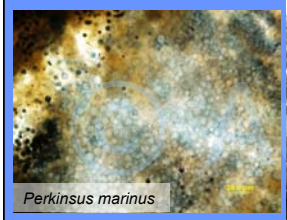
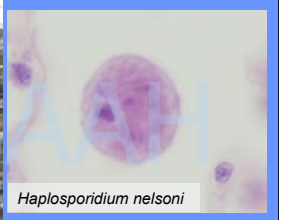
Evolutionary Ecology of *Crassostrea virginica* and its Parasites


Perkinsus marinus


Crassostrea virginica


Haplosporidium nelsoni

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Gloucester Point, Virginia USA

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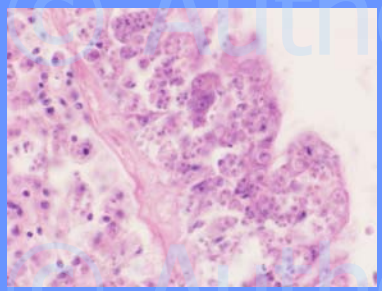
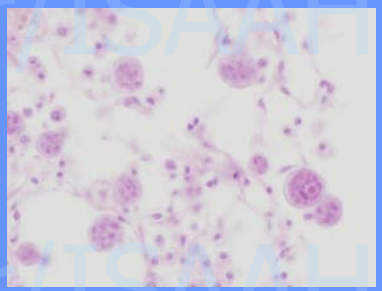
Perkinsus marinus

- Endemic species, agent of "dermo disease" in oysters
- Directly transmissible among *Crassostrea virginica*

Haplosporidium nelsoni

- Introduced pathogen (Burreson et al. 2000), agent of "MSX disease"
- Complex life cycle likely requiring intermediate host(s) for transmission





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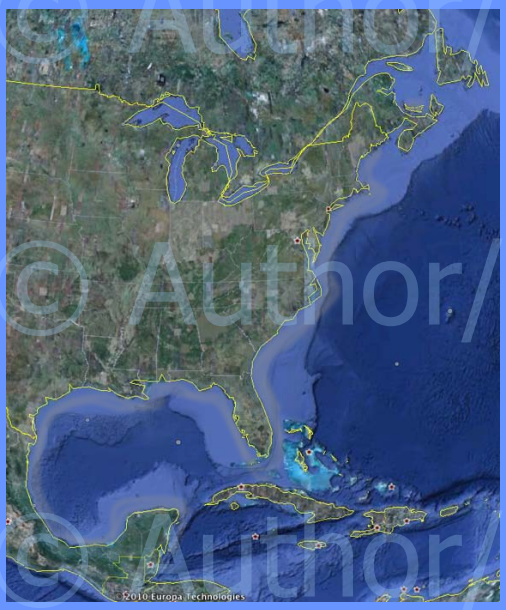
Oyster and Parasite Distribution, Present



C. virginica
P. marinus
H. nelsoni

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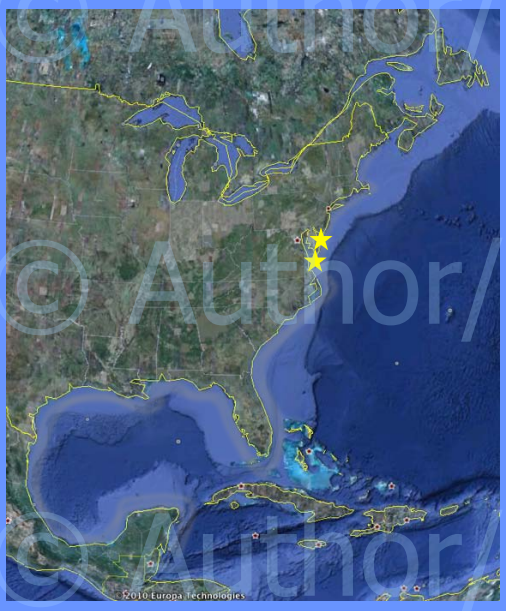
Oyster and Parasite Distribution, 1959



C. virginica
P. marinus
★ *H. nelsoni*

- MSX introduction brought sharp changes to the pre-1959 system
- Very high host mortality
- Sudden competition between two parasites, one (MSX) introduced and very virulent

