ISER 2024

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ISER 2024
PROGRAM ABSTRACTS

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Inhibiting sphingosine kinase 1 modulated autophagic flux in retinal pigment epithelial cells.

Poster number: W-33

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Introduction
Retinal pigment epithelial (RPE) cells play multiple functions in the retina, preserving visual functionality. Autophagy is crucial for RPE degradative functions, and its dysregulation contributes to pathological conditions. The roles of sphingolipids in autophagy regulation are scarcely known. Sphingosine kinase (SphK1), which catalyzes the synthesis of sphingosine-1-phosphate (S1P), has been shown to facilitate endosomal trafficking.

Objectives
Our purpose was to evaluate whether SphK1 activity modulated autophagic flux in RPE cells.

Methods
Human RPE cell line (ARPE-19) cultures were treated with 10 µM PF543, a SphK1 inhibitor, with NVP, a ceramide kinase inhibitor, with 5 µM S1P or 10 µM C1P, or with 10 µM Bafilomycin 1 (BafA1), for 24 and 48 h. Cell morphology was determined with phalloidin. Cell death was analyzed by MTT assay. Formation of autophagosomes was evaluated by immunocytochemistry, using antibodies for LC3b and p62, specific autophagosome markers.

Results
Inhibiting SphK1 with PF543 for 24 h promoted morphological changes in ARPE-19 cells and the formation of perinuclear round vesicles, scarce in controls, which increased after 48 h. PF543 also induced endoplasmic reticulum enlargement, but had neither an effect on mitochondria nor on cell viability. Blocking synthesis of ceramide-1-phosphate (C1P), whose functions are similar to those of S1P, did not induce vesicle formation. The vesicles showed intense labeling with LC3b and p62 antibodies, suggesting that they might be “autophagosomes”. Treatment of RPE cells 24 and 48 h with BafA1, which disrupts endocytic flow, led to the accumulation of LC3b- and p62-positive vesicles and alterations in cell morphology remarkably similar to those induced by PF543. The amount of LC3b- and p62-positive vesicles was further increased with the combined addition of PF543 and BafA1, suggesting that PF543 enhanced autophagic flux, and BafA1 prevented vesicle degradation. Supplementation with S1P 1 h after PF543 addition restored cell morphology but did not prevent vesicle formation.

Conclusion
Our results suggest that inhibition of SPhK1 promoted morphological changes in RPE cells and the formation of LC3b and p62-positive vesicles, possibly autophagosomes. Exogenous S1P preserved morphology but did not prevent autophagosome formation, implying S1P receptor activation did not regulate this formation and suggesting that SphK1 activity was essential for maintaining proper autophagic flux.