Autophagy in Pacific oyster, *Crassostrea gigas*

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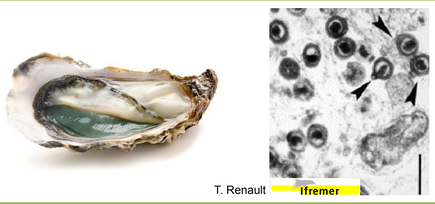
Since 2008, the oyster aquaculture industry, which mainly relies on the production of the Pacific oyster, *Crassostrea gigas*, has been affected by mass mortality outbreaks in France and Europe. Two pathogens have been associated with mass mortality outbreaks, the virus ostreid herpesvirus 1 (OsHV-1) and the bacterium *Vibrio aestuarianus*. A study was carried out in order to show that the autophagy pathway is present and functional in Pacific oysters. Moreover, interactions between oyster pathogens (OsHV-1 and *V. aestuarianus*) and autophagy were explored. Results suggested that autophagy as a conserved intracellular pathway can play a key role in innate immunity in the Pacific oyster.



1. Is autophagy flux present and functional in Pacific oyster?
2. Could autophagy protect Pacific oyster againt OsHV-1 infection?
3. Could autophagy protect Pacific oyster againt *Vibrio aestuarianus* infection?

A body of proof supporting autophagy flux in Pacific oyster

A

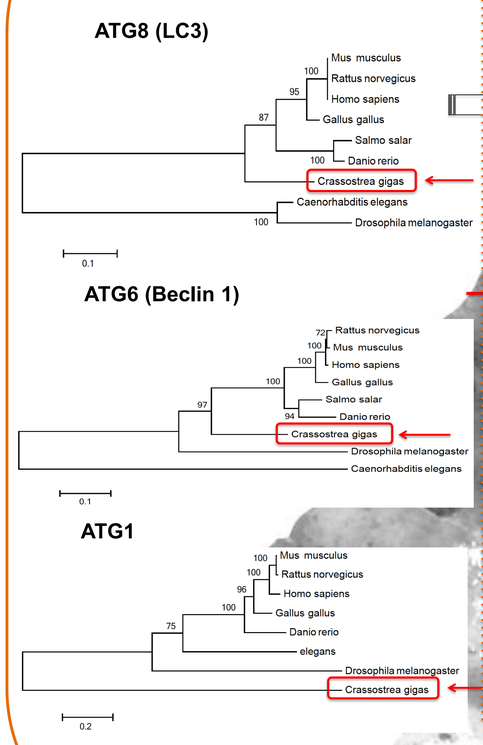


Fig A. Phylogenetic trees of ATG proteins

B

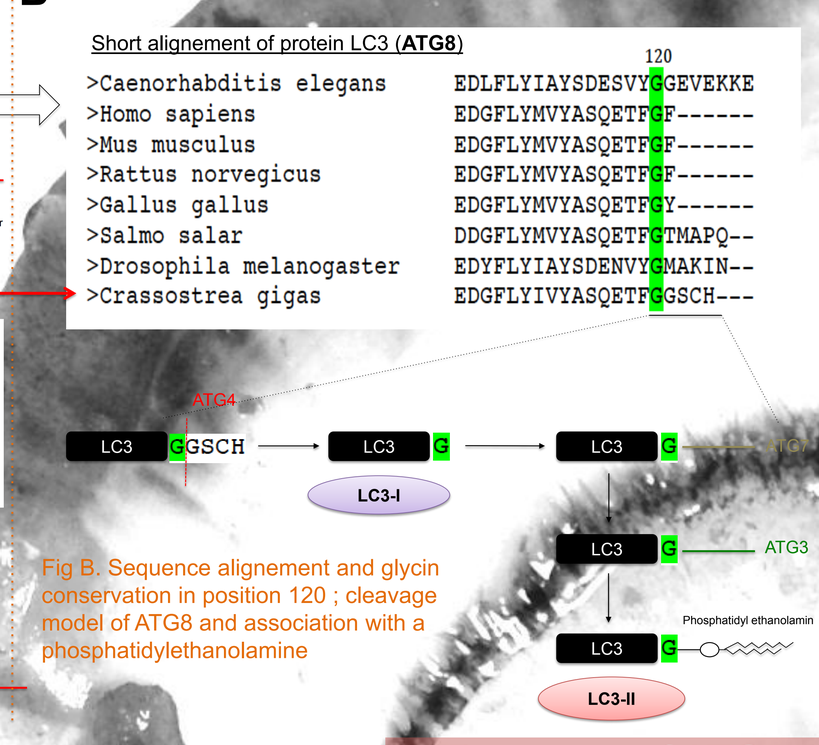


Fig B. Sequence alignement and glycin conservation in position 120 ; cleavage model of ATG8 and association with a phosphatidylethanolamine

