receptor supersensitivity, as shown by the enhanced ambulatory activity after administration of apomorphine (2 mg kg^{-1} s.c.). Glycine inhibited the development of postsynaptic dopamine receptor supersensitivity induced by repeated administration of morphine.
Opiate sensitization models demonstrate enhanced activating and rewarding effects of subsequent treatments which highlights the potential role of this phenonmena in drug addiction. Accordingly, the inhibition of glycine on the morphine-induced hyperactvity, reverse tolerance and dopamine receptor supersensitivity suggests that glycine might be useful for the treatment of morphine addiction.

[PA1-57] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Effects of adenosine on the development of tolerance to and physical dependence on morphine in mice

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This study was performed to investigate the effects of adenosine on the development of tolerance to and physical dependence on morphine. Repeated adminstration of morphine developed tolerance and physical dependence. Adenosine (1, 2 and 4 mg kg^{-1} i.p.) was administered intraperitoneally to mice for 7 days once a day 30 minutes prior to the morphine (10 mg kg^{-1} s.c.). Analgesic resposes were estimated at 0. 30. 60. 90, 120 minutes by the tail flick methods 24 hours after the final injection of morphine. The inhibitory degree of morphine tolerance development of the test morphine (10 mg kg^{-1} s.c.) by i.p. administration of adenosine was evidenced by the increase in analgesic response to morphine (5 mg kg^{-1} s.c.). Adenosine inhibited the development of tolerance to morphine. In addition, we separately measured the naloxone (5 mg kg^{-1} i.p.)-precipitated withdrawal sign (jump) in mice that had received the same morphine (10 mg kg^{-1} s.c.) for 7 days. Adenosine (1, 2 and 4 mg kg^{-1} i.p.) inhibited naloxone-precipitated withdrawal in morphine dependent mice.
These results sugest that adenosine might be useful the prevention or treatment of morphine tolerance and physical dependence.

[PA1-58] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Alternation and significance of calcium signalings through ryanodine receptor in neuronal cell death

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Pathophysiological elevation of intracellular calcium concentration ($[Ca^{2+}]_i$) in the neuron has been considered as an important responsible factor in the neuronal cell damages. However, the mechanism of increase of $[Ca^{2+}]_i$ through ryanodine receptor (RyR), the relationship between $[Ca^{2+}]_i$ level and cell damages have not been fully demonstrated. We now report that PC12 cells and primary hippocampal neuron cells exhibit greatly increased levels of RyR and enhanced