APOL2/APOL3 proteins

INTRODUCTION

CONDENSED ABSTRACT

When a cell is infected with LPS or Salmonella, the non-canonical inflammasome pathway is triggered. This in turn activates caspase 4/5(in mice) and caspase 11 (in humans). In this study, we proved that APOL2 is a substrate of caspase. However it's downstream activities are unknown. APOL3 is not a substrate of caspases.

BACKGROUND

Previous study showed that APOL3 has bactericidal properties. However, this protein failed to show up in proteomics.

CENTRAL RESEARCH QUESTIONS

- Are APOL2 and APOL3 proteins cleaved by caspase?
- What is the relationship between APOL2 and caspase?

RESEARCH

METHODOLOGY

Cell culture
Bacterial Infection
LDH cell death assay
siRNA
Western Blots
Protein purification

ANALYSIS

- Full length APOL2(37kDA) and APOL3(44kDA) clearly visible in Fig1
- Cleaved bands of APOL2 in Fig1 suggesting that it is a substrate of caspase
- Full Length APOL2 not visible in Fig2
- Last 2 bands in Fig2 correspond to protein cleavage
- Smearing suggests that APOL2 aggregates with itself to form oligomers
- Alternatively, it is also possible that APOL2 aggregates with caspase
- No cleavage of APOL3

RESULTS

Fig1. HIEC cells treated with mutated Salmonella

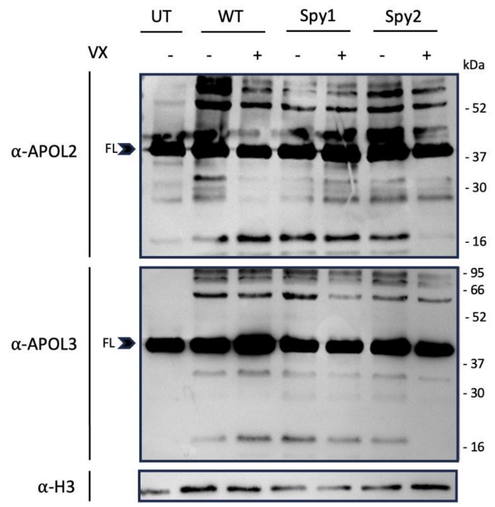
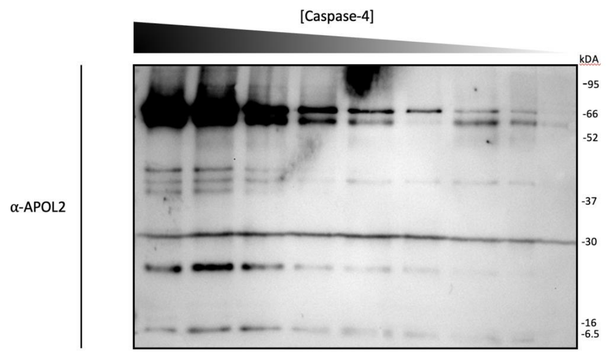


Fig2. Purified Proteins



CONCLUSION

APOL3 from previous study stated in background is very likely to have been APOL2. It is clear that APOL2 is a caspase substrate while APOL3 is not. It is also notable that APOL2 and caspase have an interesting relationship.

RECOMMENDATIONS

- Further investigation on the relationship between caspases and APOL2 on how and why it can function as both a substrate and a protein for oligomerization
- Further study on the downstream effects of the APOL2 protein and its function

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