C

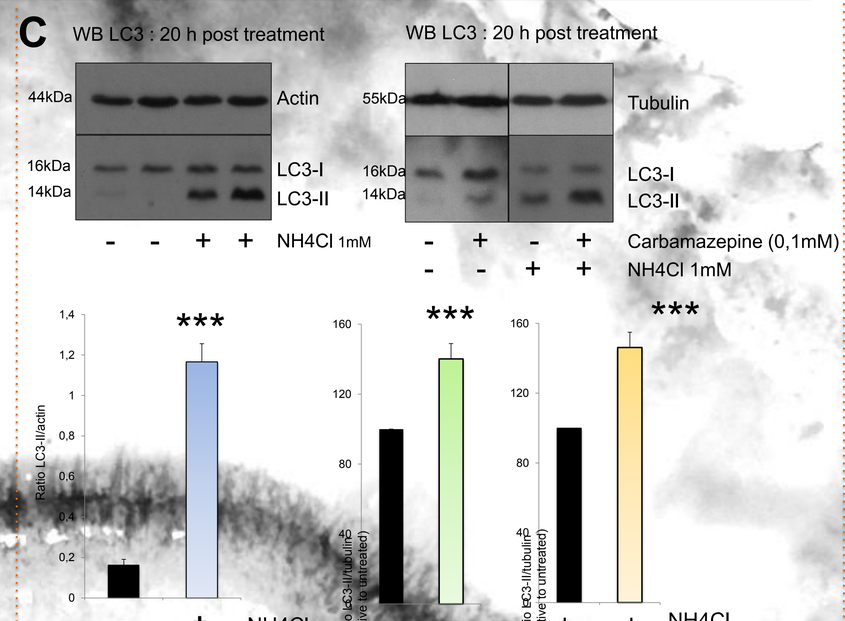


Fig C. LC3 Western blot after contact during 20h with NH4Cl, Carbamazepine and NH4Cl+Carbamazepine

D

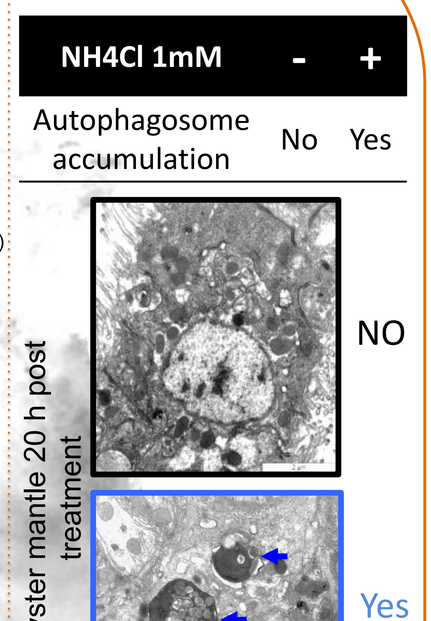


Fig.D. Transmission electron microscopy

- Presence of ATG genes, proteins and autophagosomes
- NH4Cl and Carbamazepine modulate LC3-II

