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Pineal - related changes in cyclic AMP levels of rat medial basal hypothalamus[1]

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Summary. Pinealectomy (Px) in adult male rats resulted in increased cyclic AMP accumulation by medial basal hypothalamic (MBH) explants 3 and 7 days after surgery. 24 h after superior cervical ganglionectomy (Gx) an augmented MBH cyclic AMP accumulation was observed. The effects of Px and Gx were additive, as revealed in animals subjected to Gx 3 days after Px.

It is now accepted that the mammalian pineal gland is an endocrine organ that produces hormones with activity on the neuroendocrine system[3]. One of these hormones, melatonin, is secreted as a function of time of day in all vertebrates studied as yet, and acts on the brain to influence adenohypophyseal function. Receptors for melatonin are present in medial basal hypothalamic (MBH) membranes and several hypothalamic metabolic functions change after melatonin treatment, including protein synthesis, serotonin and γ-aminobutyric content, neurotransmitter uptake and neurohormone release (for references, see Cardinali[4]). Some of the actions of melatonin on MBH probably involve effects on cyclic nucleotide levels; melatonin decreases in physiological concentrations, cyclic AMP accumulation of rat MBH in vitro[5]. The present study aimed to examine cyclic AMP accumulation in MBH explants obtained from rats subjected to pinealectomy (Px) or superior cervical ganglionectomy (Gx). The latter procedure removes almost all functionally relevant pineal innervation, and interrupts the peripheral sympathetic innervation to the CNS.

Material and methods. Adult male Wistar rats were kept under light between 07.00 h and 21.00 h daily and were given access to Purina chow and water ad libitum. Px, Gx or sham-operation were performed under light ether anesthesia as described before[6]. The rats were killed 3 or 7 days after Px; Gx was performed 24 h before sacrifice. The day before Gx the animals were kept for 24 h under light; this procedure is known to induce postsynaptic supersensitivity of β-adrenoceptors in one of the superior cervical ganglia territories at least, i.e. the pineal gland[7]. The rats were killed by decapitation and the brains were quickly removed. Individual MBH (about 4 mg wet weight) were incubated at 37 °C in 0.2 ml Krebs Ringer bicarbonate buffer, ph 7.4, containing glucose (1 mg/ml) and 1 mM theophylline, as described elsewhere[5]. Cyclic AMP was extracted by homogenization of tissue in 5% trichloroacetic

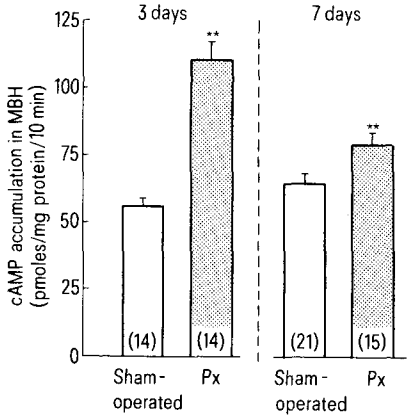

Figure 1. Effect of pinealectomy (Px) performed 3 or 7 days before sacrifice on cyclic AMP accumulation by rat medial basal hypothalamic (MBH) explants. Shown are the means + SEM (n). ** $p < 0.01$, Student's t-test.

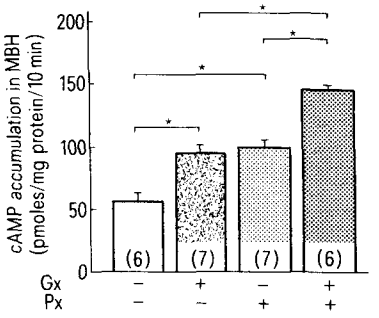

Figure 2. Effect of pinealectomy (Px) and/or superior cervical ganglionectomy (Gx) on cAMP accumulation by rat medial basal hypothalamic (MBH) explants. Gx was performed 24 h before sacrifice while Px was performed 3 days before sacrifice. Shown are the means + SEM (n).* $p < 0.05$, analysis of variance, Scheffé's test.

acid and was measured in triplicated samples by the saturation binding method of Brown et al.[8]. Results were expressed as pmoles of cyclic AMP accumulated per mg of MBH protein per 10 min. Statistical analysis of results was carried out by Student's t-test or by analysis of variance followed by Scheffé's test.

