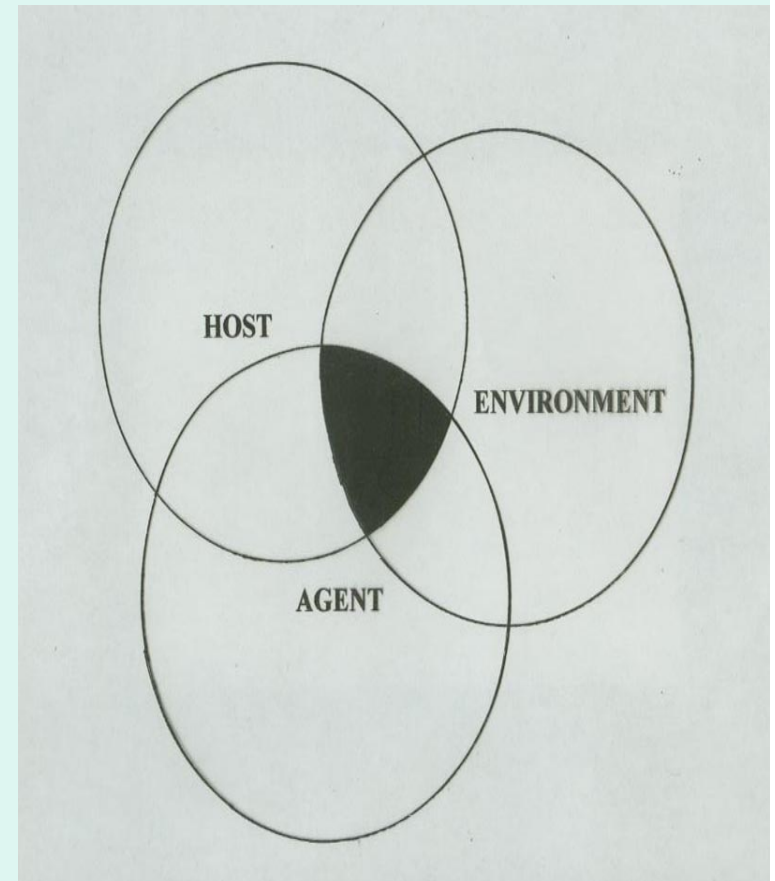


# **EFFECTS OF ENVIRONMENT AND CULTURE TECHNIQUES ON SHELLFISH DISEASE SEVERITY AND PREVALENCE**

Roxanna Smolowitz, DVM  
Director, Dept of Animal Health  
New England Aquarium  
Boston, MA

**All disease is the result of the interaction of 3 factors:**

- **Host susceptibility**
  - **Poorly conditioned animals**
  - **Genetic lack of disease resistance (susceptibility)**
  
- **Agent virulence**
  - **New agent in the environment**
  - **Mutated agent**
  - **Change in base population levels of agents (density of agent)**
  - **Direct vs. Indirect infections**
  
- **Environmental conditions**
  - **Density of host**
  - **Pollution**
  - **Decreased water quality**
  - **Decreased nutrients**
  - **Temperature/Salinity Changes**



**•The elimination or modulation of any one of these factors can decrease or eliminate the occurrence of disease.**

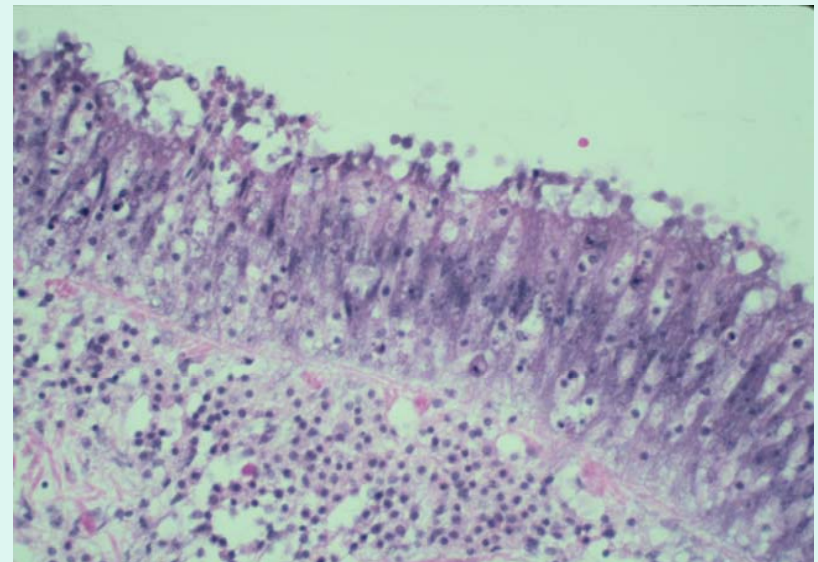
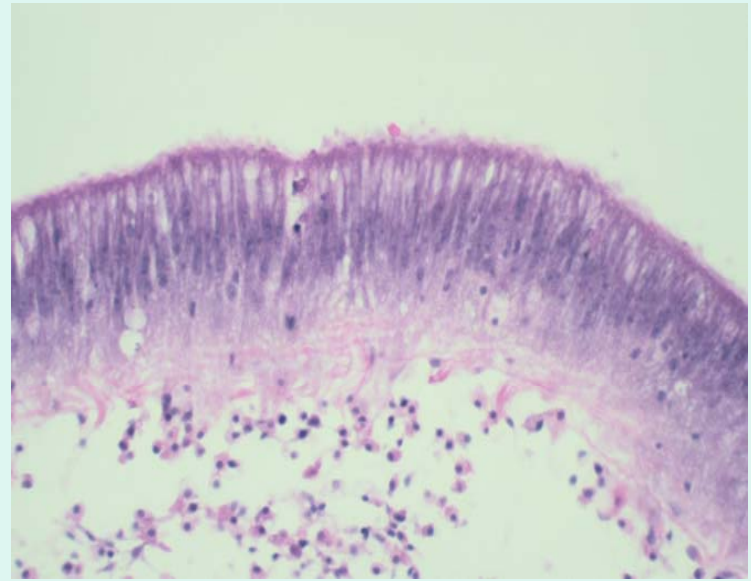
# JUVENILE OYSTER DISEASE (JOD)

- **HOST**
  - *CRASSOSTREA VIRGINICA* (EASTERN OYSTER)
- **CAUSE**
  - *Roseobacterium marinus*
  - ALPHA-PROTEOBACTERIA
- **GROSS APPEARANCE**
  - MANTLE RETRACTION
  - CUPPING OF THE LEFT VALVE
  - CONCHIOLIN DEPOSITION ON THE INNER SURFACES OF VALVES
  - REDUCED CONDITION AND DEATH
- **EPIDEMIOLOGY**
  - LATE SPRING/SUMMER IN HATCHERY/NURSERIES
  - HIGH MORTALITY FOUR TO SIX WEEKS AFTER FIRST SIGNS