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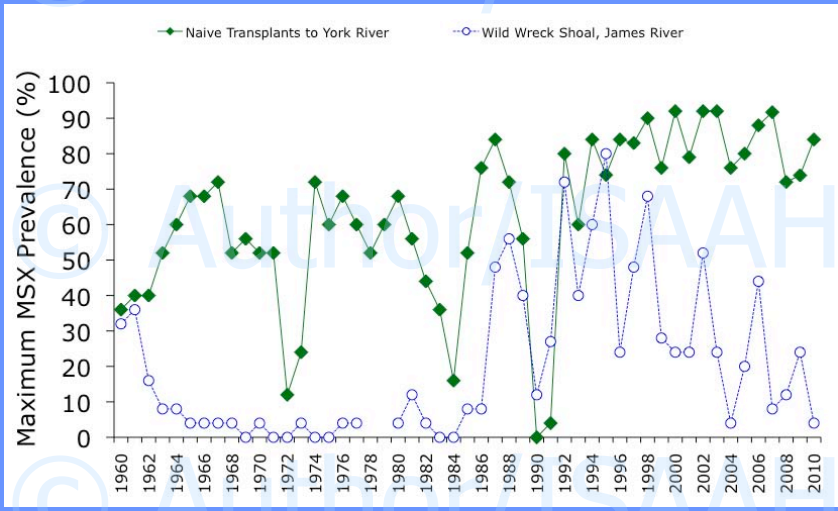
Resistance to MSX

Haplosporidium nelsoni in James River Oysters vs. Naïve Sentinels



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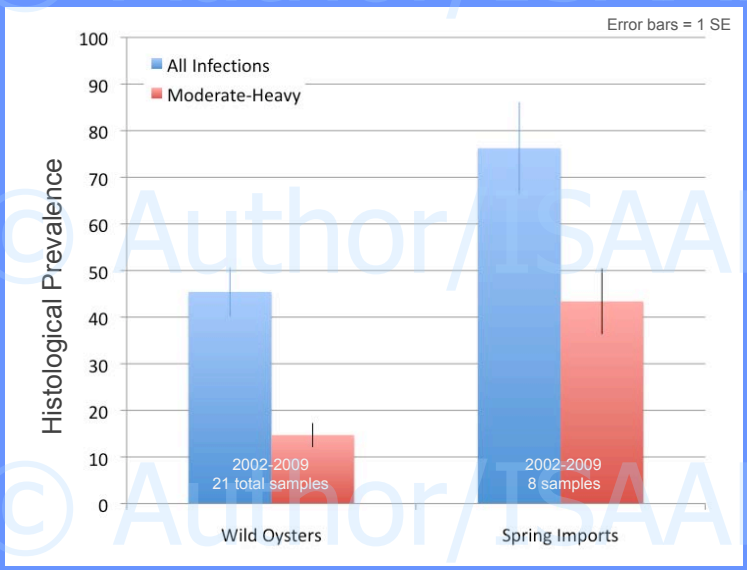
Resistance to *Perkinsus marinus*

Perkinsus marinus in Wild York/Mobjack Oysters v. Naïve Sentinels



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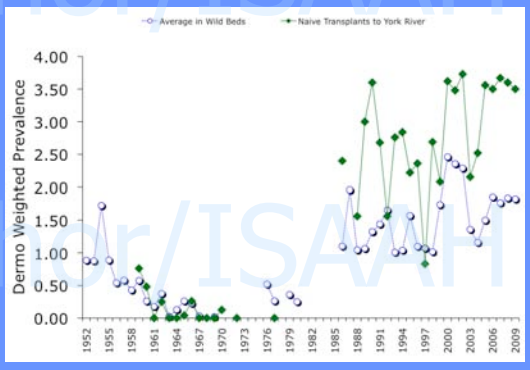
Intensification of *Perkinsus marinus*, 1980s

- *P. marinus* levels in wild oysters are much higher now than in early years
  - → Particularly *body burdens*
  - → Prevalence then and now approached 100%
- Intense disease and mortality develop much more rapidly than before the arrival of MSX
- An increase in virulence in the mid-1980s



A Question:

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Is This Not Adaptive?

