Candidature bourse ciblée pour un meeting

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CV au format PDF

https://cfatg.org/wp-content/uploads/2023/09/CV-Allioux.pdf

Résumé des travaux présentés lors du meeting

A non-canonical role of LC3B in HEV-infected polarized hepatocytes

Hepatitis E virus (HEV) is a common cause of acute hepatitis worldwide. Most infections are asymptomatic but some genotypes can lead to chronic infections in immunocompromised patients. HEV is a small non-enveloped RNA virus. However, it is released from infected cells in a lipid-associated form. The different stages of HEV cycle are not fully known. In polarized hepatocytes, we found that the ORF2 capsid protein colocalized with LC3B, an essential protein of the autophagosome formation. (Macro)autophagy is a conserved pathway in the host cell defense against pathogens. Autophagosomes are double membrane vesicles that trap cytosolic content and deliver it to the lysosome for degradation or participate to secretion pathways. Their formation involves LC3B anchoring in the phagophore membrane. Selective autophagy involves receptors recognized by LC3B via a LC3-interacting region (LIR) motif.
To investigate the links between autophagy and the HEV life cycle, we infected the polarized hepatocarcinoma cell line HepG2/C3A/F2 in conditions of autophagy activation or inhibition. This pharmacological modulation of the initial steps of autophagy did not influence HEV RNA production. LC3B labeling showed a basal level of LC3B nucleation in uninfected cells, that did not increase upon a 6-hour rapamycin treatment. This LC3B nucleation was inhibited by wortmannin in uninfected cells but not in HEV-infected cells at 6 hours post-infection (pi). LC3B nucleation was maintained during 7 days pi in HEV-infected cells treated during the first 6 hours pi. These results suggest that HEV induces LC3B nucleation independently of the first steps of the autophagy pathway. Using a coimmunoprecipitation assay, we detected that ORF2 interacted with the LC3B-I form in polarized hepatocytes. Further experimentation could help unravel the role of the LIR domain in ORF2-LC3B-I interaction and would advance understanding of the HEV life cycle. Our study highlights a non-classical role of LC3B in HEV-infected polarized hepatocytes.

Lettre de motivation

Dear Organization Committee,
I am currently pursuing a PhD at Toulouse Institute for Infectious and Inflammatory Diseases. I study the role of autophagy in hepatitis E virus spreading in polarized hepatocytes. I will come to CFATG11 scientific days to hear the latest discoveries in the field of autophagy and would appreciate the opportunity to present my research.
I have already presented my work at a meeting in a virology conference. CFATG scientific days will be a great opportunity to gather new insights on my project from other members of the autophagy's

research community. Obtaining this grant will greatly facilitate my coming to this event.
I hope my profile will be of your interest for a talk or a poster and for this grant. I will gladly provide any extra information you might need. I thank you for your time and consideration.

Sincerely,

Claire Allioux

Présentation orale

Oui

Présentation par affiche

Oui