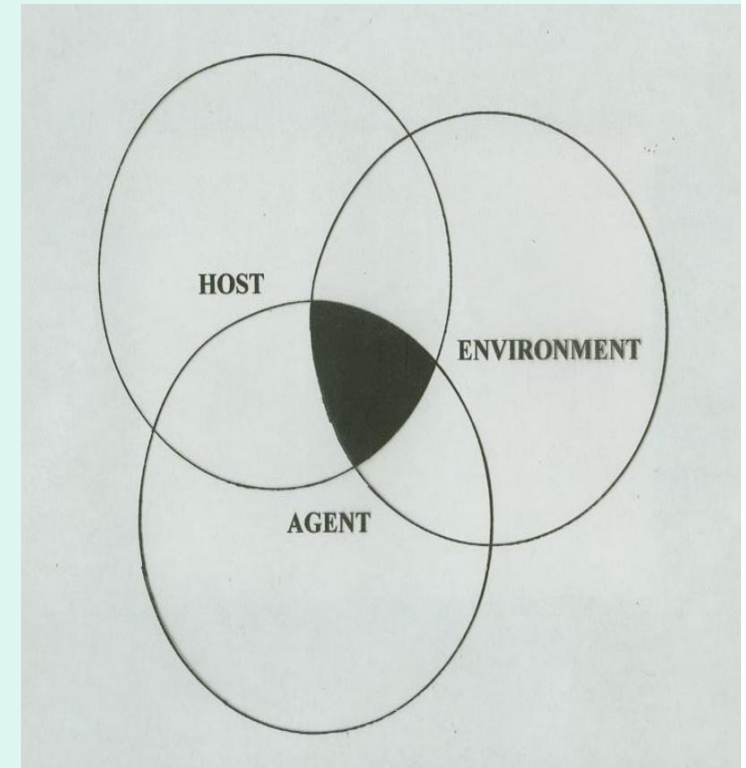
# JUVENILE OYSTER DISEASE (JOD)

- **CHARACTERISTICS OF INFECTION**
  - **JUVENILES < 25 MM IN SHELL HEIGHT**
  - **TEMP 21-22° C**
  - **SALINITIES 25 TO 32 PPT (INHIBITED AT LOWER SALINITIES)**



## JOD PROFILE

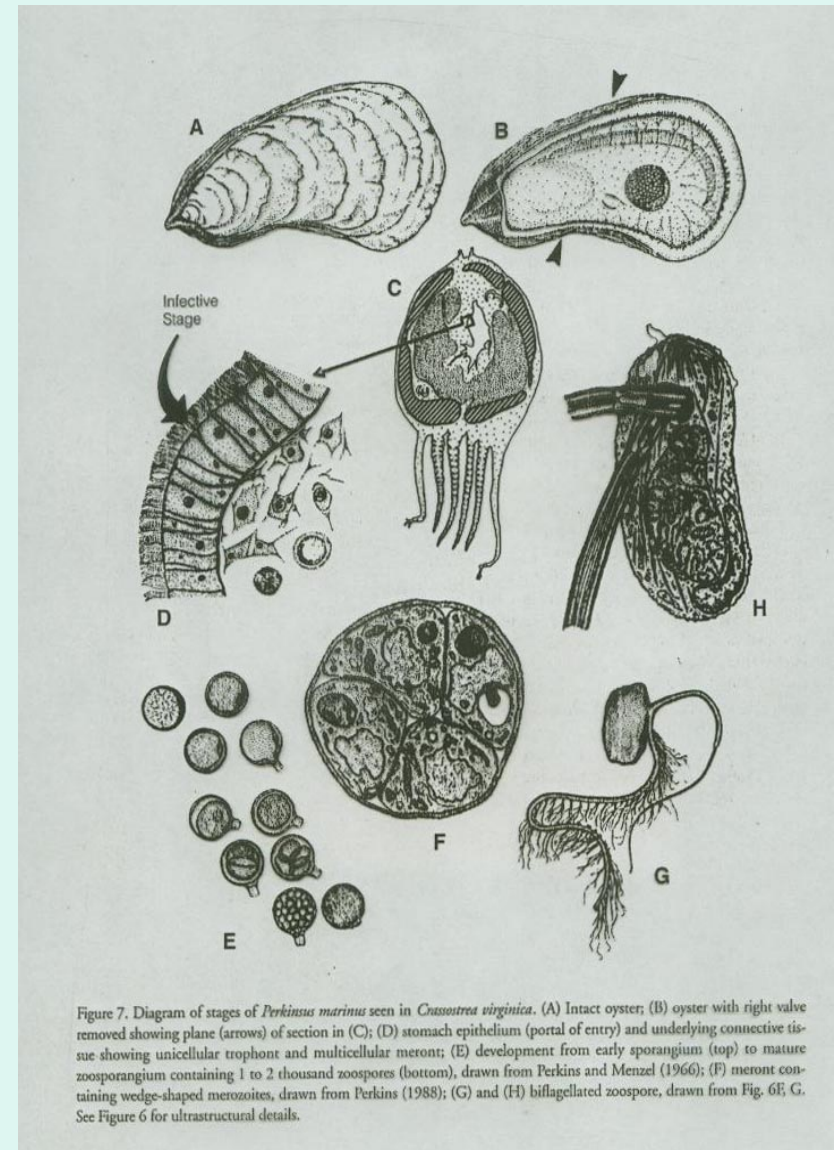
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  - **Density of host**
  - Pollution
  - Decreased water quality
  - Decreased nutrients
  - **Temperature/Salinity Changes**



- **The elimination or modulation of any one of these factors can decrease or eliminate the occurrence of disease.**

# DERMO DISEASE

- **HOST**
  - *CRASSOSTREA VIRGINICA* (EASTERN OYSTER)
- **CAUSE**
  - *PERKINSUS marinus* (DERMO)
  - FORMS IN TISSUE
    - IMMATURE MERONTS
    - SIGNET RING WITH VACUOLE CONTAINING A REFRACTILE BODY (MATURE MERONTS)
    - ROSETTES (SCHIZONT/SPORANGIUM)
  - FORMS IN SEAWATER
    - ZOOSPORANGIA CONTAINING ZOOSPORES

