Candidature bourse ciblée pour un meeting

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CV au format PDF

https://cfatg.org/wp-content/uploads/2023/08/CVextended_EJVelez.pdf

Résumé des travaux présentés lors du meeting

Lettre de motivation

Dear Organization/Scientific Committee of CFATG11,

I am writing to express my interest in the Travel Grant opportunity offered by CFATG for doctoral and post-doctoral students to take part in the coming CFATG11 conference. I am a Marie-Curie Postdoctoral Researcher in the lab of Dr. Iban Seiliez (INRAE NuMeA) and I am exploring the role of Chaperone-Mediated Autophagy (CMA) in glucose-metabolism disorders. I am excited about the prospect of presenting my research findings to the CFATG audience.

Although this selective autophagy pathway (i.e., CMA) has been extensively characterized in mammals, only the recent findings from Dr. Seiliez lab have evidenced the existence of CMA in the medaka fish, opening the opportunity to consider this process from a new evolutionary and comparative angle. In this context, my research focuses on investigating the role and regulation of CMA in a natural model of glucose intolerance, the fish rainbow trout (RT). This species exhibits a glucose intolerance phenotype, characterized by persistent hyperglycemia and enlarged hepatosomatic index, after the ingestion of carbohydrate-rich diets, reminiscent of mammalian diabetic models. Interestingly, RT counts with two paralogs of the CMA-limiting factor lysosomal-associated membrane protein 2A (Lamp2a), in contrast to the single copy present in mammals. Taken together, RT is an interesting model to (I) study whether the existence of CMA in fish is exclusive to medaka or if it is extensible in other species, and to (II) explore the regulation and role of CMA during hyperglycemic stress.

In brief, we have found that RT exhibits CMA activity, which is upregulated upon the exposure to high concentrations of glucose. Mechanistically, this glucose-dependent regulation of CMA involves the formation of oxidative stress at the mitochondria and the activation of the NRF2 antioxidant pathway. In addition, we evidenced that CMA-impairment through the silencing of one of the two RT Lamp2as compromised cell viability during hyperglycemic conditions. Together, this research highlights the role and regulation of CMA during hyperglycemic stress and points out to targeting CMA as a novel pharmacological strategy to alleviate glucose-related metabolic disorders.

The CFATG11 meeting is an invaluable platform for sharing our most recent results, exchanging

insights, and receiving constructive feedback. By covering the registration costs, the Travel Grant would enable me to reallocate funds to attend other relevant conferences and extend the dissemination of the results. I commit myself in advance to acknowledge the support of CFATG if I have the opportunity to present my data and obtain the Travel Grant. Enclosed with this letter, please find my extended CV and a summary of the work entitled “Chaperone-Mediated Autophagy Safeguards Against Hyperglycemic Stress”.

Thank you for your time considering my application. I look forward to participating in CFATG11.

Sincerely,

Emilio J. VELEZ

Présentation orale

Oui

Présentation par affiche

Oui