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Assessment of allergenicity of genetically modified foods (GMOs)

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The potential allergenicity of the transgene products in genetically modified organisms (GMOs), has been an important issue. As a part of the risk assessment of GMOs, we investigated the physicochemical stability and the immunogenicity of food allergens to determine their allergenicity. We have systematically evaluated the stability of food allergens in the gastrointestinal tract by using simple models of gastric (Stimulated gastric fluid) and intestinal (Stimulated intestinal fluid) digestion. Food allergens were divided into three groups in terms of their stability. Some (b-lactoglobulin, lectin) were highly stable to digestion for 30 min and others (peanut lectin) were moderately stable for 5 min. In contrast, casein, albumin, and ovomucoid were rapidly digested within 15 sec. We also determined the allergenicity of food allergens in Brown Norway rats and Balb/c mice. The results demonstrated that Brown Norway rats could be sensitized orally to ovalbumin allergens and Balb/c mice could be immunized intraperitoneally by ovalbumin, resulting in significant increase of IgG and IgE, respectively. Our results showed that food allergens might have diverse stability in gastrointestinal tract, although they were known to be more stable than nonallergenic proteins, and that BN rat and Balb/c mouse models could be useful as animal models for the determination of allergenicity of transgene products.

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MPP⁺-induced cytotoxicity is attenuated by induction of heme oxygenase

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MPP⁺ is known to be a neurotoxic substance that induces the degeneration of dopaminergic neurons and a Parkinsonism-like syndrome. MPP⁺ is retained intracellularly or accumulated in dopaminergic neurons via the dopamine-reuptake system. It inhibits mitochondrial electron transport in dopaminergic neurons. In addition, it generates hydroxyl radicals, which cause the peroxidation of membrane lipid or damage DNA. Heme oxygenase-1 (HO-1) can be induced by oxidative stress and protects cells against oxidative stress-induced cytotoxicity. To examine whether HO-1 is induced by MPP⁺ and has protective effect on MPP⁺-induced injury of dopaminergic cells, PC-12 cells were cultured and cell viability was measured with MTT assay and cell staining. MPP⁺ elicited a relatively rapid increase in HO-1, and the inhibition of HO increased MPP⁺-induced cell death and production of reactive oxygen radical. These results suggest that HO-1 induced by MPP⁺ may reveal the protective effect against MPP⁺-induced cytotoxicity.

[PA1-61] [10/18/2002 (Fri) 09:30 – 12:30 / Hall C]

Roles and signaling pathway of M2 pyruvate kinase in RBL-2H3 cells

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Aggregation of the high affinity IgE receptor on mast cells results in many biochemical, events leading to the release of histamine, serotonin, prostaglandins arachidonic acid metabolites, and cytokines. Previously we have shown that M2 pyruvate kinase interacts with the gamma chain of IgE receptor on the ITAM (immunoreceptor tyrosine-based activation motif) region. We also have shown that the enzymatic activity of pyruvate kinase is inhibited upon cross-linking of IgE receptors through the phosphorylation on the tyrosine residues. In this study, we permanently transfected RBL-2H3 cells with M1 pyruvate kinase to test whether the IgE receptor-mediated inhibition of M2 pyruvate kinase is essential for the degranulation of mast cells. When cells were transfected with M1 pyruvate kinase, that is, the IgE receptor-mediated inhibition of pyruvate kinase (M2) was overshadowed, the degranulation of mast cells were significantly inhibited, suggesting that IgE receptor-mediated inhibition of M2 pyruvate kinase is important for the degranulation of mast cells. Src inhibitor (PP2), but not Syk inhibitor (piceatannol), abolished the IgE receptor-mediated tyrosine phosphorylation of M2 pyruvate kinase and enzyme activity changes, showing that M2 pyruvate kinase is under the control of Src.

[PA1-62] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Heterotrimeric G protein $\gamma 12$ Subunit is Region-Specifically Expressed in Rat Brain

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The G protein $\gamma 12$ subunit (G $\gamma 12$) is widely-expressed and, given the extensive role of the $\beta\gamma$ subunit (G $\beta\gamma$) in cell signaling, is a uniquely known substrate for protein kinase C, indicating phosphorylation as a potential regulatory mechanism. The mRNAs for numerous subtypes of putative G γ s have been identified in mammalian tissues, but little is known about their expression in brain, so that the systemic survey of the localization of mRNAs encoding twelve of G γ s in brain is needed to be performed. This study presents the localization of mRNAs encoding G $\gamma 12$ by quantitative RT-PCR and Northern or in situ hybridization in 8 different regions of rat brain: (1) frontal cortex area, (2) cerebral cortex area, (3) striatum, (4) hippocampus area, (5) thalamus, (6) brain stem, (7) cerebellum area, (8) hypothalamus-amygdala-septum-preoptic area. Striking region-specific patterns of expression were observed. The results show that G $\gamma 12$ expressed very well in frontal cortex and brain stem and comparatively not in other regions. Therefore, although G $\gamma 12$ has full activity for many effectors including phospholipase C and adenyl cyclase, G $\gamma 12$ is region-specifically expressed in brain and there may be its own specialized role for G $\beta\gamma$ containing this subunit in cell signaling.

[PA1-63] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Histamine Releasing Factor (HRF) Evokes [³H]Dopamine Release by a Ca²⁺ - independent Pathway in Pheochromocytoma Cells

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The recombinant histamine-releasing factor (rHRF) has been reported to induce a secretion of histamine and cytokines from inflammation-related cell types such as basophils and eosinophils, and to function as a growth factor in immune B cells. Recently, decreased expression level of HRF protein was observed in brain of patients with Alzheimer disease and Downs syndrome, suggesting a possible significant role in neurological systems. The novel functional role of HRF in dopamine release from