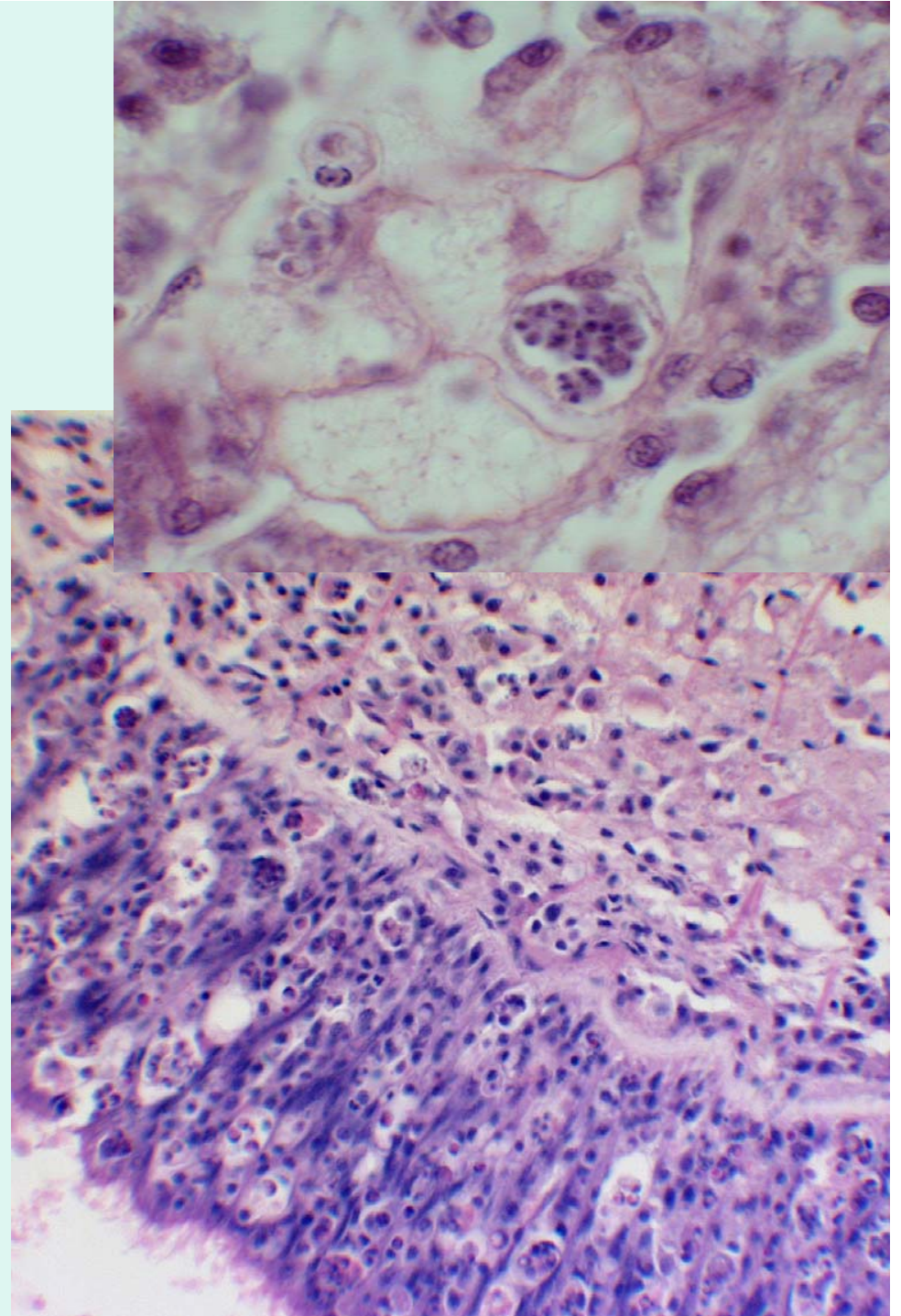


# DERMO DISEASE

- **EPIDEMIOLOGY**
  - **DIRECTLY INFECTIVE**
  - **VIA VECTORS SUCH AS DRILLS (SNAILS)**
- **CHARACTERISTICS OF INFECTION**
  - **>18° C**
  - **SALINITIES OF 15 TO 30 PPT**
  - **DERMO CAN TOLERATE LOW SALINITIES (3PPT)**

# DERMO DISEASE

- **INFECTS IN DIGESTIVE TRACT (ULCERATION OF GASTRIC AND INTESTINAL EPITHELIUM), GILL AND MANTLE**
- **HEMOCYTES ENGULF BUT CANNOT DESTROY (AT LEAST NOT ALL)**
- **PROLIFERATION OF THE ORGANISM IN HEMOCYTES AND SPREAD THROUGH THE BODY**
- **DEATH CAN TAKE 1-2 YEARS HIGHEST MORTALITY IN THE FALL (SEPT/OCT IN MA)**

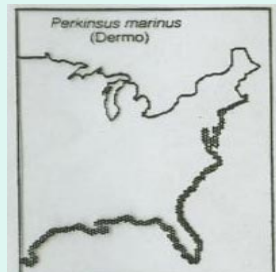




# DERMO DISEASE

## GEOGRAPHICAL LOCATION

- 1940 – CAUSED MORTALITY IN GULF OF MEXICO
- 1950 – FOUND IN CHESAPEAKE BUT NO SIGNIFICANT DISEASE
- 1957 – HIGH MORTALITY ON CHESAPEAKE SIDE OF VIRGINIA (LOWER SALINITY)
- 1980 – HIGH MORTALITY ON THE ATLANTIC SIDE OF VIRGINIA (HIGHER SALINITY WATER)
- 1990 – HIGH MORTALITY IN DELAWARE BAY (HIGH WINTER TEMPS)
- 1992 – FOUND IN MA/CT/RI



# **EFFECTS OF CHANGING ENVIROMENT ON THE OCCURRENCE AND SEVERITY OF DERMO**

- **1997 - INCREASED OCCURRENCE AND SEVERITY OF DERMO HAS BEEN IDENTIFIED IN MA AND IS CASUALLY ASSOCIATED WITH WARMER TEMPERATURES AND EXTENDED MILD FALL TIME PERIODS**
- FORD AND SMOLOWITZ

## **DIRECT EFFECTS OF POLLUTION ON THE OCCURRENCE AND SEVERITY OF DERMO**

- TBT exposure has been associated with increased Dermo prevalence in eastern oysters (Anderson et al. 1996)**
- High levels of Dermo infections were correlated with increased amounts of both high and low molecular weight aromatic pollutants in a river downstream from a creosote plant. (Chu et al., 2000)**

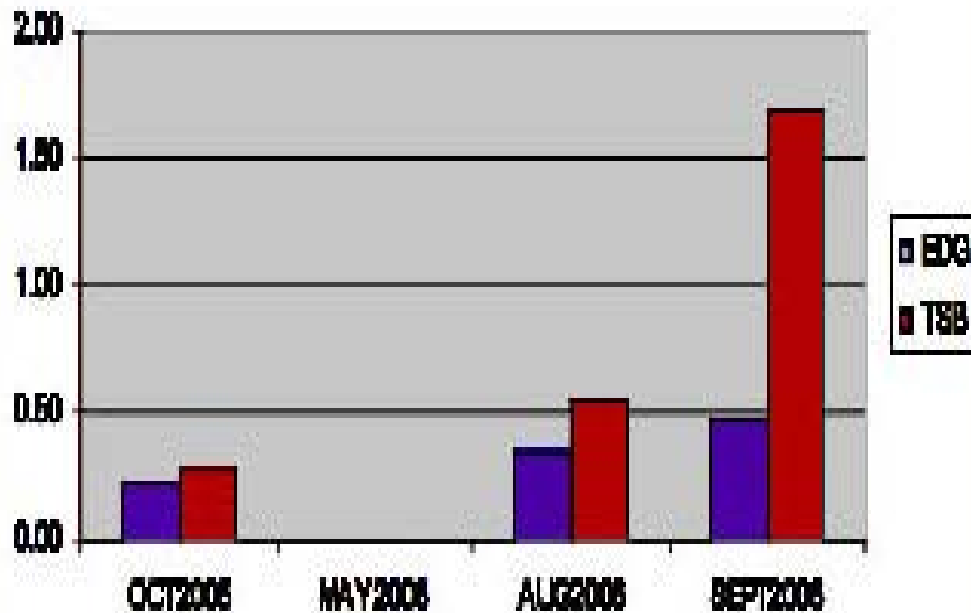
# **EFFECTS OF POLLUTION ON THE OCCURRENCE AND SEVERITY OF DERMO**

- Laboratory experiments** (Chu et al., 1996; Chu et al., 2000) **showed exposure of infected oysters to high molecular weight organics (PAH) contained in suspended clay particles**
  - significantly increased the occurrence and severity of disease**
    - resulted in increases in hemocyte**
      - » number,**
      - » protein synthesis (H-leucine incorporation),**
    - BUT ALSO, resulted in decreases in hemocyte**
      - » phagocytosis**
      - » chemotactic ability**

