

level of NO in brains. The expression of NOS was not also influenced by MFs exposure. These results suggest that MFs exposure can affect NO levels in rat brain. Further studies remains to be performed to confirm the definite relevance of MFs and NO system.

Poster Presentations – Field A2. Therapeutics

[PA2-1] [04/20/2001 (Fri) 10:30 – 11:30 / Hall 4]

Neuroprotective effect of KR-31378 in cortical cell culture and in brain ischemia-reperfusion model

Kim SO^o, Cho IS, Gu HG, Hwang GS, Lee DH, Hong L and Yoo SE

AgroPharma Research Institute, Dongbu Hannong Chemical Co., Korea Research Institute of Chemical Technology

Despite of considerable research efforts for the development of neuroprotective agents to save neurons from the biochemical and metabolic outcomes of ischemic brain injury, most of neuroprotective agents studied so far have shown their lack of clinical efficacy. This failure is probably due to the complex and multifactorial nature of ischemic brain injury. Thus, it is probably necessary to protect damages from two key events, ischemic injury and reperfusion injury.

Inhibition of lipid peroxidation induced by reactive oxygen species and stabilization of membrane have both been proposed as neuroprotective strategies in stroke. In this context, KR-31378, a novel benzopyran derivatives with N-cyanoguanidine group were synthesized as a new therapeutic strategy for neuroprotection. Possessing both anti-oxidant and potassium channel modulating activities without vasorelaxation activity, it has shown to protect cultured rat cortex neurons against oxidative injury induced by FeSO₄ and by hypoxia-reperfusion in vitro. Also, Treatment with KR-31378 significantly reduced brain damage in transient ischemia rat model, demonstrated by reduced infarct size, edema and mortality. Furthermore, treatment with KR-31378 starting up to 2h after reperfusion maintained its neuroprotective effect. All these results indicate that KR-31378 represents a potentially useful therapeutic agent for the treatment of ischemic/reperfusion brain injury.

[PA2-2] [04/20/2001 (Fri) 10:30 – 11:30 / Hall 4]

Study on toxicity and in vitro cytotoxicity of ID6105 (Hyribicin), A new anticancer agent

Ryu Jung Su, Hong-Sub Lee, Hang-Sub Kim¹, Tae-Yong Kim, Jung-Jun Lee¹, Kang Jong Gu², Kee-Won Kim

Research Laboratories, Ildong Pharmaceutical Co., Ltd., Korea Research Institute of Bioscience and Biotechnology 1, College of veterinary medicine, ChungBuk university²

New anthracyclines, ID6105 (Hyribicin) and ID6101 (Aclacinomycin X), were isolated from *Streptomyces galilaeus* ATCC 3113 containing dnrF. The in vitro cytotoxicity of ID6105 and ID6101 was tested on murine and human cancer cell lines in comparison with adriamycin and aclacinomycin A by SRB assay. The acute toxicity of these compounds for mouse was also tested. As a result, the ID6105 was 10~100 times more cytotoxic than adriamycin, and 3 ~10 times than