Investigating disease outbreaks on prawn farms: practice, theory and prevention?

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Australian Prawn Farmers Symposium 2019
– Disease outbreak investigations

– Virulence evolution theory
Disease outbreak investigations

North of Brisbane
Not white spot
Objectives & Method

- Non-random

- Temporal (time)
  - Epidemic curve

- Spatial
  - Visual (map)
  - Local clustering – SatScan
  - Global clustering – Moran’s
Results

- Temporal
  - Epidemic curve

![Epidemic curve graph]
Results

- Spatial I
  - Visual
  - Clustering at 30 days
Spatial II autocorrelation

None

Positive autocorrelation

Negative autocorrelation

https://www.google.com/search?q=spatial+autocorrelation&rlz=1C1GGRV_enAU764AU764&source=lnms&tbm=isch&sa=X&ved=0ahUKEwiBqIaM89HjAhWDXCsKHSTaC_sQ_AUIEStGb&biw=1536&bih=722#imgrc=JX506lGMMmF2ZRM:
Spatial II

- Moran’s statistic
- Outcome variable = time from index case in days
Conclusion

- Point source & propagating

- Pond to pond spread

- Typical of infectious disease (FMD, Equine influenza)

- Birds (farmers observations)
  - Pick up moribund prawns
  - Drop them in other ponds

- But
  - Some outbreaks die out for no reason
  - Always a few ponds left unaffected
Virulent evolution theory

As it relates to emerging viral diseases
Virulence evolution theory

– Virulence = case fatality rate?

– Background

– Objectives

– Results

– Discussion

– Conclusion
Pasteur 1881

“Its virulence, then reinforced by successive passages through members of this species …”


– Dog rabies -> rabbits

– Passage more than 100x

– Incubation period: > 15 days to < 7 days

– Virulence continues to increase
Virulence evolution theory

– Why aren’t we all dead?

– Anderson & May 1979 *Nature*

– Transmission trade – off

1. Within host - selection for higher virulence

2. Between host - favours long duration & low virulence (virus dies with host)

– What about prawns (and fish)?
– Infectious after die and cannibalism!
Mouse model - Anderson & May

- *Pasteurella muris*

- SIR model

- \( n = 21 (7 \times 3) \)

- Add 1 mouse every 3 days

- Plot population at equilibrium on virulence (alpha)
Virulence evolution theory

- Mouse model
  - *Pasteurella muris*
Equilibrium - Low alpha

- High number of recovered
- Low number of susceptible
Equilibrium - Medium alpha

- Low number of recovered
- High number of susceptible
Equilibrium - High alpha – disease dies out

- All are susceptible at equilibrium
Sum 3 compartments -> population equilibrium

Optimum trade-off ~ empirical alpha
Prawn model hypothesis

Virulence predicted by model = actual virulence
Prawn model

- Published model from tank experiments
- Lotz & Soto 2002. *Diseases of Aquatic Organisms*
- Soto & Lotz 2001. *J. Invertebrate Pathology*
- WSSV in White legged shrimp - *Penaeus vannamei*
Prawn model

1. Water spread - acute & chronic - low $B \ (0.01)$

2. Ingestion / cannibalism - very high $B \ (0.5)$
   - Dead are infectious
   - No trade-off

3. Unrestrained increase in virulence?!

Similar to model for Taura syndrome
Results - low alpha

- High number of chronic; latent & acute peak
- Low number of dead – water spread – wild situation?
Results - medium alpha

- Low number of chronic; latent and acute peak
- Higher number of dead
Results - high alpha

- Low number of chronic; latent & acute peak
- High number of dead with peak
Results – population equilibrium as $\alpha$ increases
Reject our hypothesis in favour of the alternative hypothesis:

Actual virulence is lower than virulence predicted by the model.
Why is this so?

1. Source of WSSV was mainland China in 1996
   - It was maintained in *P. vannamei*
   - Only water spread in maintenance environment?
   - Lost virulence?
If only acute are infectious

- Trade off evident
- Bottom of curve even closer to empirical value
Discussion

2. Trade off at pond level

• Need surface swimming sick prawns

• Moribund prawns infected with virulent strains - don’t move & not seen -> no bird spread

• Virulent strains die out
Discussion

3. “All models are wrong but some are useful”

George Box
Field evidence

1. 
   • Water spread in wild
   • Less virulent strains of WSSV & other viruses in the wild

2. Disease die out (owing to ^ virulence)
   • No visible prawns
   • No visible birds
General discussion

INCREASED DENSITY

– Initially
– Increased contact -> Increased transmission
  -> Favours increased virulence

– Then
  -> Increased prevalence
  -> Decreased susceptibles
  -> Decreased transmission
  -> Favours decreased virulence

= Same virulence but ^ prevalence
General discussion

HORIZONTAL vs VERTICAL

- Horizontal & Epidemic
  - Less constrained by transmission trade-off
    - -> Increased virulence

- Vertical & Endemic
  - Virulent agents kill host & die with host
  - Leads to lower virulence in next generation
  - Endemicity favours lower virulence
General discussion

Treatments / vaccinations (Fish)

- Imperfect vaccines
  - Favours virulence

- Treatments
  - Early dead are virulent & still infectious
  - Less virulent are killed by Tx
  - Favours virulence strains
Outbreaks & emerging disease - virus & PMMS

1. Emerging disease trigger
   - Density & horizontal spread
   - Cannibalism
   - ↑ virulence (no transmission trade off)

2. Pond to pond spread
   - Birds & moribund prawns
   - Transmission trade-off prevents ↑↑↑ virulence
Routine - year to year

Low virulent & environmental bacteria

- Tendency to ↑ Virulence
Prevention
Prevention

1. All in all out with disinfection / drying

2. Dry entire farm?
   • Kill virulent agents in reservoir

3. Stop spread from initial pond
   • Bird control (Drones?)

4. Record time / space data
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