Thyroid affects color vision

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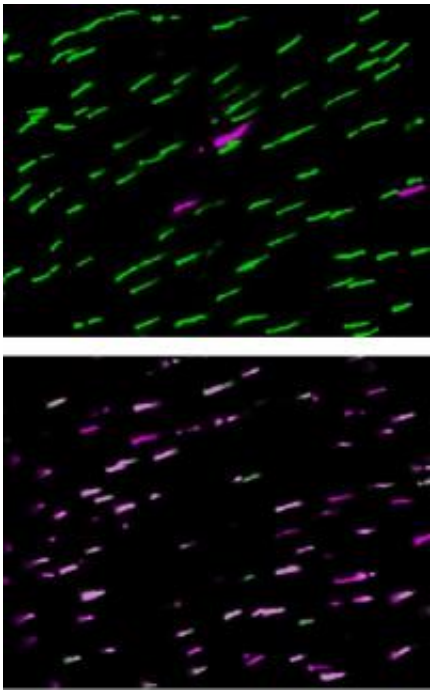

Fluorescence micrographs of the cone cells in the retina of an adult healthy rat (top) and of an adult rat with thyroid hormone deficiency (bottom). The cones were labeled with antibodies against their opsins; green opsin is shown in green and UV/blue opsin in magenta. The healthy rat has many green cones and few UV/blue cones. The rat with thyroid hormone deficiency expresses UV/blue opsin in all cones and reduces expression of green opsin. Appearing in lighter magenta in the bottom image are cones that contain some green opsin in addition to the dominant UV/blue opsin. Credit: © Martin Glösmann

What part does the thyroid gland have in vision? Thyroid hormone is crucially involved in controlling which visual pigment is produced in the

cones. Previously, it was assumed that the colour sensitivity of the cones is fixed in the adult retina.

Researchers at the Max Planck Institute for Brain Research in Frankfurt/M., together with colleagues at the University of Frankfurt and universities in Vienna, have now been able to show that in mature cones of mice and rats the production of visual pigment is regulated by thyroid hormone. It is assumed that this mechanism exists in all mammals, including humans. If so, the adult-onset of thyroid hormone deficiency would affect colour vision.

Thyroid hormone has a crucial role during development of the body and also of the nervous system. Children born with a thyroid hormone deficiency have serious defects of physiological and mental development, hence newborns are routinely checked for thyroid hormone deficiency, and hormone substitution therapy is given when indicated.

Studies in mice have shown that thyroid hormone also plays an important role in the development of the eye and particularly the cone visual cells. In the retina of the eye, the cones are the visual cells responsible for colour vision. Most mammals have two spectral cone types containing either of two visual pigments (opsins), one sensitive to shortwave light (UV/blue opsin), the other to middle-to-longwave light (green opsin). Cones express a thyroid hormone receptor. Its activation by the hormone suppresses the synthesis of UV/blue opsin and activates the production of green opsin.

Until now, the control of opsin production by thyroid hormone was considered a developmental phenomenon. Experts assumed that in mature cones the developmentally established 'opsin program' is fixed and needs no further regulation. This perception is now challenged by a study carried out by lead authors Martin Glosmann and Anika Glaschke

in Leo Peichl's team at the Max Planck Institute for Brain Research, Frankfurt, and their colleagues at the universities of Frankfurt and Vienna. The study shows that opsin production in mature cones continues to depend on the thyroid hormone level. The researchers had started with an analysis of thyroid hormone involvement in the early postnatal development of mouse cones. "Then we wanted to know how long the time window for the hormone effect was, at what point the hormone's influence on opsin production stopped", says Anika Glaschke.

"To our surprise we did not find such an endpoint, even several weeks after birth there was a hormone effect". So the team analysed the cones in adult mice and rats that had been rendered hypothyroid for several weeks. In these mice all cones switched to the production of UV/blue opsin and reduced green opsin production. After termination of the treatment, hormone levels returned to normal and the cones reverted to the production of their 'regular' opsin - one cone type to green opsin, the other to UV/blue opsin. The researchers conclude that the spectral cone types, which are defined by the opsin they express, are dynamically and reversibly controlled by thyroid hormone throughout life.

"In addition to their importance for basic retinal research, our findings may also have clinical relevance", says Martin Glösmann, who currently examines the genetic foundations of the process at the University of Veterinary Medicine, Vienna. "If this mechanism also acts in human cones, the adult-onset of thyroid hormone deficiency - e.g. as a consequence of dietary iodine deficiency or removal of the thyroid - would also affect the cone opsins and colour vision". There are no such reports in the clinical literature, presumably because the general symptoms of thyroid hormone deficiency are so severe that therapy is initiated before the cone opsin shifts would show up.

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