- *Perkinsus marinus* is transmitted primarily through the deaths of infected oysters (Ragone Calvo et al. 2003)—before MSX this began 2-3 years after initial exposure (Andrews 1956)
- Parasite body burdens were relatively low in the 1950s (Andrews 1980), but still ensured effective transmission among oysters at a time when oysters were particularly abundant

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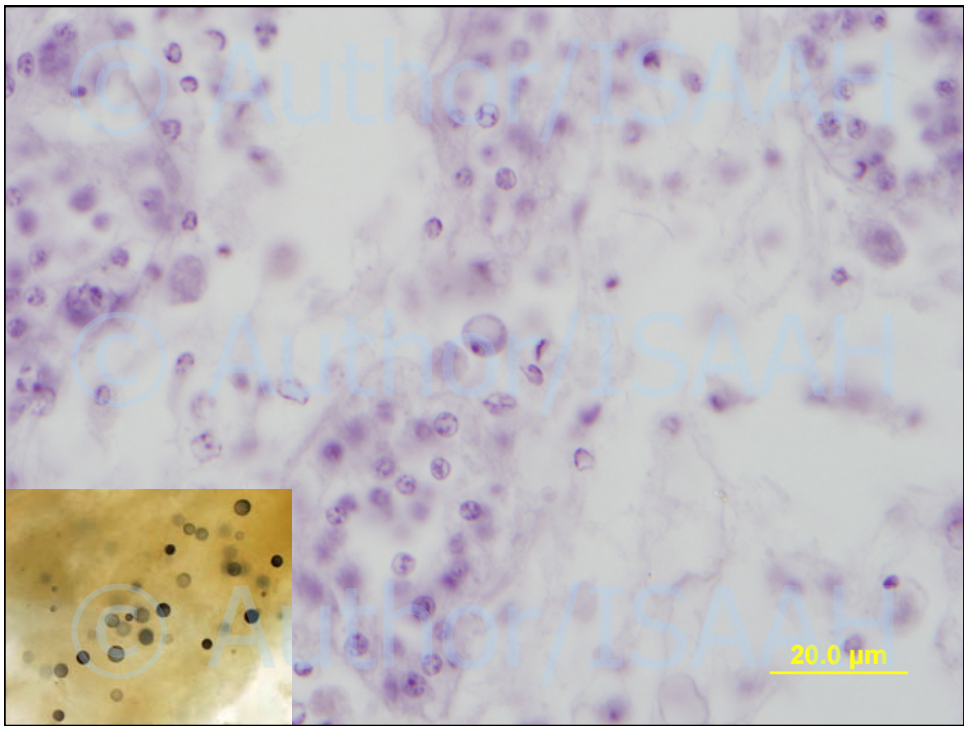
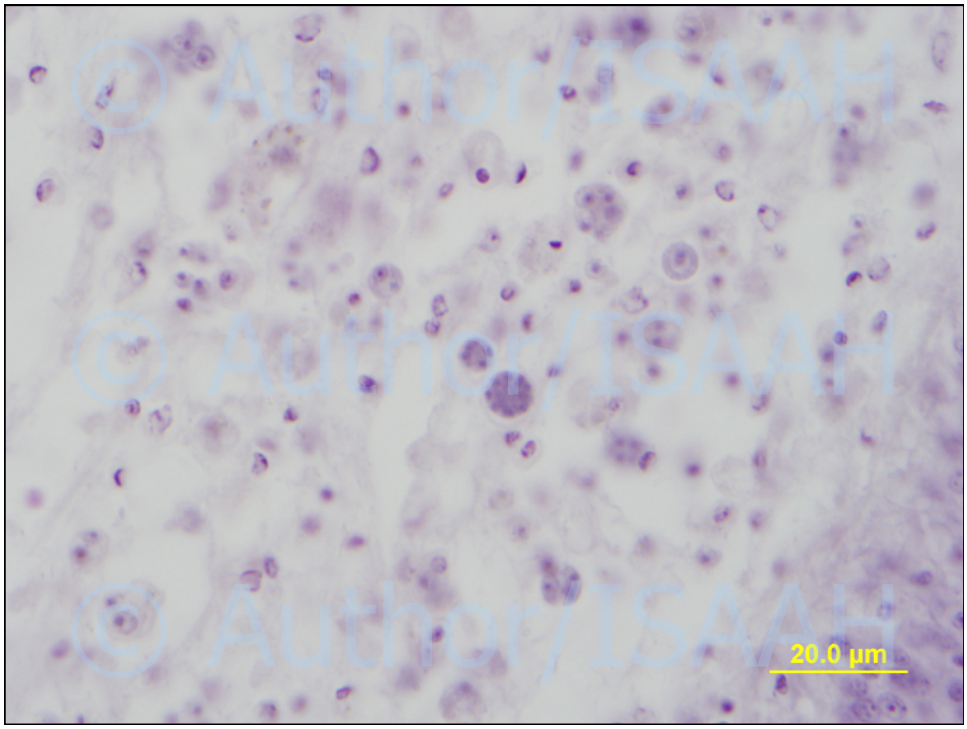
Is an increase in body burdens (higher weighted prevalences since the 1980s) and generally more rapid rate of infection development convincing evidence of a virulence increase?

