**EFFECT OF OLIVE OIL PHENOL HYDROXYTYROSOL (HT) IN PATIENT CELL MODEL WITH MITOCHONDRIAL COMPLEX DEFICIENCY**

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**Background and objectives**

**Background**

There is growing evidence suggesting that daily consumption of high phenolics-containing extra-virgin olive oil (EVOO) has beneficial effects on preventing neurodegeneration related to the high level of antioxidants (1-2). Previous literature demonstrated that phenolics such as hydroxytyrosol (HT) in EVOO, with strong antioxidant properties, are potential dietary supplements against the oxidative stress in brain tissue and protective effect on both acute and chronic neurodegenerative diseases by in vitro study and animal models (1,3).

**Objectives of our study**

We aim to investigate the effect of EVOO phenol, HT, on cell viability and oxidative stress induced by hydrogen peroxide in patient fibroblasts with mitochondrial complex IV deficiency, a neurodegenerative disease.

**Free radical scavenging ability of HT showing its antioxidant effect in patient cells**

- Patient cells with complex IV deficiency (caused by SURF1 or SCO2 genetic defect) were post-treated with HT after loading with DCFDA fluorescent probe for quantification of cellular ROS level.
- Figure A & B: Patient cells were exposed to DPBS and cellular ROS increased along 3 hours. 1 µM and 10 µM of HT abolished the cellular ROS increase demonstrating the ROS scavenging and antioxidant capacity of HT.
- Figure C & D: Patient cells had a even higher increase in ROS by exposing to H₂O₂ and 10 µM of HT was enough to scavenge all ROS induced by H₂O₂.

**Dose-dependent HT protection of cell viability towards oxidative stress of H₂O₂**

- Patient cells with complex IV deficiency (caused by SURF1/P gene defect) were pretreated with 0, 1, 10 and 20 µM of HT for 24 hours and then exposed to 100 µM H₂O₂ for 1.5 hours which decreased cell viability.
- 10 and 20 µM of HT pretreatment rescued the cells and increased cell viability significantly.

**Conclusions**

- The present study demonstrated that HT could protect patient cells with mitochondrial complex deficiency against oxidative stress of hydrogen peroxide by: 1) scavenging of ROS; 2) reducing cellular ROS production; 3) increase of cell viability.
- In mitochondrial diseases, defects of mitochondrial respiratory chain result in alteration of cellular redox state and increase in ROS production. The protective effect of HT against oxidative stress indicated its potential therapeutic role in mitochondrial diseases.

**Acknowledgement and References**

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