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HYPERGLYCAEMIA INCREASES P300 ACETYL-TRANSFERASE ACTIVITY IN COLON CANCER CELLS

Custodia García Jiménez[1], Ana Chocarro Calvo[1], Jose Manuel García Martínez[1], María Gutiérrez Salmerón[1], Maria Carmen Fiuza[2] and **Antonio De la Vieja[3]**

[1]King Juan Carlos University, Alcorcon, Spain
[2]Alcorcon Foundation University Hospital, Spain
[3]Carlos III Health Institute, Spain

Failure to maintain an adequate acetylation balance of chromatin, transcription factors and enzymes by p300/CBP is widely involved in human disease from cancer to neurodegeneration. How physiological or pathological conditions or metabolites alter the acetylation balance to drive broad changes in cancer cell gene expression, imposing epigenetic marks has been elusive. Here we show the molecular mechanisms that underlie induction of p300 activity under hyperglycaemia, which results in general chromatin opening to allow high rates of proliferation in gastrointestinal cancer cells. We show the mechanistic details *in vitro* to discuss new molecular targets and then we compare human samples from diabetic and non diabetic patients.

Biography

Custodia García Jiménez graduated in Biology from University of Murcia and from Autonomous University of Barcelona (Madrid) as Doctor in Biology. She studied (Predoctoral) at Harvard Medical School- Massachusetts General Hospital, Boston (USA) and (Postdoc) at European Molecular Biology Laboratory, Heidelberg, (Germany) and Murdoch Children's Research Institute, UK with national (Spanish Ministry of Science & Education) and international fellowships (Marie-Curie Fellowships-EC). In 2002, she joined King Juan Carlos University (URJC) where she teaches Physiology and leads her research focused on molecular links between diabetes and cancer. She has collaborations with several international (UK and Japan) and national institutions and hospitals attached to URJC.

custodia.garcia@urjc.es