Or can this still be explained by simple numerical abundance?

Not a definitive sign that *P. marinus* has fundamentally changed or evolved





Perkinsus marinus Life Cycle

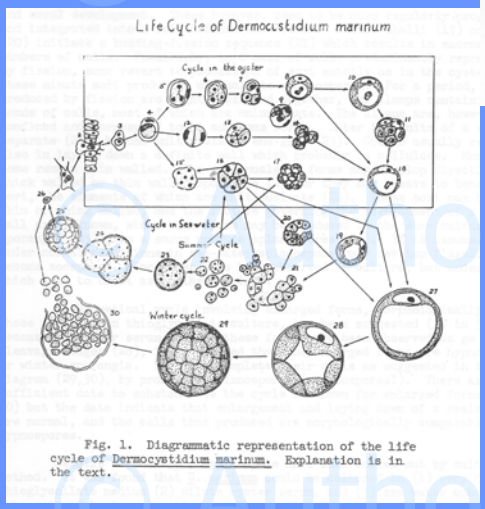


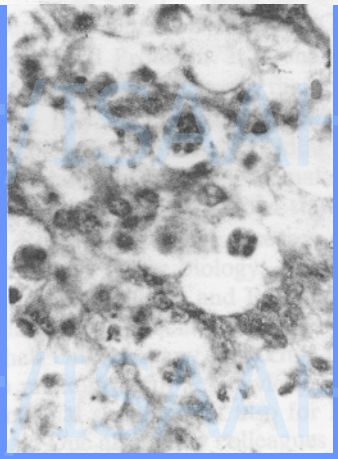
Fig. 1. Diagrammatic representation of the life cycle of Dermocystidium marinum. Explanation is in the text.

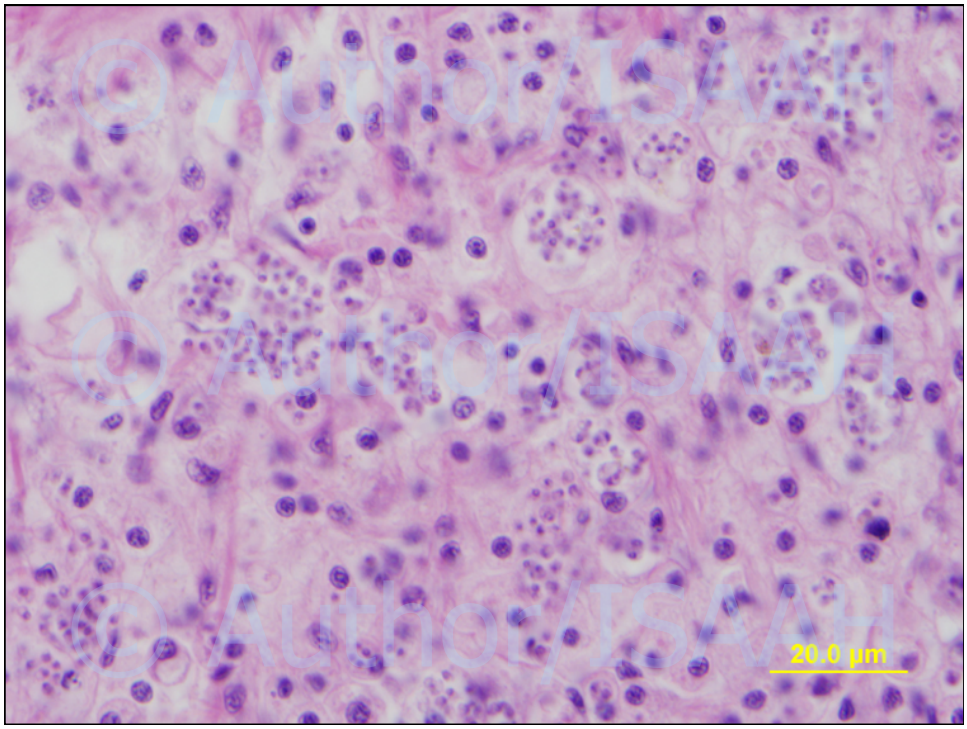
Histopathology of Infection of *Crassostrea virginica* (Gmelin) by *Dermocystidium marinum* Mackin, Owen, and Collier

