Dr S Macip - New modulators, effectors and functions of the p53 tumour suppressor pathway in cancer

p53 is a critical regulator of the cell cycle and the “guardian of the genome”. The majority of human tumours show loss of p53 function, which demonstrates its importance in tumour suppression. In response to cellular stress, levels of p53 increase and trigger cell cycle arrest, senescence or apoptosis to prevent the emergence of transformed cells. The factors determining these cell fate decisions are not well understood. We have shown that the cellular responses to p53 expression can be influenced by a wide range of factors, including reactive oxygen species (ROS) and pro-survival signals induced by p53 itself. Our aim is to identify new pathways that contribute to p53 functions to understand the antineoplastic mechanisms of the cell, with the ultimate goal of designing better therapies.

We study the p53 pathway at several levels. We are characterizing novel p53 target genes that will help understand the cellular effects of p53. This leads to the identification of new p53 functions beyond its classic antineoplastic activity. For instance, we are studying the role of p53 in the defence against viral infection. We also investigate how cell fate decisions after p53 activation can be modulated, with special interest in the mechanisms involved in senescence and apoptosis. Finally, we are exploring how oxygen tension, hypoxia and reactive oxygen species and the retinoic acid pathway can contribute or interfere with the p53 response.

Prospective PhD students will be trained in a variety of cutting edge techniques including cell culture, protein and RNA isolation, real time PCR, flow cytometry and RNAi technology. Applications for PhD studentships are welcome from candidates who hold or expect to hold a first or upper second class degree.

Informal enquiries, including a CV, should be sent to Dr. Salvador Macip by email at sm460@le.ac.uk. Further information is available at http://www.le.ac.uk/biochem/staff/sm460/sm460.html

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