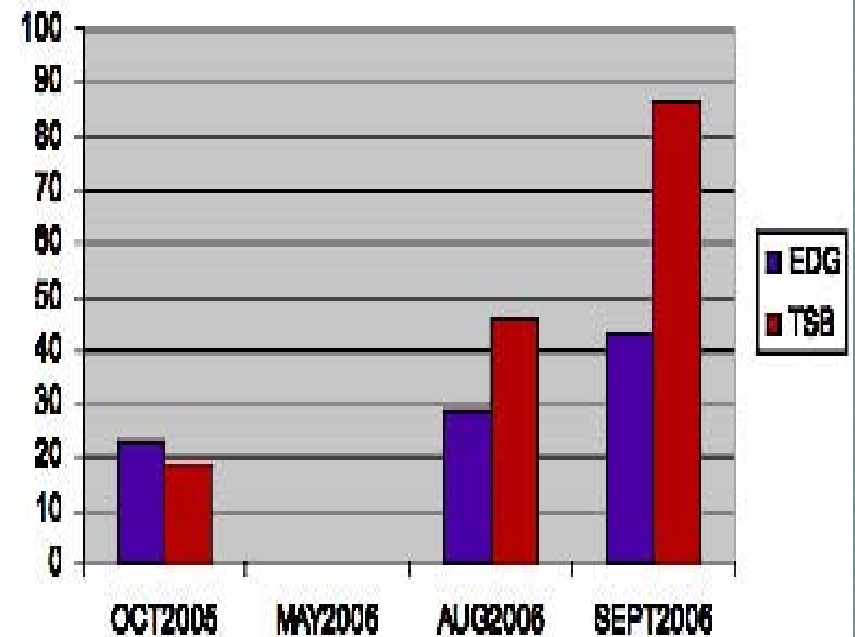
# RESISTANCE TO DERMO

OYSTERS THAT HAVE NOT EXPERIENCED DERMO  
HAVE LITTLE TO NO RESISTANCE TO DISEASE

WEIGHTED INTENSITY

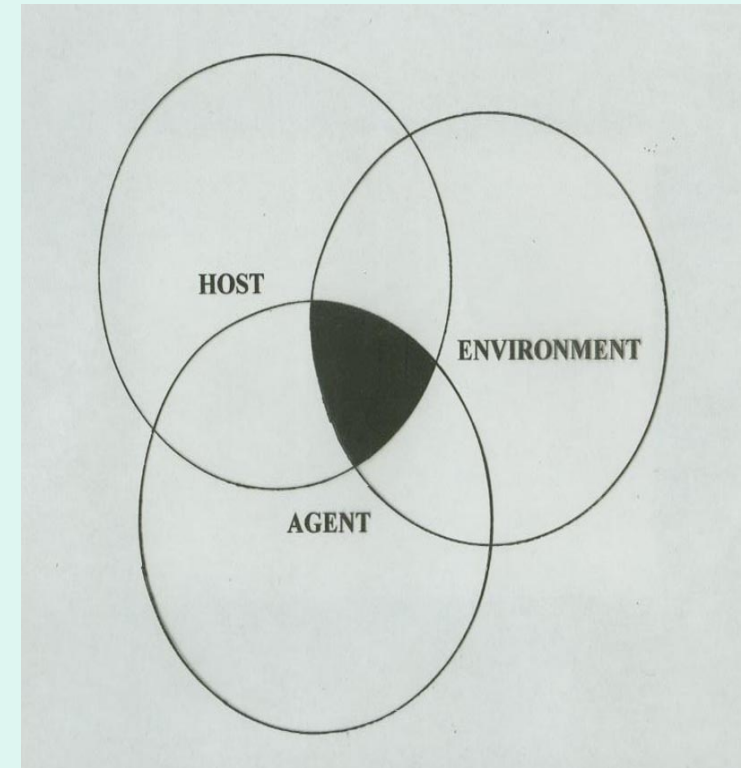


PREVELENCE



## DERMO PROFILE

- Host susceptibility
  - Poorly conditioned animals
  - **Genetic lack of disease resistance (susceptibility)**
  - Age vulnerability
- Agent virulence
  - **New agent in the environment?**
  - **Mutated agent ?**
  - **Change in base population levels of agents (density of agent)?**
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  - **Pollution**
  - **Decreased water quality**
  - **Decreased nutrients**
  - **Temperature/Salinity Changes**



•The elimination or modulation of any one of these factors can decrease or eliminate the occurrence of disease.

# QPX (QUAHOG PARASITE UNKNOWN) DISEASE

- OCCURS IN
  - DENSE GROUPS OF HARD CLAMS (*MERCENARIA mercenaria*)
  - APPROXIMATELY TWO YEARS OF AGE
  - INTERTIDAL TO SUBTIDAL
  - 30PPT SALINITY
- DISEASE PRESENTS AS
  - CLAMS ON THE SURFACE OF THE SEDIMENT
  - SHELLS ONLY OR MILD GAPPERS
  - MANTLE NODULES AND SWELLINGS IN 25 TO 75%

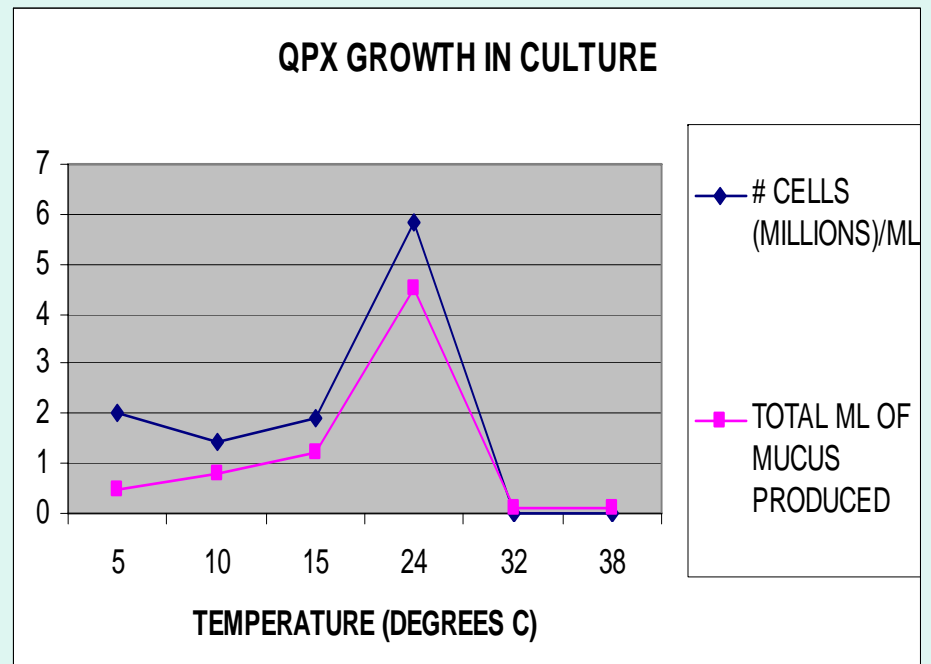
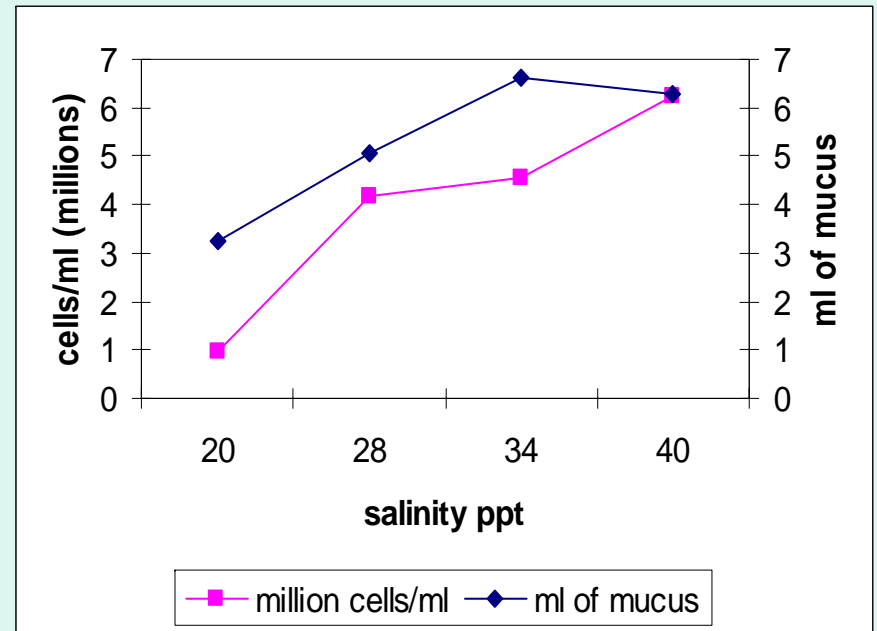
PHOTO BY D. LEAVITT



