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CIC biomaGUNE
CENTER FOR COOPERATIVE RESEARCH IN BIOMATERIALS

seminar

Wednesday, 4th March, 12.00 pm, Seminar Room

Host: Dr. Jesús Ruiz-Cabello

Looking for targets to prevent non-alcoholic fatty liver disease progression

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The epidemic of obesity has lead to a higher incidence of non-alcoholic fatty liver disease (NAFLD) and increased risk of suffering from hepatocellular carcinoma (HCC). Inflammation and fibrosis are no longer the only factors associated with liver cancer, fatty liver also represents an increased risk factor for HCC. A hallmark of the pathogenesis of NAFLD is the metabolic dysregulation that will induce accumulation of lipids. The lipotoxicity derived from certain lipids will induce insulin resistance, activation of ER stress and/or inhibition of autophagy, which will complicate the metabolic status and induce the progression of the disease. Here, through the usage of knockout mice and different approaches to manipulate in vivo liver lipid metabolism we show, among others, the role of E2F1 and E2F2 transcription factors in NAFLD associated HCC development. We have found that E2F2 is a master regulator of metabolism in obesity related liver disease progression. It regulates the PPAR axis, involved in the homeostasis of liver lipid metabolism. Deficiency of E2F2 prevents the lipid storage required for NAFLD development and progression to HCC, which points out the value of E2F2 as a potential therapeutic target.