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## (FBM / DB)


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| **PROJECT TYPE** | ERC Starting Grant (FP7) |
| **TITLE** | Inflammatory signals emerging from the endoplasmic reticulum |
| **ACRONYM** | Erinflammation |
| **DURATION** | 01.01.2012 – 31.12.2016 |
| **BUDGET** | 1 498 076 € |

The endoplasmic reticulum (ER) serves many general functions, including the facilitation of protein folding and the transport of synthesized proteins, but it also has an important and more specialized role in sensing cellular stress. Stress pathways such as ER-stress trigger a group of signals that induce a transcriptional program enabling cells to survive loss of cellular homeostasis.

New findings suggest that these pathways may regulate cellular processes that promote inflammatory responses. We have previously shown that some innate immune receptors such as Toll-like receptors specifically activate a branch of the ER-stress pathway to enhance cytokine production. However, this is an emerging field of research and little is known on the specific nature of the stress-signaling pathways and their function in promoting inflammation.

The long-term goals of this proposal were to elucidate the molecular mechanisms and pathways triggered by perturbations of cellular homeostasis that promote innate immune responses. The project was also aimed at addressing the physiological role of specific stress-signaling pathways in inflammation. Three complementary research sub-projects were designed to provide a comprehensive study of molecular mechanisms and to address the physiological role of stress signaling pathways in regulating immune responses. The first sub-project identified and characterized compounds and conditions that activate specific stress-signaling pathways. The second sub-project focused on the biochemical characterization of signaling pathways emerging from the perturbation of cellular homeostasis. The third sub-project was aimed at investigating mechanisms by which these signaling pathways affected innate immune and inflammatory responses.

This study identified new pathways and mechanisms engaged upon perturbations of cellular homeostasis. It also provided evidences implicating these pathways in physiological responses as well as diseases including infections, aging and cancer.