# QPX GROWTH IN CULTURE

- Ph = 8
- SALINITY = 30 TO 40 PPT
- PREFERRED TEMPERATURE = 24° C
  - DEAD AT 32° C
  - ALIVE, BUT NOT REPRODUCING, AT 0° C

Brothers et al.,





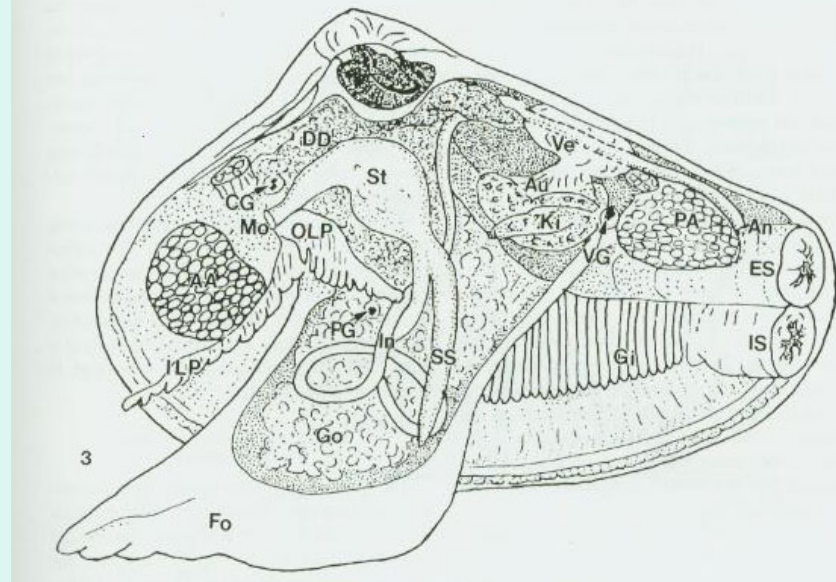
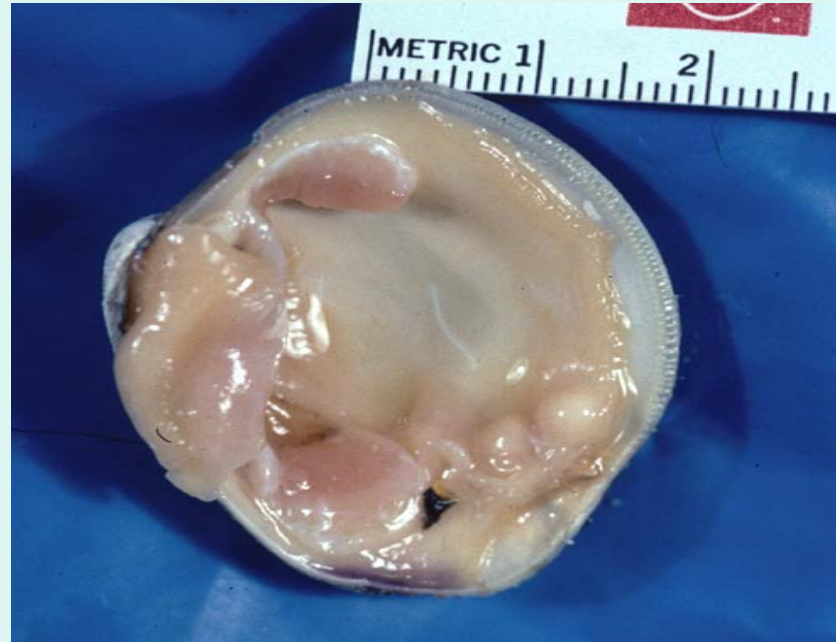
# DISEASE PRESENTATION

**MANTLE NODULES  
AND SWELLING**

**INFECTION OCCURS  
MOST OFTEN AT THE  
BASE OF THE SIPHON**

**DIRECTLY INFECTIVE**

(Smolowitz and Leavitt)



# **DISEASE OF HARD CLAMS**

## **QUAHOG PARASITE UNKNOWN (QPX)**

- **IDENTIFIED IN HEAVY SET OF WILD STOCKS IN CANADA in 1970**
- **IDENTIFIED IN CULTURED STOCKS**
  - **MASSACHUSETTS – 1995**
  - **VIRGINIA -1996**
  - **NEW YORK – 1998**
  - **RHODE ISLAND – 2003**
- **IDENTIFIED IN HEAVY SETS OF WILD STOCKS**
  - **MASSACHUSETTS – 1996**
  - **MASSACHUSETTS (RETROSPECTIVELY) – 1990**
  - **NEW JERSEY (RETROSPECTIVELY) - 1985**
  - **NEW YORK - 2001**

# WHERE DOES QPX COME FROM?

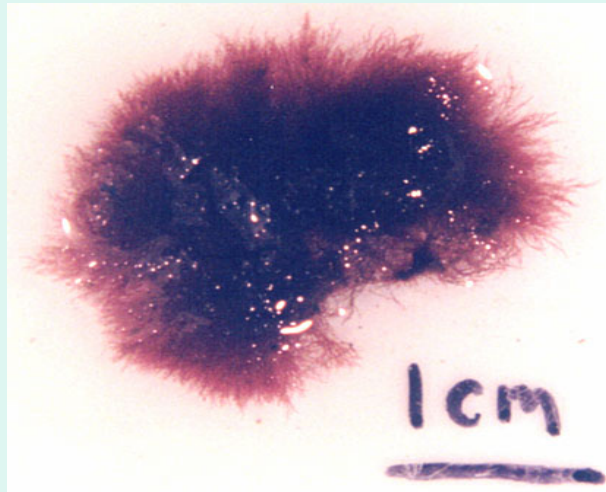
NESTED PCR EVALUATION OF ENVIRONMENTAL SAMPLES  
(GAST, ET AL., )