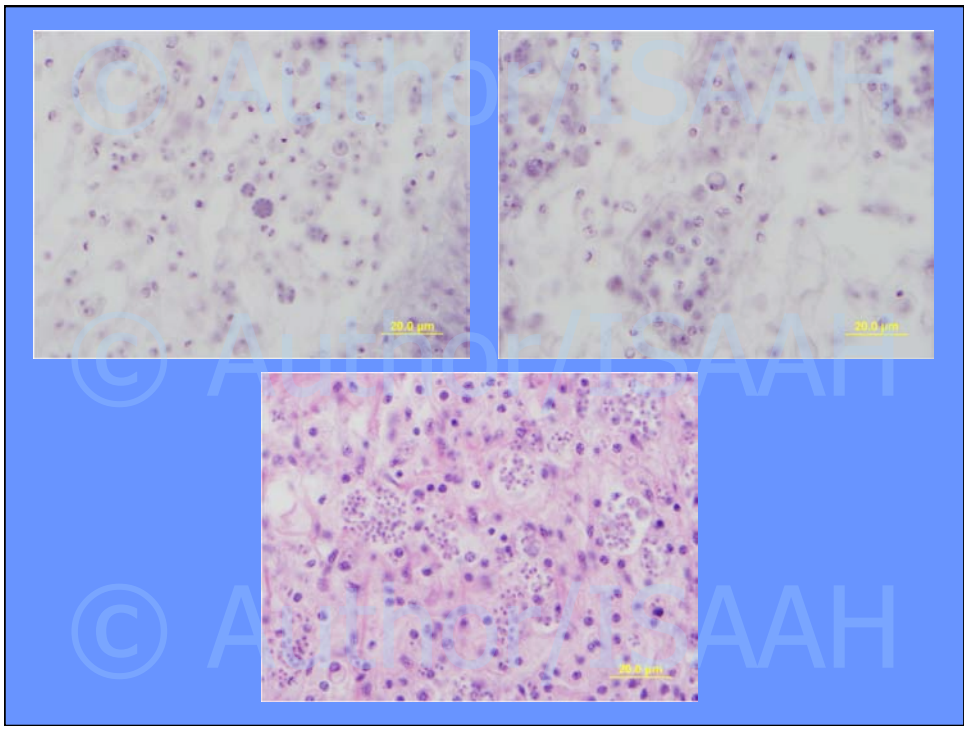
J. G. MACKIN

Department of Oceanography and Texas A.&M. Research Foundation, Agricultural and Mechanical College of Texas[1]

