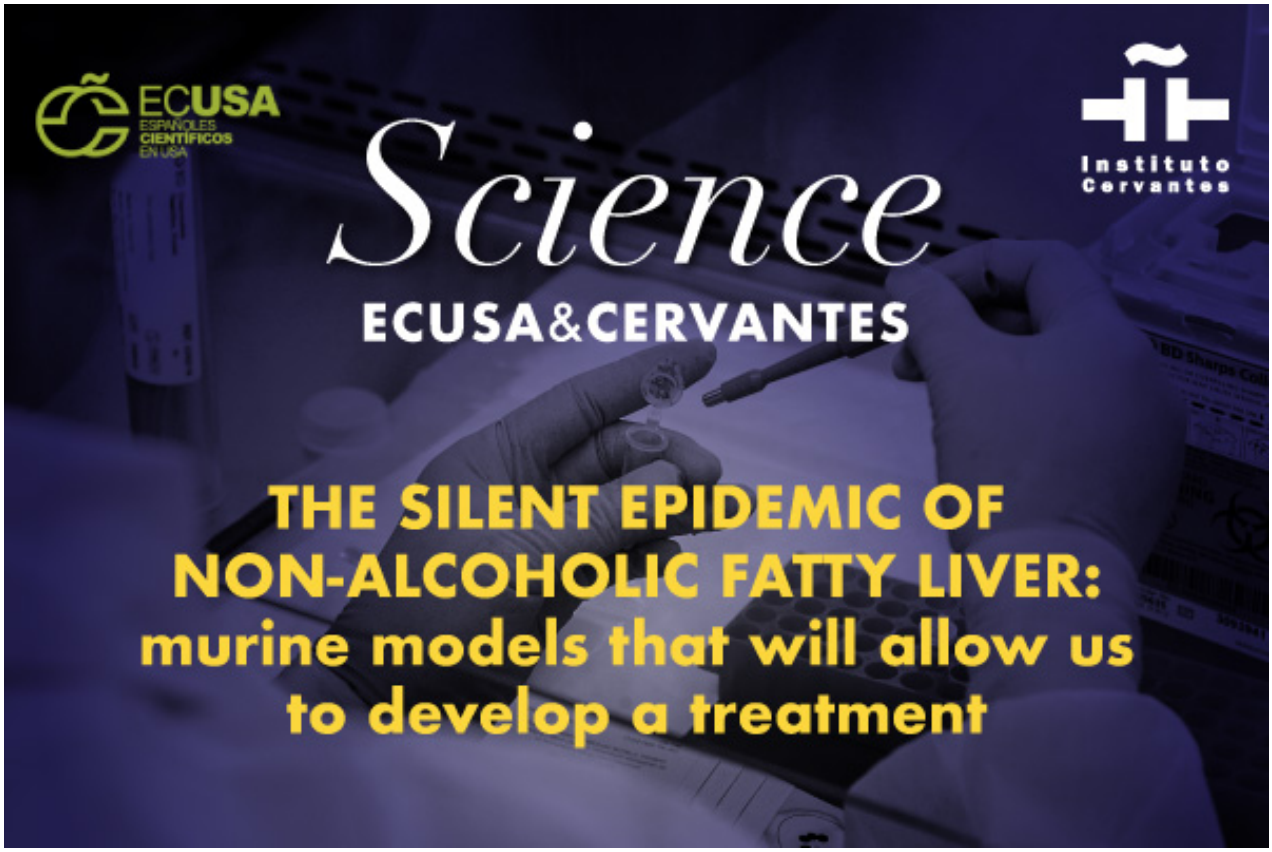
Instituto Cervantes

Science



Science ECUSA & Cervantes

The silent epidemic of non-alcoholic fatty liver: murine models that will allow us to develop a treatment

Wednesday,
June 16,
12:30 pm CT

Free and open tothepublic
The event will be held in Spanish

Registration Link

Dr. Córdoba will be interviewed by scientific journalist Verónica Fuentes followed by a Q & A. Freeandopen tothepublic

The event will be held in Spanish
If you are interested in learning Spanish, **please visit our website,** you will find a wide range of classes for all levels. **You can also visit our library** with all the necessary resources to learn Spanish

If you have booked a seat to a virtual event, you will receive a link to the virtual event space.