**Macrophytes: Green Algae**

**57% of total samples (8/14)  
were positive for QPX**

**Macrophytes: Red Algae**

**29 % of total samples  
(5/17) were positive for  
QPX**

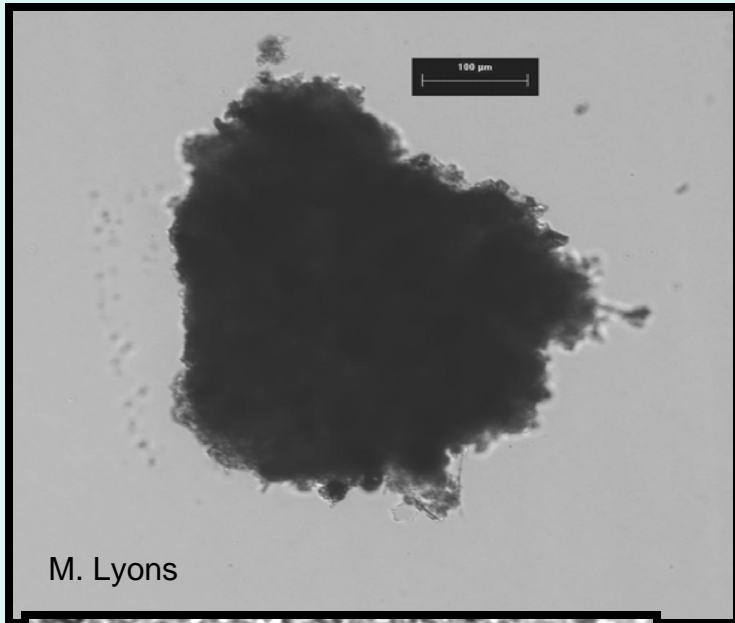


*Spermotheramnion repens*



*Codium fragile*

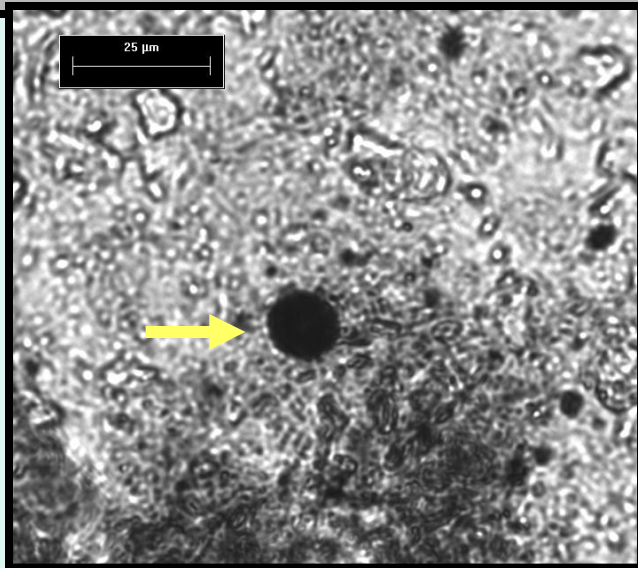
# HOW DO INITIAL INFECTIONS OCCUR Marine Aggregates? (LYONS, M., ET AL.)



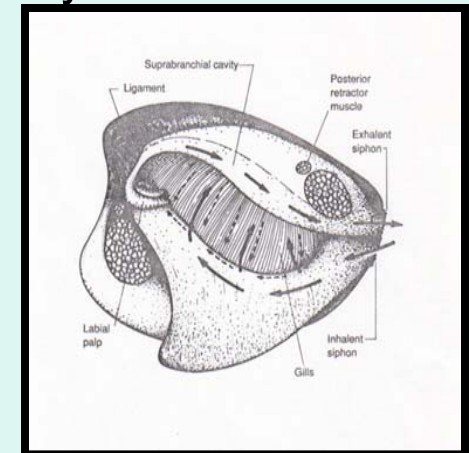
Polysaccharide rich organic matter (originating from bacteria etc.) coalesced to form Transparent Exopolymer Particles (TEPs)

TEPs attract phytoplankton and other particles to form aggregates in the water

TEP quantity and quality is effected by temperature, salinity and nutrients



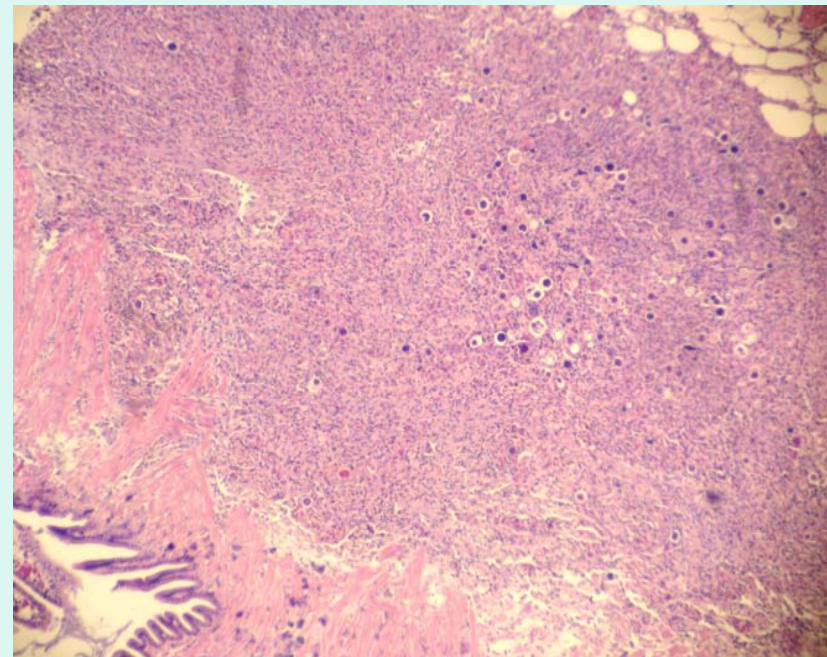
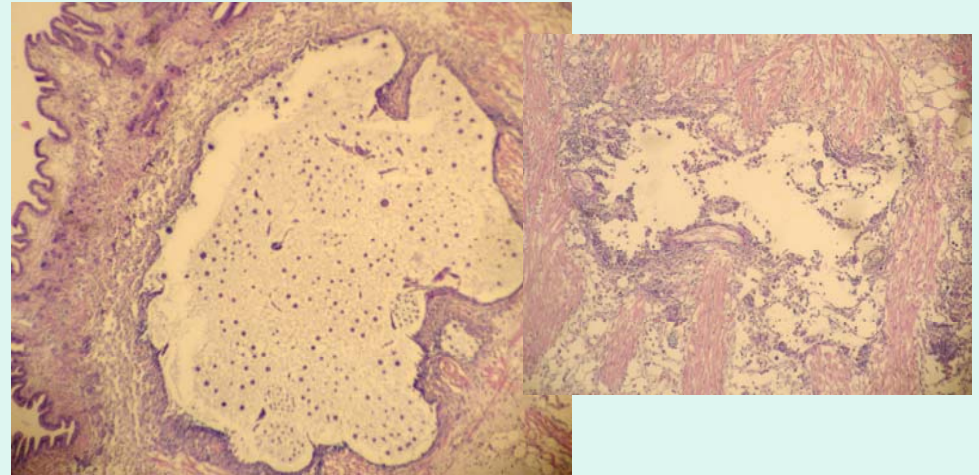
**Natural marine aggregates from QPX-infected shellfish beds**



