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MITOPHAGY, AN ORGANELLE QUALITY CONTROL MECHANISM WAS ACTIVATED AFTER UVB RADIATION EXPOSURE[1]

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Introduction: Increased ultraviolet B (UVB) radiation, mainly caused by the depletion of the ozone layer, has become an environmental concern that potentially affects aquatic organisms. The wavelengths (280 – 215 nm) of this radiation, have the ability to induce damage to cell organelles such as mitochondria. As an important target of this radiation, mitochondria in *Macrobrachium olfersii* embryo cells, a suitable emerging model, could be affected compromising embryos and larvae development, as well as adult organisms. Mitophagy is an important mechanism that contributes to maintenance of cellular homeostasis by degrading altered mitochondria, and can be induced by UVB radiation. **Objectives:** This study aimed to investigate mitophagy activation after the UVB radiation exposure. **Materials and Methods:** In order to recognize the mitophagy activation during the embryonic development, embryos of *M. olfersii* were exposed to UVB radiation in laboratory conditions, using a 6W UVB lamp for 30 min, toward to simulate the natural UVB irradiation. Non-irradiated embryos were used as controls. For that, adults of *M. olfersii* were collected in Lagoa do Peri on the Santa Catrina Island - IBAMA (Authorization nº 15294-1/2008), for reproduction to occur in the laboratory. After 6 and 12 h of irradiation procedure, embryos were analyzed. To quantify transcription levels, genes associated with mitophagy (*PINK1* and *MAP1LC3*) were found in the transcriptome of *M. olfersii* embryos. cDNA synthesis was used to analyze gene expression by RT-qPCR, using Rpl8 as a reference gene. To evaluate the products of these genes, PINK1 and LC3B protein content were analyzed using immunofluorescence analysis. Data were evaluated by Students t-test for independent samples, differences between groups were considered significant when $p < 0.05$. **Results:** After UVB radiation exposure, PINK1 and MAP1LC3 transcript levels were overexpressed 6 h (2.76-, 7,29-fold, respectively, $p < 0.05$) and the protein content assays showed that PINK and LC3B increased 12 h after exposure. **Conclusions:** Our results indicate that mitophagy was activated after exposure to UVB radiation and could act as a protective mechanism to maintain cellular homeostasis. Together, these novelties contributed to the understanding of the impacts of UVB radiation, showing the ability of embryonic cells to mitigate its negative impacts, through the induction of selective autophagy of mitochondria.
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