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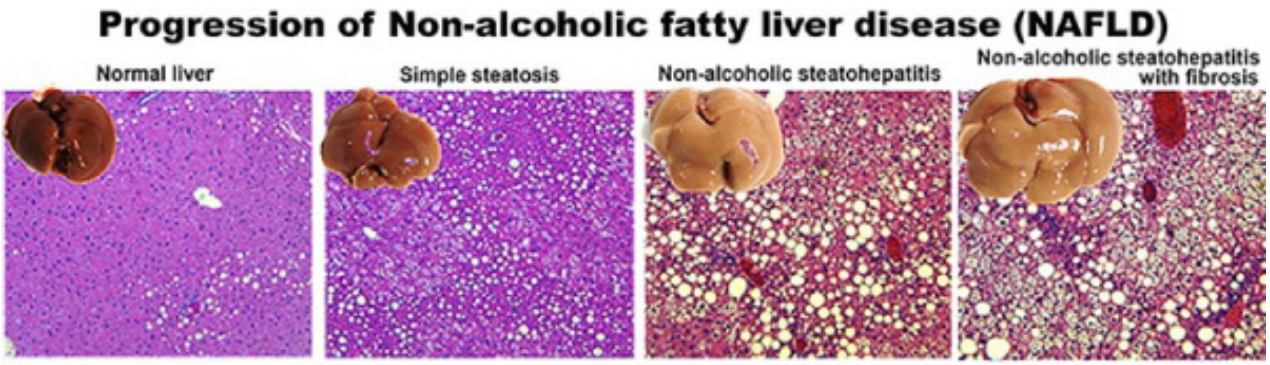
Co-organized by the Instituto Cervantes of Chicago and ECUSA Midwest

This lecture series will take place entirely online, with a new speaker on the second or third Wednesday of the month. **You can access all past lectures at this link.**

Non-alcoholic fatty liver disease (NAFLD) is highly prevalent. To date, up to 25% of the general population has NAFLD. This disease is characterized by the accumulation of lipids in hepatocytes, and with the onset of inflammation and fibrosis in the liver that are strongly associated with the increase in cardiovascular disease and diabetes. NAFLD also increases mortality and is becoming the leading cause of liver transplantation because it can progress to cirrhosis and liver cancer. Currently, it is a challenge to detect early cases of NAFLD, and we do not have a specific treatment to reduce or reverse NAFLD yet. However, due to the efforts of the scientific community in the last decades, several mouse models have been developed that allow us to reproduce the progression of NAFLD as occurs in humans. These tools have increased significantly the study of the molecular mechanisms that promote NAFLD and contribute to the development of potential therapies for NAFLD. The great advantage of the mouse models is that we can alter the expression of genes in different liver cells (hepatocytes, macrophages, hepatic stellate cells, and endothelial cells) from birth or in adults (inducible models). In addition, we can induce NAFLD with genetic models that have a predisposition to develop it, or with special diets. In my laboratory we are exploring the role of a nuclear receptor in hepatocytes, and how it contributes to the development of NAFLD. Using mouse models, we have shown that this receptor decreases the efficacy of a drug that has been tested in humans to reverse fatty liver disease. Our results are leading us to study in detail how the metabolism of specific amino acids and their related molecules are regulated in hepatocytes. This could help us to learn more about the progression of NAFLD and develop research projects that identify possible therapies.

