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| Dietary interventions in high anxiety: Focus on mitochondria  **Maria P. Papageorgiou** 1,2#, **Markus Nussbaumer** 1,2, **Martina Samiotaki** 3, **Marianthi Firoglani Moschi** 1,2and **Michaela D. Filiou** 1,2\*  1 Laboratory of Biochemistry, Department of Biological Applications and Technology, School of Health Sciences, University of Ioannina, Ioannina, Greece  2 Biomedical Research Institute, Foundation for Research and Technology-Hellas (BRI-FORTH), Ioannina, Greece  3 Biomedical Sciences Research Center "Alexander Fleming", Institute for Bioinnovation, Vari, Greece  # Presenting author: Maria P. Papageorgiou, email: <m.papageorgiou@uoi.gr>  \* Corresponding author: Michaela D. Filiou, email: <mfiliou@uoi.gr> |

abstract

Modern lifestyle comes along with high stress levels and unhealthy eating habits, leading concomitantly to a rapid increase in the rates of anxiety and eating disorders. Approximately half of eating disorder patients also suffer from mood disturbances, further highlighting the crosstalk of eating and anxiety disorders [1]. Mitochondria have been reported to modulate anxiety and disordered eating phenotypes [2,3]. However, how disordered eating patterns in highly anxious populations are mediated by mitochondria remains unexplored.

Here, we exposed female high anxiety-related behavior (HAB) and normal anxiety-related behavior (NAB) mice to temporal food restriction, using a limited food access (LFA) protocol, according to which mice had ad libitum access to food 2h/day, while the control group had ad libitum diet 24h/day. We then investigated the LFA effects on mouse behavior as well as hypothalamic proteome, mitochondrial gene expression and mitochondrial DNA copy numbers. We found that LFA exposure increased anxiety- and depression-like behavior in HAB LFA female mice compared to HAB controls, while exerting no effect on NAB mice. In the hypothalamus, diverse proteomic signatures associated with mitochondrial functions were observed in HAB LFA vs. HAB controls. Furthermore, LFA significantly reduced hypothalamic mitochondrial dynamics gene expression of fission markers in HAB LFA vs. HAB controls, although no differences were found in mitochondrial DNA copy numbers between the two groups. Our findings indicate a modulatory role of mitochondrial pathways in mood and eating disorders which may act as potential therapeutic targets for pertinent pathologies.

**REFERENCES**

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