



Seminars in Biotechnology BTEC 592 & BTEC 692

“Elucidating The Role Of Rac1 Signalling On The Regulation Of Autophagy During Mammary Gland Development”

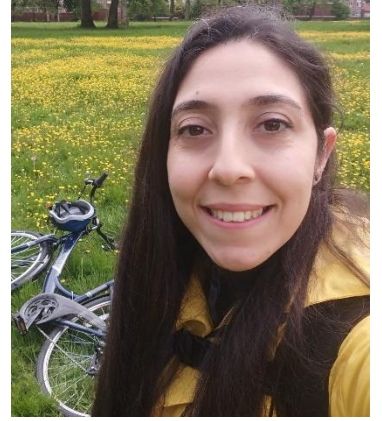
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13.30

Microsoft Teams

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Dr. Secil Eroglu received her bachelor's degree from Ege University, Department of Biochemistry. She completed her master's thesis as an exchange research student at the Institute of Medical Biochemistry and Genetics at Copenhagen University and received her master's degree from Dokuz Eylul University Faculty of Medicine, Department of Medical Biology and Genetics. After getting her doctoral degree from Gaziantep University Faculty of Medicine, Medical Biology and Genetics, she was awarded with TUBITAK postdoctoral fellowship and worked at the University of Manchester, Division of Cancer Science as a postdoc researcher. She is currently working at GIBTU Faculty of Medicine, Department of Medical Biology as an assistant professor. Her research focuses on understanding the biology and regulation of autophagy and its impact on cancer progression.

Autophagy is an important physiological process that helps the clearance of the damaged organelles and long-lived structural proteins in cells, and thereby is crucial for tissue homeostasis. The mammary gland is a unique organ as most of its development takes place in postnatal life and it undergoes frequent tissue remodelling events to fulfil its development and function throughout the reproductively active life in females. The involvement of autophagy during the early phase of involution has been previously identified. However, the molecular mechanisms that control autophagy in mammary epithelial cells (MECs) are still not well understood. Rac1 belongs to the Rho family of small GTPases and functions as a key signalling node downstream of various microenvironmental signalling pathways including cell-cell and cell-ECM adhesion and growth factor signalling. Rac1b is the only known splice

variant of Rac1 and it functions as a constitutively active form of Rac1 due to its impaired intrinsic GTPase activity and inability to interact with negative regulators of Rac1 function. Given the earlier observations that indicate a mechanistic link between Rac1 signalling and autophagy and the crucial role of autophagy in the involution stage of mammary gland development, we aimed to elucidate whether and how Rac1 and Rac1b may regulate the autophagic response in MECs.