The effects of this substance on the locomotor activity of rats were compared to those of d-amphetamine. Both substances were found to induce a similar degree of hypermotility with (−)cathinone being approximately one third as potent as d-amphetamine. Furthermore, the effect of (−)cathinone on the locomotor behavior of hypophysectomized rats was analogous to that reported for d-amphetamine in such animals. The results support the claim that the symptoms caused by the chewing of khat are amphetamine-like.

Indirect sympathomimetic actions of the MAO inhibitor deprenyl at high doses

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L-Deprenyl (D), a type B MAO inhibitor, combined with L-DOPA has been proposed for the treatment of Parkinsonians (Birkmayer et al., Lancet *I*, 439, 1977). The MAO inhibition is probably due to D itself since the microsomal enzyme inhibitor proadifen (PRO) increased D-induced MAO inhibition in rat liver and brain and enhanced D-induced decrease of HVA in rat brain. However, D must also be considered a prodrug since it is metabolized to metamphetamine (MA, Reynolds et al., Br. J. clin, Pharmac. *6*, 542, 1978). In rats with 6-OHDA-induced unilateral lesion of the nigro-striatal pathway, D (10 mg/kg i.p.) induced circling as seen with MA. Haloperidol antagonized both D- and MA-induced circling, but PRO only D-induced circling. In rats with low plasma noradrenaline (NA) after ganglionic blockade and MAO inhibition (pargyline) D increased plasma NA levels several-fold. Indirect sympathomimetic activity of D is also exerted in the CNS since D enhanced K^+-induced release of ^{3}H-NA from rat cerebral cortex slices.

Computer analysis of the rats' sleep stages

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Simultaneous EEG recordings are made from 4 rats using a 16-channel Mingograph. From each rat EEGs of the cortex occipitalis and EMG are recorded. Since in paradoxical sleep (PS) signals of high frequency are superimposed on the characteristic ϑ-rhythm, one cortical EEG is additionally filtered above 10 Hz. The EMG is transformed into a frequency band of 1–32 Hz by an amplitude discriminating frequency-voltage converter. A Normalized-Slope-Descriptor computes the Hjorth parameters of activity (μV), mobility (Hz) and complexity (Hz) (Hjorth, Electroenceph. clin. Neurophysiol. *34*, 321, 1973) during a selected epoch duration of 8 sec. The parameters of each epoch are continuously stored on a digital magnetic tape and computer analyzed off line to determine the means and standard deviations of the parameters during the whole recording time. In a second step these values are used to attach to each epoch one of 6 different patterns of the Hjorth parameters associated with different stages of sleep and waking.

Early biochemical effects of a tumor promoting phorbol ester on mouse fibroblasts and their modulation by dexamethasone

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The phorbol ester 12-O-tetradecanoylphorbol-13-acetate (TPA) induces inflammation, proliferation and tumor promotion. In addition, TPA is a potent releaser of prostaglandins in vitro. It has been suggested that prostaglandins might be initiators of a cascade of events finally causing increased cell proliferation. We have tested this hypothesis in cultures of confluent mouse 3T3 fibroblasts. We found that addition of 10^{-7} M TPA to the cells is followed successively by release of prostaglandin E_2 (PGE_2) induction of the enzyme ornithine decarboxylase (ODC), stimulation of ^{3}H-thymidine incorporation into DNA and cell proliferation. Pretreatment of the cells with the anti-inflammatory steroid dexamethasone (10^{-6} M) inhibits TPA-induced PGE_2 release but enhances the other effects of TPA. Addition of PGE_2 to the culture fluid only at high doses (10^{-5} M) has a marginal stimulating effect on ODC induction and cell proliferation. These data suggest that the proliferative effect of TPA on 3T3 cells is not mediated by PGE_2.

Blood platelets as models for neurons: receptor- and nonreceptor-mediated membrane effects

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In human and rabbit platelets the shape change reaction (transition from discoid to spheroid from) caused by 5-hydroxytryptamine (5HT) agonists showed stereoselectivity and was specifically inhibited by 5HT-antagonists. Butanol (3%) which removes adhering plasma proteins from the cell membrane abolished this shape change. D-LSD a mixed 5HT agonist/antagonist showed saturable, high affinity binding to the platelet membrane. Various drugs, which counteracted the LSD- and 5HT-induced shape change also antagonized LSD-binding. Random coil basic proteins, e.g. myelin basic protein also caused a shape change reaction. This was enhanced after butanol treatment of the platelets and antagonized by the polyanion heparin though not by 5HT-antagonists. In conclusion, 5HT-agonists and antagonists, in contrast to basic proteins probably influence platelet function by interfering with specific membrane receptors. Both types of shape change inducers have been reported to act on neurons of the central nervous system.

5HT neurons in rat brain after acute and chronic treatment with p-chloro-amphetamine (PCA) and p-chloro-N-methylamphetamine (PCMA)

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PCA and PCMA are classified as serotonin (5HT) neurotoxins since they induce e.g. a long-term decrease of brain 5HT. However morphological results showing neuronal degeneration are inconclusive. We hoped to find more marked (toxic) effects after 20-day PCA or 32-day PCMA treatment (increasing doses 5–30 and 5–40 mg/kg i.p., respectively) than after 1×35 mg/kg. However no further decrease of brain 5HT and no delay of 5HT recovery during 40 days was obtained. Formaldehyde-induced 5HT fluorescence accumulated in swollen axons 1–6 days after 1 dose, but similar axons were rare after chronic treatment possibly indicating acute degeneration and ongoing de- and/or regeneration of some 5HT axons. No signs of degeneration were revealed by either quantitative fluoresc.-histo-chem. of metencephalic 5HT cells or by EM of n.suprachiasmaticus (rich in 5HT nerve terminals) and identified (supraependymal) 5HT terminals. Damage to other 5HT nerve terminals cannot be excluded.