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A Hormonal Component in Central Vestibular Compensation

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In developing clawed toads *Xenopus laevis*, the compensation mechanism of behavioural defects caused by unilateral labyrinthectomy consists of two components, a vestibular one and a hormonal one. Thyroxine accelerates this compensation.

Unilateral labyrinthectomy induces many behavioural defects, for example rotatory body movements, asymmetrical eye positions and limb postures, etc. [1]. The compensation mechanisms of these defects have been investigated using different methods [1, 2]. These investigations proved the strong influence of the intact labyrinth, the cerebellum, the formatio reticularis, and the spinal cord on the restoration of symmetrical behavioural reactions. In tadpoles of the South African Clawed Toad *Xenopus laevis* Daudin, Horn and Rayer [3] demonstrated that the compensation time for the movement defect depends on the developmental stage at which the operation was performed. The older tadpoles are at the operation the longer it takes for the compensation.

During the development, thyroxine plays an important role in the differentiation processes of the brain [4]. Therefore, besides the vestibular component a hormonal component may influence the compensation processes. Hypophysectomy is a suitable method of seperating the vestibular component from the hormonal one, because this operation causes a retardation of the further differentiation in the tadpoles [5]. On the other hand, thyroxine treatment accelerates the differentiation.

Tadpoles hemilabyrinthectomized at stage 54 were hypophysectomized one day later. The first normal swimming tadpole was observed 6 weeks after both operations, whereas in the non-hypophysectomized group, this happens two weeks after the operation (Fig. 1). In contrast, the compensation of movement defects in thyroxine-treated tadpoles,

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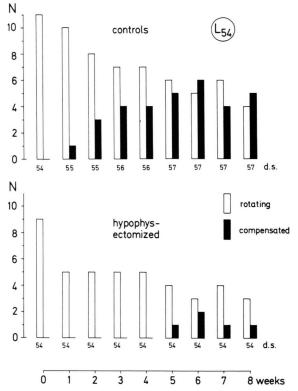


Fig. 1. The influence of hypophysectomy on the compensation of rotatory movements after unilateral labyrinthectomy. The tadpoles were operated at stage 54. Below each column, the developmental stage d. s. of the experimental animals is indicated. N number of tadpoles.

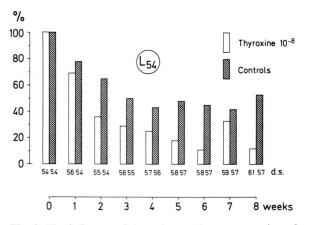


Fig. 2. The influence of thyroxine on the compensation of rotatory movements after unilateral labyrinthectomy. The tadpoles were operated at stage 54. Below each column, the developmental stage d. s. of the experimental animals. % percentage of rotating tadpoles; 100% = 27 tadpoles.



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which were hemilabyrinthectomized at stage 54, is remarkably accelerated. While in the non-thyroxine treated group only 45–50% of the hemilabyrinthectomized tadpoles have compensated the movement defect after 8 weeks, more than 80% of the thyroxine-treated tadpoles swim normally after the same time (Fig. 2). In thyroxine-treated tadpoles, hemilabyrinthectomized at stage 58, the acceleration of the compensation occurs especially during the first and second week.

These investigations prove that in tadpoles of *Xenopus laevis* the compensation mechanism of behav-

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 W. Precht, Handbook of Sensory Physiology VI/2 (H. H. Kornhuber, ed.), pp. 451-462, Springer-Verlag, Berlin 1974. ioural defects caused by unilateral labyrinthectomy consists of at least two components, a vestibular one and a hormonal one. It is possible, that other hormones which play a role in differentiation processes of developing animals, for instande prolactin in amphibians [6] and growth hormone in higher vertebrates may also influence the central vestibular compensation during development.

Acknowledgement

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