

# Gill diseases; Field experiences and possible interpretations



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almost 30 years in  
aquaculture.

Mostly atlantic  
salmon, some trout.

West Coast of  
Norway: Hordaland,  
partly fjord locations,  
partly coastal  
locations.

Also working with:

Farm animals,  
dogs, cats,  
turkeys, and  
laboratory.

# Fieldwork:

Gill issues in Atlantic salmon seem to be on the rise.

Fieldwork include:

- Diagnosing
- Treating?
- Trying to understand the entire background of the disease, not just the pathogen
- Try to find out what we can do to prevent disease next year?

"The perfect job for someone who enjoys reasoning and philosophizing.

# The difference between pathogen and disease

Pathogens are often involved in diseases – but:

- One can be infected with pathogens without triggering a disease
- Environment and stress factors will always be part of a disease where pathogens are present. Always try to understand them!
- It's important to create overviews and look for recurring events that are repeated across different cases. It's essential to reason from a fundamental understanding of how the disease develops.
- Disease diagnostics - clinical or pathological - is more useful than PCR alone. PCR alone can give you a misplaced focus.

# Key environmental factors that can open up to pathogens and trigger diseases?

## Weakened immune system

- Smolt quality – what is smolt quality?
- Feed quality - does it affect the local epithel quality of gills as well as the immune system in general?
- Stress factors - physical and psychological. Natural or induced.

## Gill damage as an entry point for pathogens?

- Algae and jellyfish – an important factor?
- Net washing?
- Hot water – lice treatment??
- Particles in the water?
- Low oxygen?

# Relevant gill pathogens in atlantic salmon.

- *Ca. Branchiomonas cysticola* - in epitheliocystis (and some others)
- *Desmozoon lepeophtherii* - Microsporidia
- Amoebae - AGD (Amoebic Gill Disease)
- Pox-virus
- *Tenacibactulum*

Usually several concurrently – In what sequence do they appear, and what significance does each of them have?"



# Clinical experiences with various gill pathogens

Typical findings in epitheliocystis: Frayed gills and eventually slim fish.



# Epitheliocystis and infection with microsporidia

Usually both are present in varying proportions, but I have mostly seen epitheliocystis.

- Partially linked to specific locations – does this mean localized transmission?
- There can be significant variations in the course of disease among different smolt groups at the same location.
- Branchiomonas is also found in freshwater.
- Clinical disease by us usually starts in August, subsiding by October. If it begins early in August, it often results in a severe outbreak, while if it starts in late August or in September, it's usually milder.

- Mortality often decreases from the beginning to the middle of October. Does this imply a temperature threshold of 12-14°C?
- Does it mean that the disease begins to develop at temperatures above 12-14°C (May, in our region)?
- Some years are worse than others - why? Could an increase in algae or jellyfish that harm gills be a possible cause?
- There's often a milder course in autumn fish compared to spring fish. Is this due to partial immunity from the previous autumn, or because larger fish can tolerate more than smaller?
- If there is partial immunity, would detecting subclinical branchiomonas in smolt by pcr be an advantage or a disadvantage?



# Amoebae

- First autumn with amoebae in Norway, we did see extensive mucus masses on the gills, with proliferations underneath.
- Later on - usually smaller quantities, not clinically visible, but detected in submitted samples and PCR.
- Less at lower temperatures but can be found year-round. Common occurrence. Less problem i fjord locations, with salinity <25‰.
- Clinical significance with smaller quantities? Can be present without any clinical signs. Partial immunity? – since I do not see much mucus any more, and it often does not seem to develop to the worse?



- Can a stepwise damage lead to more severe outcomes?  
Amoebae in smaller quantities does probably not cause enough damage to be lethal on its own, but can be fatal if it occurs on top of other injuries.
- Can smaller quantities open up to other pathogens and lead to more severe outcomes of other pathogens, compared to a situation without amoebae?



# Tenacibactilum

- I have no personal experience with this pathogen on gills.
- Can cause necrosis and severe gill disease where it is abundant.

# Pox

Three cases with mortality connected to pox:

- Juvenile fish facility with a purchased low-quality group. High mortality. In the rest of the facility pox was found by PCR, but without increased mortality. Individual fishes with clinical findings typical of pox were also discovered in the other tanks.
- A juvenile fish facility with pox and branchiomonas. No branchiomonas mortality until pox was added. High mortality. Other tanks in the facility had much milder courses.
- A sea facility with weak to moderately gill issues dominated by epitheliocystis. Developed pox on top of it with very high mortality, approximately when a temperature-related decrease was expected in october. Duration was about 8 weeks. One net from another smolt supplier had a much milder course.
- Detected pox multiple times in screening without visible disease.
- Typical image on direct microscopy in juveniles: No increased slime on the gills compared to what is normal, but disturbed structure. Secondary lamellae stick together at the tips, creating gaps between others.