# TEMPERATURE EFFECT ON DISEASE IN CLAMS

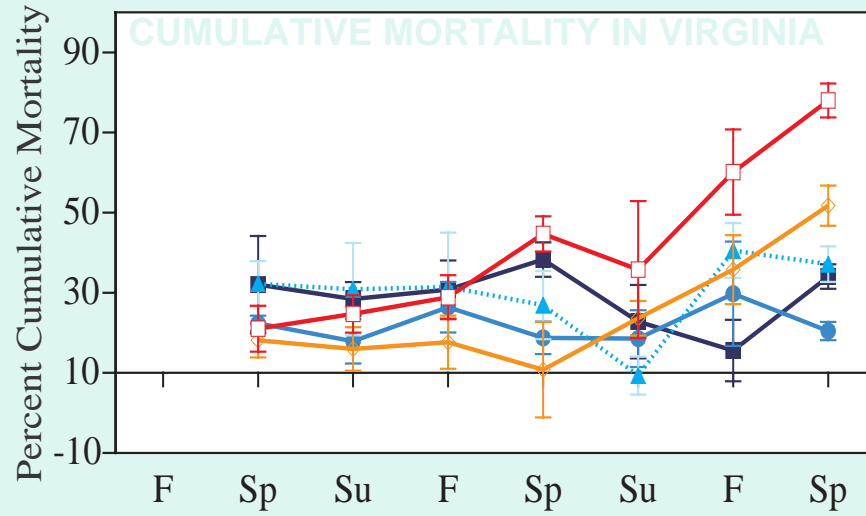
## FROM FIELD STUDIES

- INFLAMMATORY SYSTEM OF CLAMS SHOWED LESS RESPONSE TO INFECTIONS AT LOWER TEMPERATURE
- AT HIGH TEMPERATURE, INFLAMMATORY RESPONSE WAS GREATER AND THE NUMBER OF VIABLE QPX ORGANISMS WAS DECREASED IN THE TISSUES



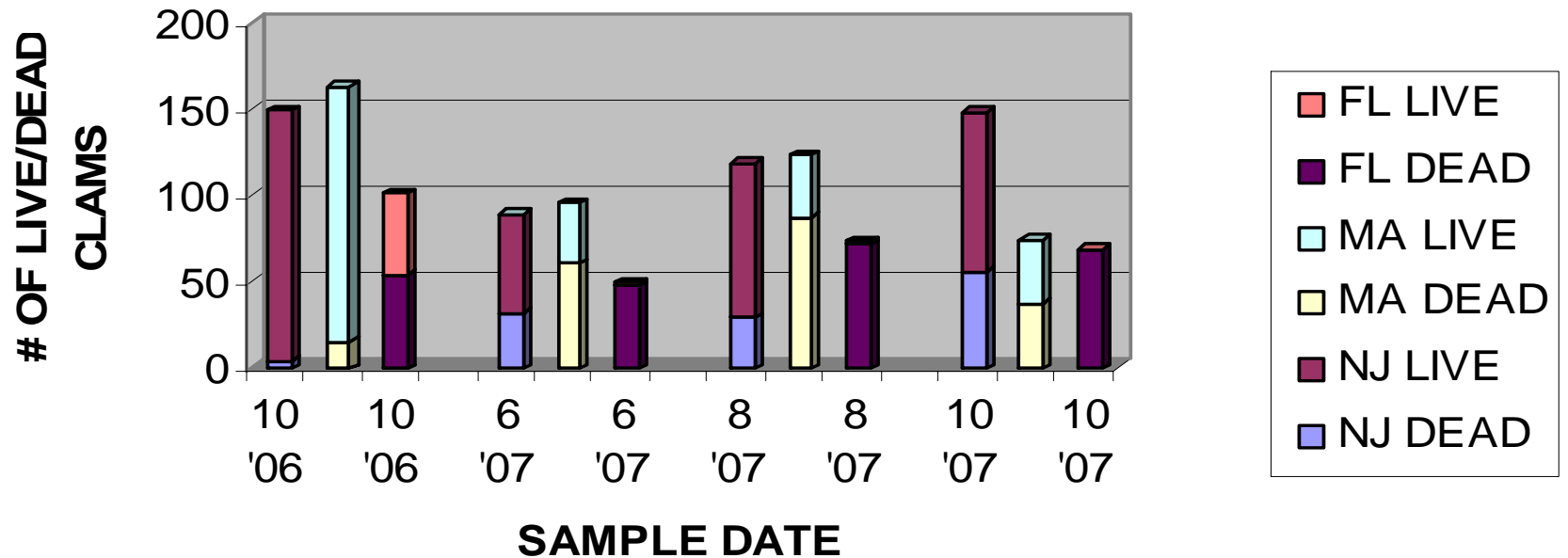
(SMOLOWITZ ET AL.)

# EFFECT OF CLAM STRAIN ON QPX CAUSED MORTALITY



■ MA  
 ● NJ  
 ▲ VA  
 ◆ SC  
 □ FL  
 Ragone-Calvo et. al., 2006

## LIVE/DEAD CLAMS PER STRAIN AND SAMPLE PERIOD FROM CORES

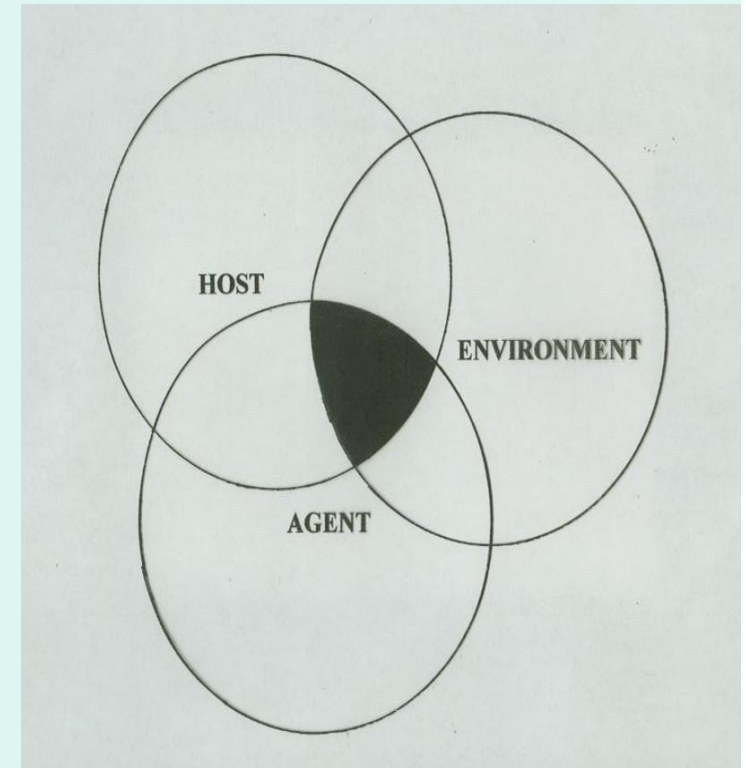


# **POTENTIAL EFFECTS OF ENVIRONMENT/POLLUTION**

- **INCREASED DETRITUS IN THE ESTUARY**
  - (I.E. EFFECTING THE QUANTITY OF MARINE AGGREGATES, SEaweEDS AND QPX)
- **WATER TEMPERATURES- Milder WINTERS?**
- **HIGH SALINITY IN ESTUARIES (SUMMER DROUGHT)**

## QPX PROFILE

- Host susceptibility
  - Poorly conditioned animals
  - **Genetic lack of disease resistance (susceptibility)**
  - Age vulnerability
- Agent virulence
  - **New agent in the environment**
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- **USUALLY A MULTIPLE OF FACTORS RESULTS IN DISEASE**
- **WE NEED TO UNDERSTAND EACH AND THE INTERACTION BETWEEN THEM TO IDENTIFY**
  - **HOW, WHEN, WHAT AND WHO OF DISEASE**

