



## Seminars in Biotechnology BTEC 591 & BTEC 691

### "Epibrassinolide: not just for plants anymore"

**Thursday, December 24, 2020**

**13:30**

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Pınar Obakan Yerlikaya completed her high school education in Istanbul Private Saint Joseph French High School and her undergraduate studies at the Department of Biology at Istanbul University. She completed her MSc studies in the field of Molecular, Cellular and Behavioral Neurosciences at the University of Paul Sabatier, Toulouse III (France), with the joint scholarship of the Turkish Education Foundation-French Embassy as well as Centre régional des œuvres universitaires et scolaires (CROUS) de Toulouse-Occitanie. She completed her doctoral studies in the field of Cancer Molecular Biology at Istanbul University and graduated in 2013. During her PhD studies, she worked as a visiting scientist at Rome La Sapienza University (Italy) in 2011. Following her PhD, she worked in Prof. Merali's Lab at Temple University, School of Medicine, Fels Institute for Cancer Research and Molecular Biology (USA) in 2013. She was promoted to Associate Professor position in the molecular cell biology field in 2017. She gives lectures on Molecular Biology Techniques, Neuroscience, RNA Biology, Genetics Laboratory and Animal Cell Culture. Her main research interest is the determination of molecular targets of various drug candidates in different cancer and neurodegenerative disease models (using in vitro cell culture, in vivo, and *Caenorhabditis elegans* models). Pınar Obakan Yerlikaya published 36 SCI-indexed papers and is co-author of 4 international book chapters and the "Molecular Biology Techniques" book. She holds 2 national patents and 2 PCT patent applications. She conducted TUBITAK-3001, COST Action (Genie BM1408) projects and had an EMBO networking grant. She also worked as a researcher in three TUBITAK-1001 projects, and one Horizon 2020 project, which has won the above-threshold award. Obakan-Yerlikaya is also the co-founder of DAP Genomics Biotech Company funded by the TUBITAK-1512 grant.

#### **Abstract**

Epibrassinolide (EBR), a member of brassinosteroids plant hormones with cell proliferation promoting role in plants, is a polyhydroxysteroid with structural similarity to steroid hormones of vertebrates. We previously demonstrated that EBR-induced apoptosis was due to increased unfolded protein response (UPR) as a consequent outcome of endoplasmic reticulum (ER) stress (1). We determined that ER stress-related chaperone calreticulin (CALR) was the primary target of EBR-mediated apoptotic response in cancer cell lines and colon carcinoma SCID mouse xenograft model (2).

Brassinosteroid signaling in plants requires the inhibition of BIN2 (brassinosteroid-insensitive 2), a plant GSK3-like kinase, to initiate the translocation of transcription factors to induce BR responsive gene expression (3). We recently showed that EBR could inhibit GSK3 $\beta$  in *Caenorhabditis elegans* models of Alzheimer's disease (AD) as well as in vitro neuronal cells. EBR treatment was successful to protect cells and increase the inhibitory phosphorylation of GSK3 $\beta$ . Besides, EBR exhibited a positive effect on survival and fertility rates of *C. elegans* strains. More interestingly, the paralyzed phenotype of AD *C. elegans* model was prevented (submitted, patent-holding).

**References:**

1. Obakan P, Barrero C, Coker-Gurkan A, Arisan ED, Merali S, Palavan-Unsal N. "SILAC-based mass spectrometry analysis reveals that epibrassinolide induces apoptosis via activating endoplasmic reticulum stress in prostate cancer cells" PLOS One, 10(9):e0135788. doi: 10.1371/journal.pone.0135788., 2015
2. Obakan-Yerlikaya P, Arisan ED, Coker-Gurkan A, Kaan Adacan Utku Ozbey Berna Somuncu Didem Baran Narcin Palavan-Unsal. Calreticulin is a fine tuning molecule in epibrassinolide-induced apoptosis through activating endoplasmic reticulum stress in colon cancer cells. Molecular Carcinogenesis. 2017 Jan 23. doi: 10.1002/mc.22616.
3. De Rybel, B., et al., Chemical inhibition of a subset of Arabidopsis thaliana GSK3-like kinases activates brassinosteroid signaling. Chem Biol, 2009. 16(6): p. 594-604.