The group of Mitochondrial Regulation of Cell Death, in the IIBB-CSIC Institute, is currently seeking a talented and highly motivated postdoc to join in the laboratory headed by Dr. Fernández-Checa. Our research is focused on:

**Sphingolipid and mitochondrial oxidative-stress regulation of cell death. Role in Steatohepatitis.**

- **Targeting cholesterol at different levels in the mevalonate pathway protects fatty liver against ischemia-reperfusion injury.**
  
  J Hepatol. 2011 May; 54(5):1002-10

- **Specific contribution of methionine and choline in nutritional nonalcoholic steatohepatitis: impact on mitochondrial S-adenosyl-L-methionine and glutathione.**
  

- **Enhanced free cholesterol, SREBP-2 and STAR expression in human NASH.**
  
  J Hepatol. 2009 Apr; 50(4):789-96

- **Mitochondrial cholesterol in health and disease.**
  

Our group and Institute offer an interdisciplinary and dynamic environment within an international research group. An excellent laboratory infrastructure with state-of-the-art equipment is available.

**CANDIDATE PROFILE**

- Qualified candidates with a Ph.D. in natural sciences and track record of success, as indicated by first-author publications, are encouraged to apply.
- The successful applicant should be highly motivated with a prior experience in cellular and molecular biology with excellent verbal and written communication skills.
- The ideal candidate must have very proactive attitude and enthusiasm for exposure to a diversity of scientific projects. Troubleshooting abilities.
- Candidate must be capable of individual initiative and of interacting as part of team. Ability supervising students will be considered.
- The successful candidate is expected to apply for Postdoctoral Fellowships.

**CONTACT**

Interested applicants should send a cover letter (briefly describing research experience and scientific interests), a CV (with a list of publications), relevant certificates and three references (with address, phone number and e-mail) to Dr. Fernández-Checa, checa229@yahoo.com
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**Role of Mitochondrial Cholesterol in Alzheimer’s Disease**

- *Endoplasmic Reticulum Stress Mediates Amyloid β Neurotoxicity via Mitochondrial Cholesterol Trafficking.*  
  *Am J Pathol.* 2014 Jul; 184(7):2066-81
- *APP/PS1 mice overexpressing SREBP-2 exhibit combined Aβ accumulation and tau pathology underlying Alzheimer’s disease.*  
  *Hum Mol Genet.* 2013 Sep 1; 22(17):3460-76.
- *Mitochondria, cholesterol and amyloid beta peptide: a dangerous trio in Alzheimer’s disease.*  
- *Mitochondrial cholesterol loading exacerbates amyloid beta peptide-induced inflammation and neurotoxicity.*  

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**Interplay between Methionine Metabolism and Sphingolipids**

- *Specific contribution of methionine and choline in nutritional nonalcoholic steatohepatitis: impact on mitochondrial S-adenosyl-L-methionine and glutathione.*  

- *Pharmacological modulation of sphingolipids and role in disease and cancer cell biology.*  

- *Glycosphingolipids and mitochondria: role in apoptosis and disease.*  
  *Glycoconj J.* 2004; 20(9):579-88

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**Cholesterol in Hepatocellular carcinoma**


Cholesterol metabolism is deregulated in carcinogenesis, and cancer cells exhibit enhanced mitochondrial cholesterol content whose role in cell death susceptibility and cancer therapy has not been investigated. Mitochondria from rat or human hepatocellular carcinoma (HC) cells (HCC) or primary tumors from patients with HC exhibit increased mitochondrial cholesterol levels. HCC sensitivity to chemotherapy acting via mitochondria is enhanced upon cholesterol depletion by inhibition of hydroxymethylglutaryl-CoA reductase or squalene synthase (SS). Isolated mitochondria from HCC with increased cholesterol levels were resistant to mitochondrial membrane permeabilization and release of cytochrome c or Smac/DIABLO in response to various stimuli including active Bax. Similar behavior was observed in cholesterol-enriched mitochondria or liposomes and reversed by restoring mitochondrial membrane order or cholesterol extraction.

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