After bleomycin (BLM) instilled in mice, alveolar macrophage (AM) often migrated into alveolar spaces surrounding the fibrotic areas and were activated in the inflammatory phase. The activation of AM maybe important for development of fibrosis. Tranilast suppressed an increase of AM activity to produce reactive oxygen species in BLM-instilled mice, and it inhibited the subsequent development of pulmonary fibrosis. In vitro tranilast-treatment suppressed the reactive oxygen species production from murine peritoneal macrophages. The results suggest that tranilast suppresses fibrosis by inhibiting AM activation but not by scavenging reactive oxygen species.