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SEX HORMONE-BINDING GLOBULIN AS A NEW THERAPEUTIC TARGET AGAINST OBESITY AND NAFLD DEVELOPMENT

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Low plasma SHBG levels are present in obese subjects of all ages and in overweight individuals, these are biomarkers for the metabolic syndrome and predict type 2 diabetes and cardiovascular disease risk. There are no *in vivo* models to study SHBG expression and regulation during obesity development, since rodents unlike humans do not express the SHBG gene in their livers. In the present study, we have developed a unique mouse model that expresses the human SHBG and it develops obesity, by crossing the human SHBG transgenic mice with the C57BL/ksJ-db/db mice. The characterization of this SHBG-C57BL/ksJ-db/db mouse model have allowed us to determine: (1) the molecular mechanisms and transcription factors causing the SHBG downregulation during obesity development, which involved changes in liver HNF-4α and PPARγ mRNA and protein levels. These results were further confirmed in human liver biopsies; (2) that SHBG overexpression protects against body weight increase, adiposity and NAFLD development. The SHBG overexpression in C57BL/ksJ-db/db mice significantly reduced adipose tissue and liver weight. Overall, we have created the first mouse model that resembles what occurs in human obese subjects in terms of SHBG expression and regulation. More importantly, our results point out to SHBG as a protective factor against adiposity and NAFLD. Therefore, SHBG could be a new therapeutic target whereby increased expression may reduce obesity and NAFLD.

Biography

David Martinez Selva received Bachelor's Degree in Biology in 1996 at the University of Barcelona. He obtained my PhD in Biochemistry and Molecular Biology at the Autonomous University of Barcelona in 2001. After his PhD he has accepted as a post-doctoral position for 7 years in Professor Hammond laboratory first at the LRCC in the UWO, Canada and later on at the Child and Family Research Institute (CFRI) in the University of British Columbia (UBC), Vancouver, British Columbia, Canada where he worked on the molecular mechanisms regulating hepatic SHBG production in several human SHBG transgenic mice and HepG2 cells. Eight years ago he obtained a principal investigator position through the Miguel Servet Program in the Diabetes and Metabolism Department at the Vall d'Hebron Research Institute in Barcelona, Spain.

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