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upon parasitic stimulation. Furthermore, the presence of schistosomin coincides with a special developmental stage of the parasites, i.e. with the occurrence of differentiating cercariae in daughter sporocysts. Apparently cercariae induce the release of schistosomin. Research is now in progress to characterize the parasitic inducing factor involved.

18. H. DE BRABANDER, J. P. G. DE BRUIN & C. G. VAN EDEN (Netherlands Institute of Brain Research, Amsterdam): *Is dopamine involved in functional recovery after cortex lesions in male rats.*

It is generally known that the prospects for behavioural recovery are better after damage of the central nervous system sustained early in life than in adulthood. The question to consider is which neural mechanisms are responsible for this difference in recovery.

Behavioural tests demonstrated that animals with neonatal medial prefrontal cortex (mPFC) lesions were unimpaired in the performance of the learning task spatial delayed alternation, whereas animals with similar brain damage sustained in adulthood performed poorly in this task. Neural reorganisation of a PFC system could be responsible for this sparing of function. We have examined whether neonatal lesions resulted in changes in the afferent innervation of the PFC. The dopaminergic innervation was found to differ considerably: in animals with neonatal lesions the density of dopaminergic fibers, in the cortex adjacent to the lesion, was higher, the fibers were thicker and there were more and thicker varicosities. No such changes were found in animals with adult mPFC lesions.

Thus, we postulate that the increased dopaminergic innervation could contribute to the observed sparing of function following neonatal mPFC damage.

19. A. E. LAMERS, P. H. M. BALM, H. E. M. G. HAENEN & S. E. WENDELAAR BONGA (Department of Animal Physiology, University of Nijmegen): *The pars intermedia and stress acclimation in Fish.*

POMC derived ACTH is traditionally considered the activator of the pituitary-adrenal-axis. However, we provided evidence, that also  $\alpha$ MSH, the background adaptation hormone that is processed from POMC as well, stimulates the release of cortisol from interrenal tissue in tilapia (*Oreochromis mossambicus*). Moreover we demonstrated that the different  $\alpha$ MSH forms have different corticotropic potencies: mono-acetyl < des-acetyl < diacetyl. Hence we concluded that acetylation is of functional significance.



We observed that all three  $\alpha$ MSH forms are present in tilapia  $\alpha$ MSH cells and plasma. The di-acetyl/mono-acetyl  $\alpha$ MSH ratio is higher in  $\alpha$ MSH cells than in plasma. This suggests that the  $\alpha$ MSH forms are not released in proportion to their intracellular concentrations. We investigated the possibility of regulation of this differential release by secretagogues. TRH increased the di-acetyl/mono-acetyl ratio of released  $\alpha$ MSH. These results show that fish can modulate not only the quantitative, but also the qualitative signal of the  $\alpha$ MSH cells. This shift in di-acetyl/mono-acetyl ratio could also be observed in fish acclimating to low environmental pH. Since cortisol is of importance during acclimation in these fish, the possibility that  $\alpha$ MSH plays a role in pH stress acclimation will be discussed.

20. I. S. WEIMAR, D. M. PREKER & H. J. Th. GOOS (Department of Experimental Zoology, University of Utrecht): *Steroid feedback: receptor modulation?*

In the African catfish (*Clarias gariepinus*) gonadotropin (GTH) is stimulated by gonadotropin releasing hormone (GnRH). The effect of GnRH is inhibited by dopamine, which is—like GnRH—produced in the hypothalamus and released in the pituitary by neurosecretory fibers. Also steroid hormones have a decreasing effect on the GTH-release, but it is unknown yet at which level the steroid hormones exert their negative influence.

A hypothesis is, that steroid hormones exert their negative feedback on the GTH-release by a down-regulation of pituitary GnRH receptors. In castrated fish, the number of GnRH receptors two weeks after castration is significantly elevated. If castration is followed by replacement of androstenedione or testosterone, GnRH receptor density is decreased.

To establish whether the steroids exert their decreasing effect on the pituitary GnRH receptors directly or indirectly, the following experiment was carried out.

Adult male African catfish were castrated. After two weeks the pituitaries were collected, and treated in a perfusion system with the following steroid hormones: androstenedione, testosterone and their non-aromatizable forms:  $11\beta$ OH-androstenedione and  $11\beta$ OH-testosterone. After a six hour treatment, the pituitaries were collected from the perfusion system and the GnRH binding was measured using a GnRH radio receptor assay.

The GnRH receptor content of the castrated group was significantly elevated compared to the sham operated group, as was already shown in previous experiments.