Review of: "Melanosomes degrade lipofuscin and precursors that are derived from photoreceptor membrane turnover in the retinal pigment epithelium—an explanation for the origin of the melanolipofuscin granule"

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In this paper, the authors provide results for a new method, they patented, to get rid of ocular lipofuscin from retinal pigment epithelial (RPE) cells. The idea idea is very interesting and definitely needs to be investigated in more detail. Briefly, they show that an intraocular injection of nitric oxide (NO) generators promotes the reduction of lipofuscin-like material from melanolipofuscin, through a process that would involve the oxidation of melanin in those granules. The way oxidized-melanin promotes lipofuscin degradation is not clearly explained. In addition, in previous publications the authors showed that in ABCA4-/- retinas, the ocular lipofuscin mainly resides in the lipofuscin granules and secondarily in the melanolipofuscin granules. The method described here, would potentially get rid of the lipofuscin from the melanolipofuscin but not from the lipofuscin granules (that lack melanin). In addition, melanosomes are lysosomal related organelles. Their biogenesis of melanosomes in ABCA4-/- animals has not been studied but it could potentially be altered. Thus, the less dense material that the authors call here lipofuscin-like material has to be better characterized to rule out that it is not actually pheomelanin (a less dense form of melanin commonly co-existing with the dense eumelanin in the RPE). Finally, the thin (3–4 nm) lamellar membranes (TLMs) described here, as partially degraded outer segments, were actually described as residual bodies resulting from the degradation of melanosomes in a previous publication by the same group.