# REPORT

*Al collegio docenti del Dottorato in Medicina Molecolare,*

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**Introduction:** autophagy is an essential process in maintaining the normal cellular homeostasis and energy balance under physiological conditions. In retinal diseases, autophagy helps retinal cells defend themselves against harmful stress; however, excessive autophagy may result in retinal deterioration. Modulating autophagy may provide an alternative therapeutic strategy to treat retinal diseases. Retinopathy of prematurity (ROP) is a human vascular disease characterized by the formation of neovascularization in the premature newborn retina. There is only limited information on the role of autophagy in ROP.
**Methods:** a rat model of oxygen-induced retinopathy (OIR), an acknowledged model of ROP, was used to evaluate changes in the expression of key mediators of autophagy induced by the disease. In this model, rat pups are exposed to alternating cycles of 50% and 10% oxygen for 24-h for the first 14 days followed by exposure to the room air until post-natal day 18. To analyzed retinal vasculature in the OIR model, two techniques were employed: immunofluorescence and Evans Blue dye. The expression of autophagy markers from birth to post-natal day 18 was assessed at transcript and protein levels by real-time PCR and Western blot, respectively. In addition, the phosphorylation status of signaling molecules known to activate or to repress autophagy was investigated.
**Results:** in control rats, the autophagic flux decreased over time during post-natal development. In contrast, in OIR rats the levels of autophagic markers transiently increased at post-natal day 7 to then gradually decrease until post-natal day 18.

**Gratification course:** good
**Congress:** Noemi Anna Pesce, Filippo Locri1 Helder André, Massimo Dal Monte. *"Changes in the expression of autophagy markers in a rat model of retinopathy of prematurity".* Joint Meeting *of the* Federation of European Physiological Societies and the Italian Physiological Society; September 2019 Bologna.

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