1951




20.0 µm

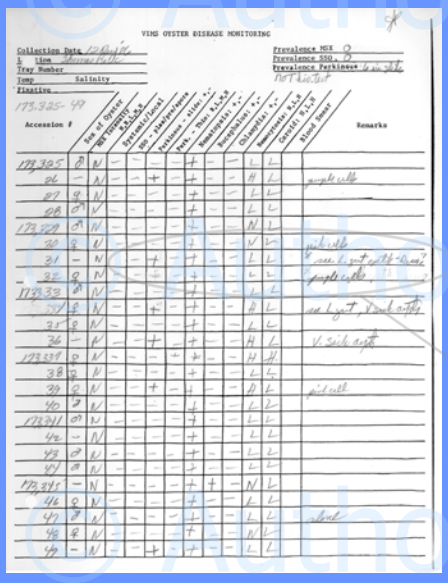


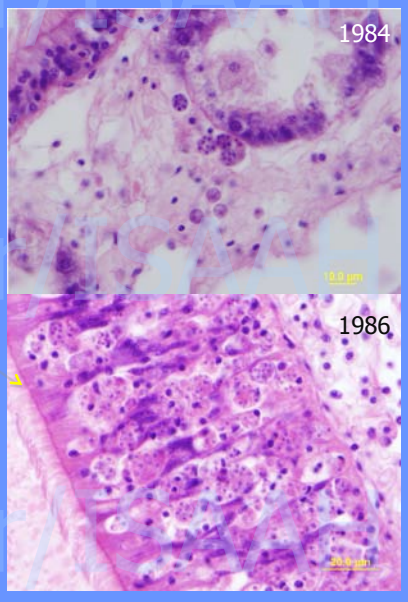
Where are the Schizonts?

- Contemporary histology suggests far more binary division than schizogony

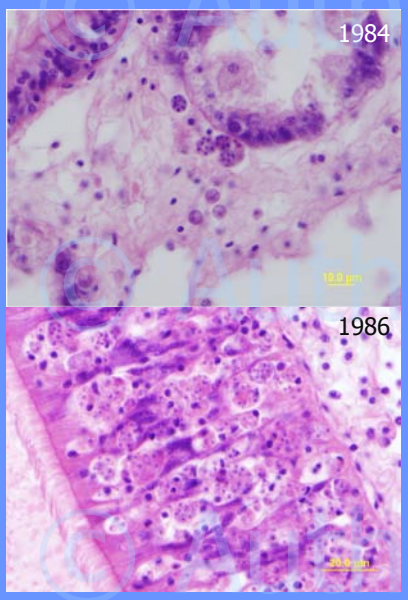
- Is binary division by cells that never grow to large size adaptive?
- Is *P. marinus* population growth rate increased by having a high proportion of cells going through binary division, rather than a small subset going through schizogony?
- Anecdotal evidence: more binary division in faster-growing cultures—suggests link between histological presentation (i.e., phenotype) and virulence (La Peyre 1996)

When Did the Change in Phenotype Occur?





Perkinsus marinus Conclusions



- Fundamental *phenotypic* change in *P. marinus* between 1985 and 1986
  - → General abandonment of schizogony in favor of binary division by small cells
- Simultaneous increase in virulence
- Hypothesize 1st that the virulence increase is associated with the change in *P. marinus* expression
- Hypothesize 2nd that this reflects selection for increased virulence induced by *H. nelsoni*, at least indirectly

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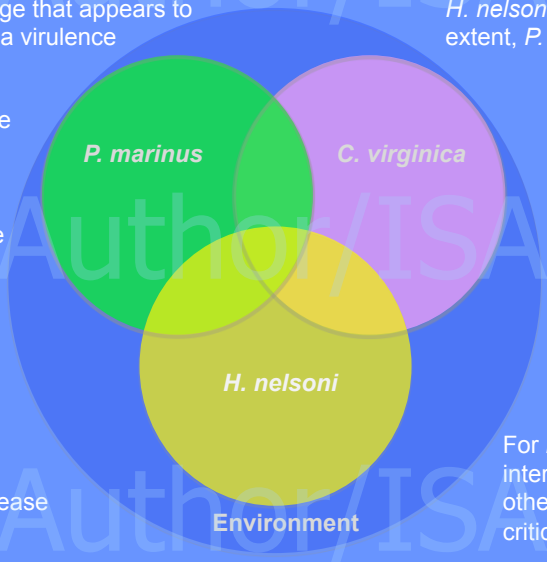
Summary

In *P. marinus,* a fundamental phenotypic change that appears to correspond with a virulence increase

What induced the change?

Can we find genetic evidence for it?

In oysters: resistance to *H. nelsoni*, and to a lesser extent, *P. marinus*



In *H. nelsoni,* no evidence for an increase or decrease in virulence

For *H. nelsoni*, interactions with other hosts may be critical

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Gene Burreson
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