Oral Presentations - Field A

[A1. Pharmacology] [A2. Therapeutics] [A3. Hygienics] [A4. Toxicology]

[OA-1] [04/18/2003 (Fri) 13:30 - 13:45 / Orchid]

Esophagitis and IL-1β-induced alteration of MAP kinase activity in esophageal smooth muscle

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We investigated whether experimental esophagitis and IL-1β could induce the activation of MAP kinases in esophageal smooth muscle. With two models of experimental esophagitis, we assessed the activity of p38 MAP kinase, p44/42 MAP kinase and JNK. In feline acute experimental esophagitis, immunoblotting of normal and esophagitis-induced smooth muscle with each types of MAP kinase antibodies revealed the slight increase of phosphorylated form of p38 MAP kinase, especially in membrane fraction. JNK activity was also increased, but the increase was detected in cytosolic fraction. The amount of phosphorylated form of p44/42 MAP kinase in esophagitis-induced smooth muscle showed the increase of activity in cytosol and the decrease in membrane fraction, compared with normal esophagus. Total PKCβII and PKCε were also increased by inflammation, especially in cytosolic fraction. Second, surgically induced reflux esophagitis of rats showed time-dependent increase of ulcer index (UI), resulting in UI 4 after 6 hours. After the increase of phosphorylation of p38 MAP kinase in 4 hours (UI = 1), it was decreased below the basal level in 6 hours. The activity of JNK was increased with accordance with the progression of esophagitis. The level of phosphorylation of p44/42 MAP kinase was increased in 1 hour and decreased in 4 hours. After 6 hours, it was recovered to the basal level. The amount of COX2 expression was not changed with the progression of esophagitis.
In cultured feline esophageal smooth muscle cells, the phosphorylated forms of p44/42 MAP kinase and p38 MAP kinase were increased 1 hour after IL-1β treatment (25ng/ml), which is maintained to 24 hours. PLC inhibitor neomycin decreased the density of p44/42 MAP kinase band to the basal level. Tyrosin kinase inhibitor tyrphostin 51 and PKC inhibitor GF109203X also reduced the IL-1β-induced MAP kinase activity.
With these results, we suggest that the each type of MAP kinases shows different features of activation and deactivation in experimental esophagitis models and IL-1β -induced MAP kinase activation might be mediated by tyrosine kinase, PLC and PKC.

[OA-2] [04/18/2003 (Fri) 13:45 - 14:00 / Orchid]

On Sample Size Calculation in Bioequivalence Trials

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Sample size calculations plays an important role in a bioequivalence trials and is determined by considering power under the alternative hypothesis. The regulatory guideline recommends that 2 x 2 crossover design is conducted and raw data is log-transformed for statistical analysis. In this