Results. The effect of Px on MBH cyclic AMP is shown in figure 1. Pineal ablation increased MBH cyclic AMP accumulation significantly, by 98 and 33%, 3 and 7 days after surgery, respectively. 24 h after Gx an augmented cyclic AMP accumulation was observed in rat MBH (fig. 2). Gx and Px had additive effects, as revealed by the increased MBH cyclic AMP accumulation in rats subjected to Gx 3 days after Px. In agreement with the results of figure 1, Px alone increased MBH cyclic AMP accumulation by 52% 3 days after surgery (fig. 2).

Discussion. Norepinephrine increases cyclic AMP synthesis in rat hypothalamic and cortical slices by interacting with both α- and β-adrenoceptors, the α-adrenergic response requiring the synthesis of prostaglandin E_2[9]. After Gx some of the hypothalamic noradrenergic terminals undergo degeneration, as is revealed by the decreased transmitter uptake[10] and content[11] in MBH or median eminence of Gx rats. Therefore, a possible explanation for the enhanced MBH cyclic AMP accumulation 24 h after Gx (fig. 2) could be a direct effect of the norepinephrine released from degenerating peripheral nerve terminals in situ, known to occur within the first 24 h after nerve section[12]; however, further experiments employing adrenergic blocking agents are needed before a definitive conclusion is reached in this respect. A link between Gx and α-adrenergic mechanisms in MBH is suggested by the increase in α-adrenoceptor sites and α-adrenergic responses in MBH of rats killed 7 days after Gx[10].

There is good agreement between our prior observations on the melatonin-induced cyclic AMP decrease of rat MBH explants[5], and the fact that the abolition by Px of the main melatonin source in the body augmented cyclic AMP accumulation in MBH of rats (fig. 1). Within 16-24 h after Gx, and as a consequence of degeneration of peripheral sympathetic nerve endings in the vicinity of the pinealocytes, melatonin synthesis increases several-fold in rats[12]. Since melatonin decreases MBH cyclic AMP, the additive effect of Px on Gx stimulation of cyclic AMP synthesis is best explained in terms of the removal of a negative influence on the hypothalamic parameter examined. However, the inhibitory influence exerted by the pineal gland and presumably melatonin, is not strong enough to overcome the stimulation of MBH cyclic AMP synthesis produced by the norepinephrine release in situ from degenerating nerve varicosities.

Melatonin depresses in vitro the spontaneous and norepinephrine-induced release of prostaglandin E_2 by rat MBH[13]; at the same physiological concentrations, melatonin also inhibits cyclic AMP accumulation by hypothalamic explants[5]. Since the α-adrenoceptor - mediated effect of norepinephrine on MBH cyclic AMP requires prostaglandin E_2 synthesis[9], the possible involvement of these mechanisms in pinea-mediated influence on cyclic nucleotide levels should be considered.

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Association of the degree of methylation of intercellular pectin with plant resistance to aphids and with induction of aphid biotypes

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Summary. Increased methylation of middle lamellar pectin in plants hinders aphids in penetrating host-plant tissue. In sorghum, a new aphid biotype has overcome this host-plant barrier by having increased pectin methylesterase activity. Results suggest that resistance in crop-plants against sap-feeding insects may possibly be manipulated by altering middle lamellar chemistry either through breeding or use of certain plant-growth regulators.

This paper discusses how resistance of sorghum to an aphid-pest is associated with the structure of middle lamellar pectin in the plant. This material plays a major role in mediating the penetration by stylets of sap-feeding insects through host-plant tissues. The manipulation of the nature of plant intercellular pectins, either through plant-breeding or by application of certain plant-growth regulators, could result in improved resistance in crops against pests whose feeding sites are in localized, internal tissues of plants.

Host-plant resistance (HPR) to insects is frequently ascribed to the presence of secondary plant metabolites[1]. In earlier studies on HPR in sorghum[2] towards the aphid,