Autophagy may play a protective role during OsHV-1 infection

A

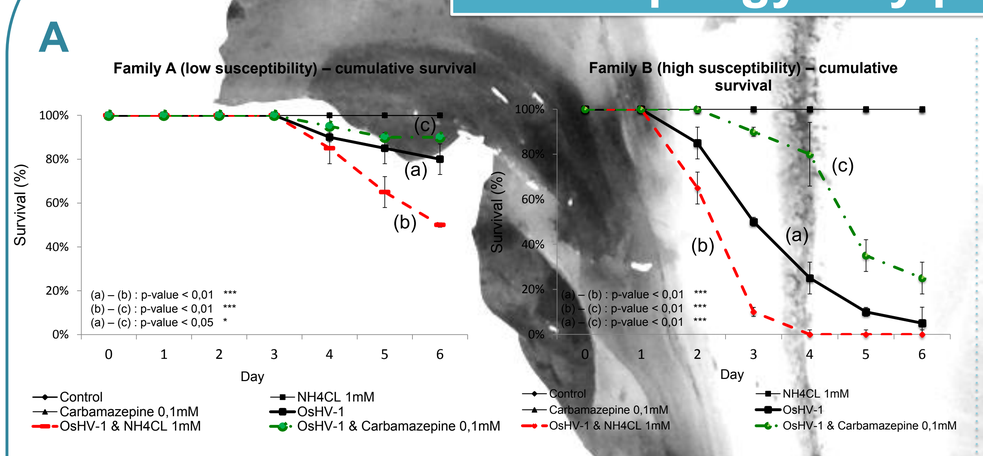


Fig A. Monitoring of oyster survival during OsHV-1 infection

B

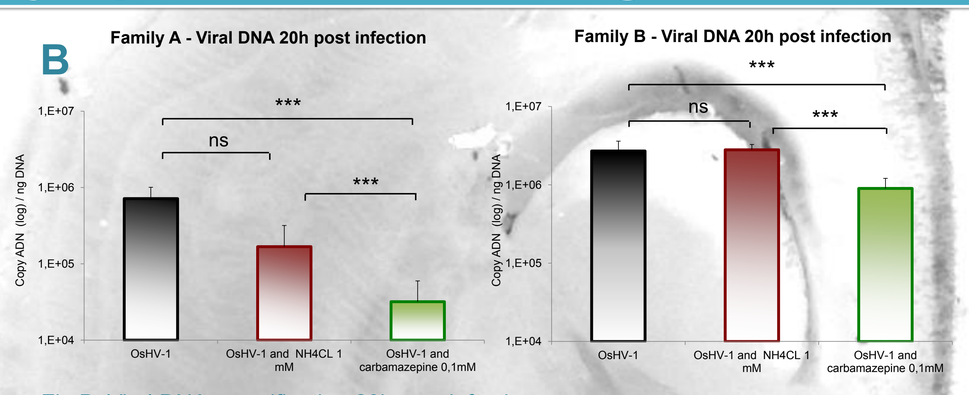


Fig B. Viral DNA quantification 20h post infection

C

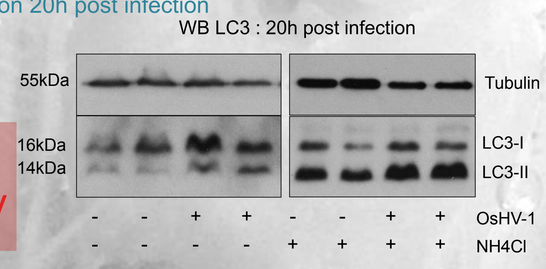


Fig C. Western blot LC3 20h post infection

D

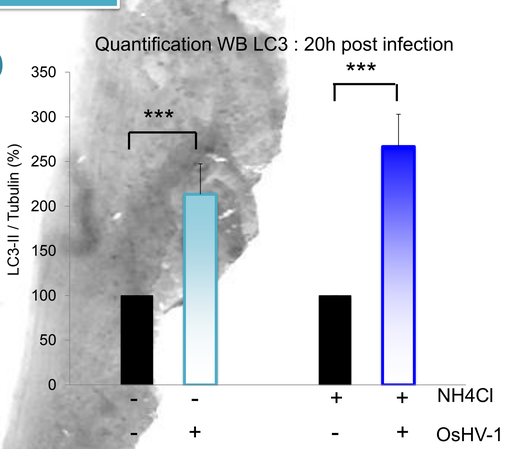


Fig D. Quantification Western blot LC3 20h post infection

- LC3-II detection increases during viral infection
- Autophagy modulation is related to OsHV-1 infection susceptibility

