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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 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.

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

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