**WE MAY HAVE NOT CONTROL OVER THE ENVIRONMENT, BUT WE DO HAVE CONTROL OVER CULTURE METHODS!**

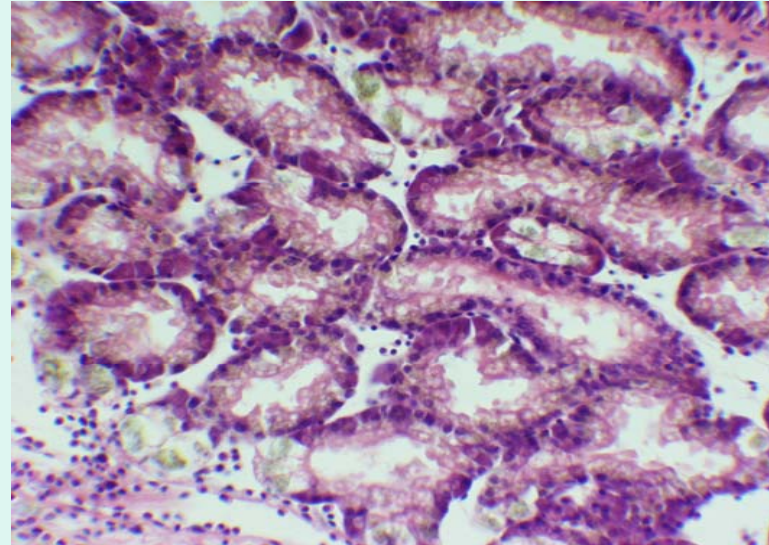
# **EFFECTS OF HARMFULL ALGAL BLOOMS (HABS) ON THE HEALTH OF BIVALVES**

- **Recent evidence has shown red and brown tides (caused by dinoflagellates and other picoplanktonic algae) effect bivalves**
  - **Direct effects**
  - **Indirect effects**
- **Effects of a specific HAB varies with bivalve**

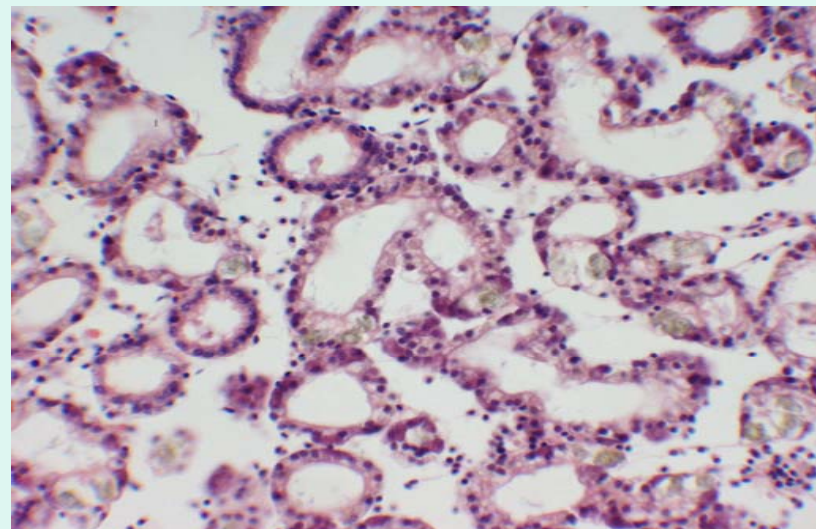
# *Effects of Dinoflagellates vary by life stage of the bivalve*

## *Prorocentrum minimum*, a dinoflagellate

- Argopectin irradians (Bay scallop)
  - Causes acute necrosis of the digestive gland absorptive cells and clotting of hemocytes in the hemolymph of post-metamorphic juveniles
  - Older juvenile scallops are unaffected



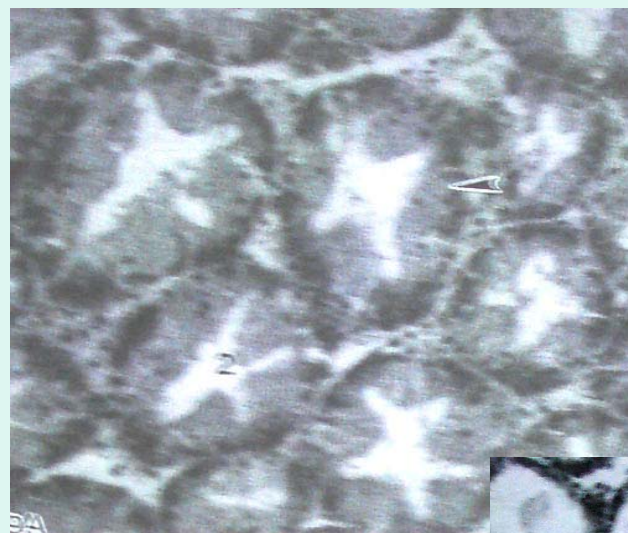
CONTROL



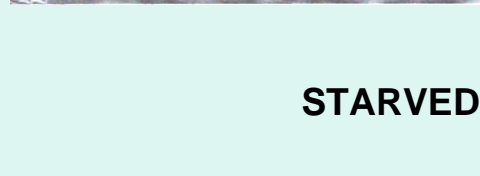
PROROCENTRUM  
FED

# DIRECT EFFECT OF BLOOMS ON BIVALVES

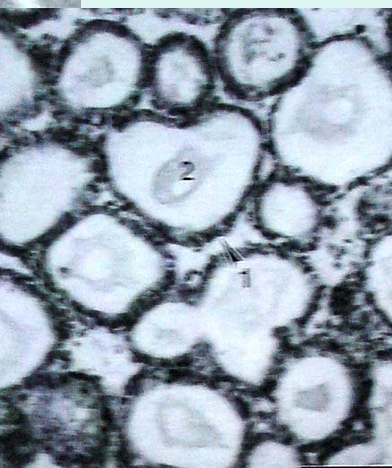
- Effects may result from decreased digestibility of the bloom organisms.
  - *Prorocentrum minimum* (EXUV) fed to juveniles of the eastern oyster resulted in apparent histological activity of the absorptive cells in the digestive gland but poor growth of the animal over time.



CONTROL



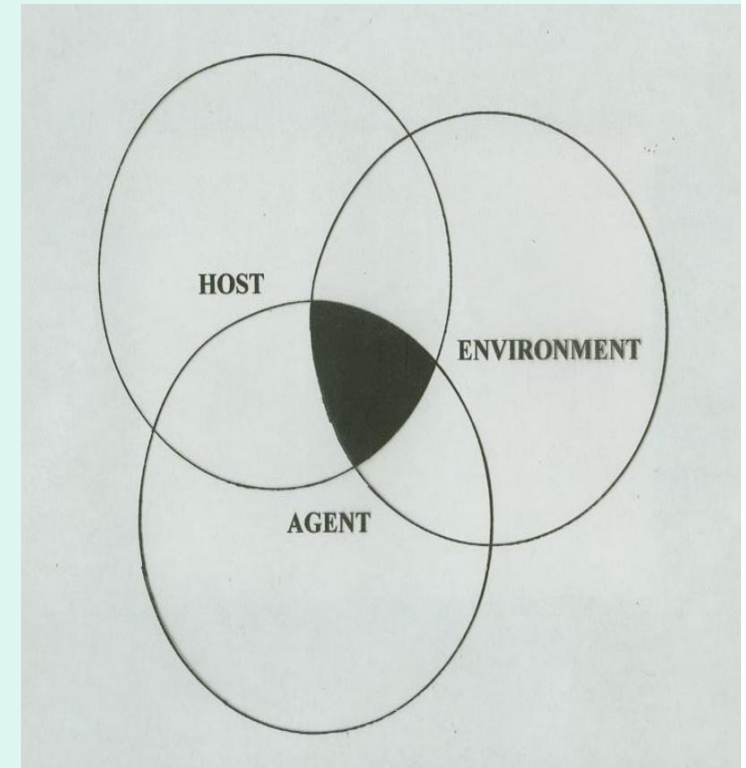
STARVED



EXUV FED

## DINOFLAGELLATE PROFILE

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