Autophagy may protect oysters from *Vibrio aesturianus* infection

A

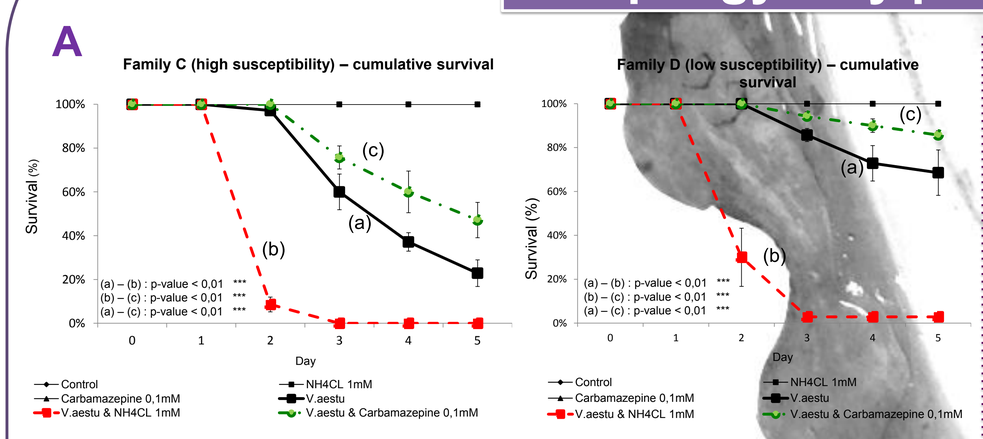


Fig A. Monitoring of oyster survival during *V. aestuarianus* infection

B

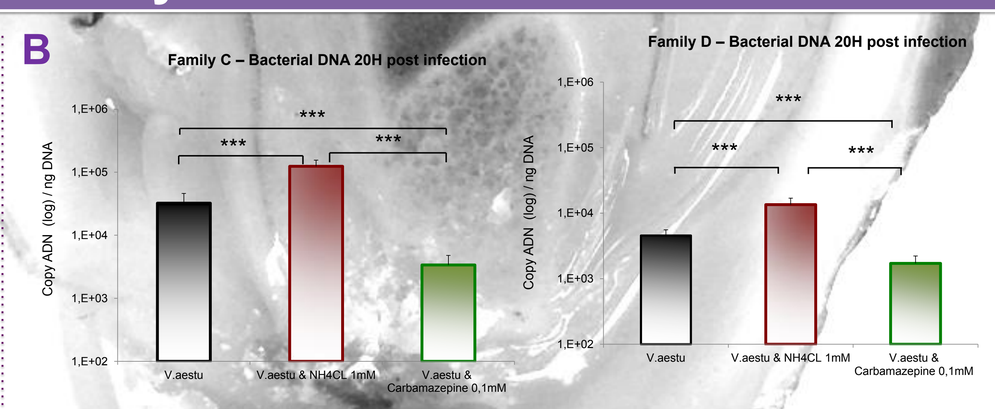


Fig B. Bacterial DNA quantification 20h post infection

C

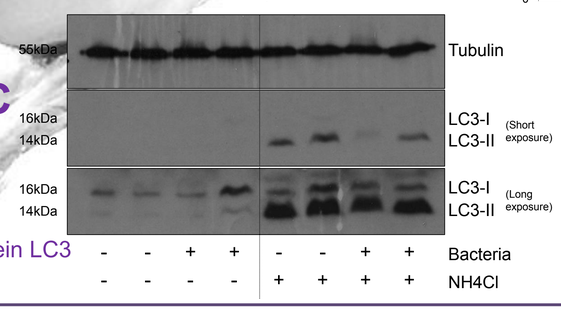


Fig C. Western blot of protein LC3 20h post infection

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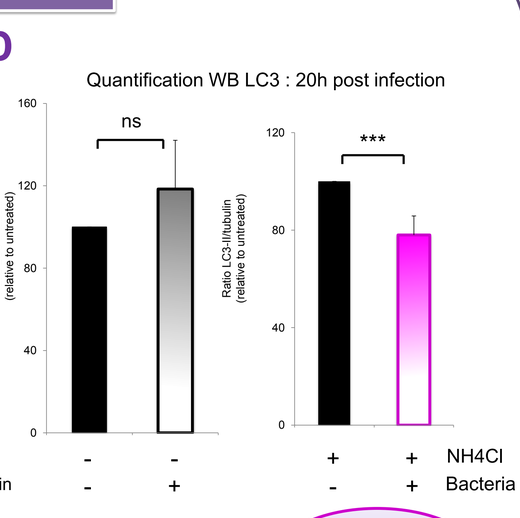

Fig D. Quantification Western blot of protein LC3 20h post infection

- LC3 II detection decreases during bacterial infection
- Autophagy modulation is related *V. aestuarianus* infection susceptibility

Conclusions

- Results suggest that autophagy is induced upon OsHV-1 infection and unmodified (inhibited with NH4Cl) upon *V. aestuarianus* infection.

	NH4Cl	OsHV-1	*V.aestuarianus*
Autophagy (LC3-II)	-	↗	ns
Autophagy (LC3-II)	+	↗	↘

- Autophagy could be interpreted as protective pathway against OsHV-1 and *V. aestuarianus* infections

Perspectives

Mortality outbreaks are often observed during spring and summer where concentrations of algae, which represent the main source of energy for oysters, are important.

✓ **It is well known that starvation is a potent stimulus of autophagy. It would be of interest to analyze if there is a correlation between food supply, autophagy and mortality outbreaks**.