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Abstract

Epibrassinolide Treatment Caused Autophagy or Apoptosis Decision in a Time-Dependent Manner through ER Stress in Colon Cancer Cells †

Pınar Obakan-Yerlikaya *, Kaan Adacan, Elif Damla Arısan, Ajda Çoker-Gürkan and Narçın Palavan-Ünsal

Department of Molecular Biology and Genetics, Istanbul Kultur University, Atakoy Campus, Istanbul 34156, Turkey; p.obakan@iku.edu.tr; Tel.: +90-212-498-4567

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Abstract: Epibrassinolide (EBR) is a natural plant polyhydroxysteroid with structural similarity to steroid hormones. Lately we showed by SILAC assay that EBR treatment induces apoptosis by significantly altering the expressions of proteins having role in unfolded protein response (UPR) and endoplasmic reticulum (ER) stress. ER stress level has been proposed as a critical step in cancer chemotherapy. Moderate ER stress was shown as an inducer of pro-survival machinery *via* autophagy, which is an important catabolic process that delivers cytoplasmic material to the lysosome for degradation. However chronic ER stress lead cells to apoptosis. EBR treatment induced ER stress in a time-dependent manner within 48 h in SW480 and DLD1 colon cancer cells. Downregulation of p62, LC3 and increase in ATGs expressions indicated that autophagy is induced in these cell lines after 12 h EBR exposure. The mammalian target of rapamycin (mTOR), a coordinator between nutritional stress and cellular growth machinery, is associated with ER stress. Our results indicated that short time EBR exposure induced mTOR expression accompanied by Ser2448 dephosphorylation. After 48 h EBR treatment, with prolonged ER stress, both cell lines undergo apoptosis. Therefore we conclude that time-dependent EBR treatment caused autophagy or apoptosis decision through ER stress in colon cancer cells.

Keywords: epibrassinolide; colon cancer; autophagy; apoptosis

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