Dr. Córdoba-Chacón earned a Master of Science in Biochemistry (2006), and another in Cellular and Molecular Biotechnology, and Genetics (2007) and a Ph.D. in the Biomedicine program of the University of Córdoba in 2011. In 2011 he moved to the University of Illinois at Chicago as a postdoctoral research fellow to study the metabolic effects of growth hormone on the physiology of the beta-pancreatic cell and on hepatic metabolism. Since 2017, Dr. Cordoba-Chacon leads a research group in the division of endocrinology, diabetes, and metabolism of the department of medicine at the University of Illinois at Chicago, where he studies the molecular mechanisms that promote the development of non-alcoholic fatty liver disease, and control glucose production in models with lipodystrophy. His research group is funded by the National Institute of Diabetes, Digestive and Kidney Diseases (NIDDK), the University of Chicago Diabetes Research and Training Center (DRTC), and the Central Society for Clinical and Translational Research (CSCTR). Dr. Cordoba-Chacon has published numerous research articles, obtained several scholarships and post-doctoral contracts from the Alfonso Martín Escudero Foundation, the Endocrine Society, and the Chicago Biomedical Consortium, as well as the Early Investigator Award and the Eugenia Rosemberg Award from the Endocrine Society and the Young Investigator Award from the University of Illinois at Chicago Obesity and Diabetes Research Day.



La epidemia silenciosa del hígado graso: modelos murinos que nos permitirán desarrollar un tratamiento

La enfermedad de hígado graso no alcohólica tiene una enorme prevalencia en la población mundial. Se caracteriza por la acumulación de grasa en el hígado y su progresión a estados inflamatorios que se asocian con el incremento de la enfermedad cardiovascular y diabetes. También aumenta la mortalidad y se está convirtiendo en la primera causa de trasplante hepático porque puede progresar a cirrosis y cáncer hepático. Actualmente carecemos de sistemas eficientes de detección precoz de esta enfermedad o de algún tratamiento específico que permita reducir y revertir la enfermedad de hígado graso a tiempo. Sin embargo, debido al esfuerzo de la comunidad científica en las últimas décadas se han desarrollado diferentes modelos de ratón que permiten reproducir en gran medida la progresión de esta enfermedad y aumentar de forma exponencial el estudio detallado de ella. Con estos modelos podemos alterar la expresión de genes en diferentes células del hígado (hepatocitos, macrófagos, células estrelladas, y endoteliales) de manera específica desde el nacimiento o de manera inducida. Además, podemos inducir la enfermedad usando modelos genéticos con predisposición a desarrollarla o con dietas especiales. Usando algunos de estos modelos, en mi laboratorio estamos explorando el papel de un receptor nuclear en los hepatocitos, y como puede contribuir a desarrollar la enfermedad de hígado graso. De hecho, hemos mostrado que la expresión de este receptor en hepatocitos disminuye la eficacia de un medicamento que se ha ensayado en humanos para revertir la enfermedad de hígado graso. Nuestros resultados nos están guiando a estudiar más detenidamente como se regula el metabolismo de ciertos aminoácidos y sus derivados en los hepatocitos, lo que podría ayudarnos a conocer más sobre esta enfermedad y desarrollar proyectos de investigación que nos permitan identificar posibles terapias.

Dr. Córdoba se licenció en bioquímica en 2006, obtuvo un master en biotecnología celular, molecular y genética en 2007, y un doctorado en el programa de biomedicina de la Universidad de Córdoba en 2011. En 2011 inicio una estancia postdoctoral en la Universidad de Illinois en Chicago para estudiar los efectos metabólicos de la hormona del crecimiento en la fisiología de la célula beta-pancreática y en el metabolismo hepático. Desde 2017, Dr. Córdoba lidera un grupo de investigación en la división de endocrinología, diabetes, y metabolismo del departamento de medicina en la Universidad de Illinois en Chicago. Su grupo de investigación usa diferentes modelos experimentales para estudiar los mecanismos moleculares que promueven el desarrollo de la

enfermedad de hígado graso no alcohólico, y controlan la producción de glucosa en modelos con lipodistrofia. Su grupo de investigación está financiado por el National Institute of Diabetes and Digestive and Kidney diseases (NIDDK), el Diabetes Research and Training Center de la Universidad de Chicago, and la Central Society for Clinical and Translational Research. Dr. Córdoba ha publicado numerosos artículos de investigación, conseguido varias becas y contratos post doctorales de la Fundación Alfonso Martin Escudero, la sociedad de endocrinología americana, el consorcio biomédico de Chicago, así como el premio Early Investigator Award y el Eugenia Rosemberg Award de la sociedad de endocrinología americana y el Young Investigator Award del Obesity and Diabetes Research Day de la Universidad de Illinois en Chicago.

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