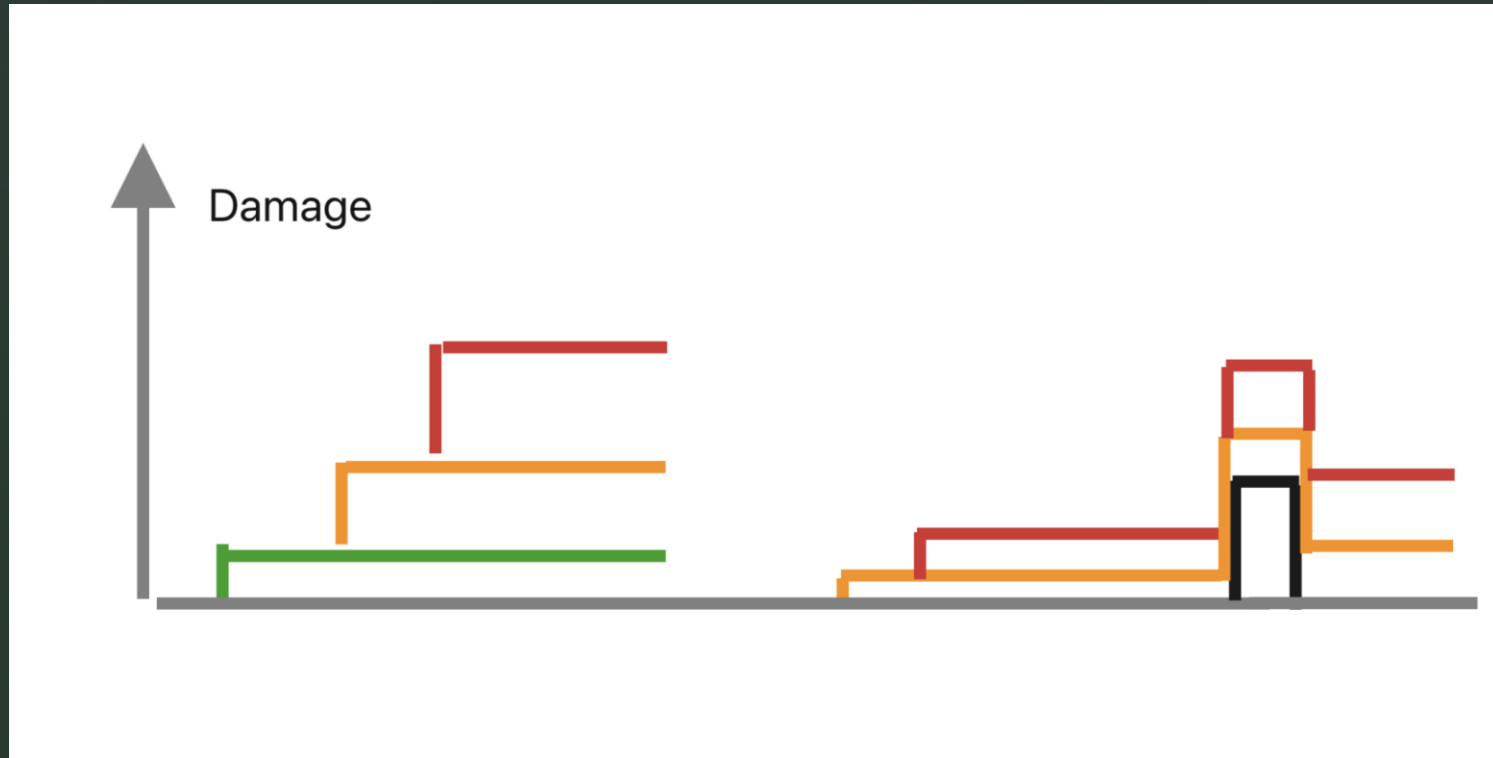
Do the damages stack up like steps, or will a pathogen like amoebae lead to more severe courses for the other pathogens??

Blue –  
amoebae

Orange -  
microsp.

Red –  
epitelio

Black –  
algae or  
jellyfish

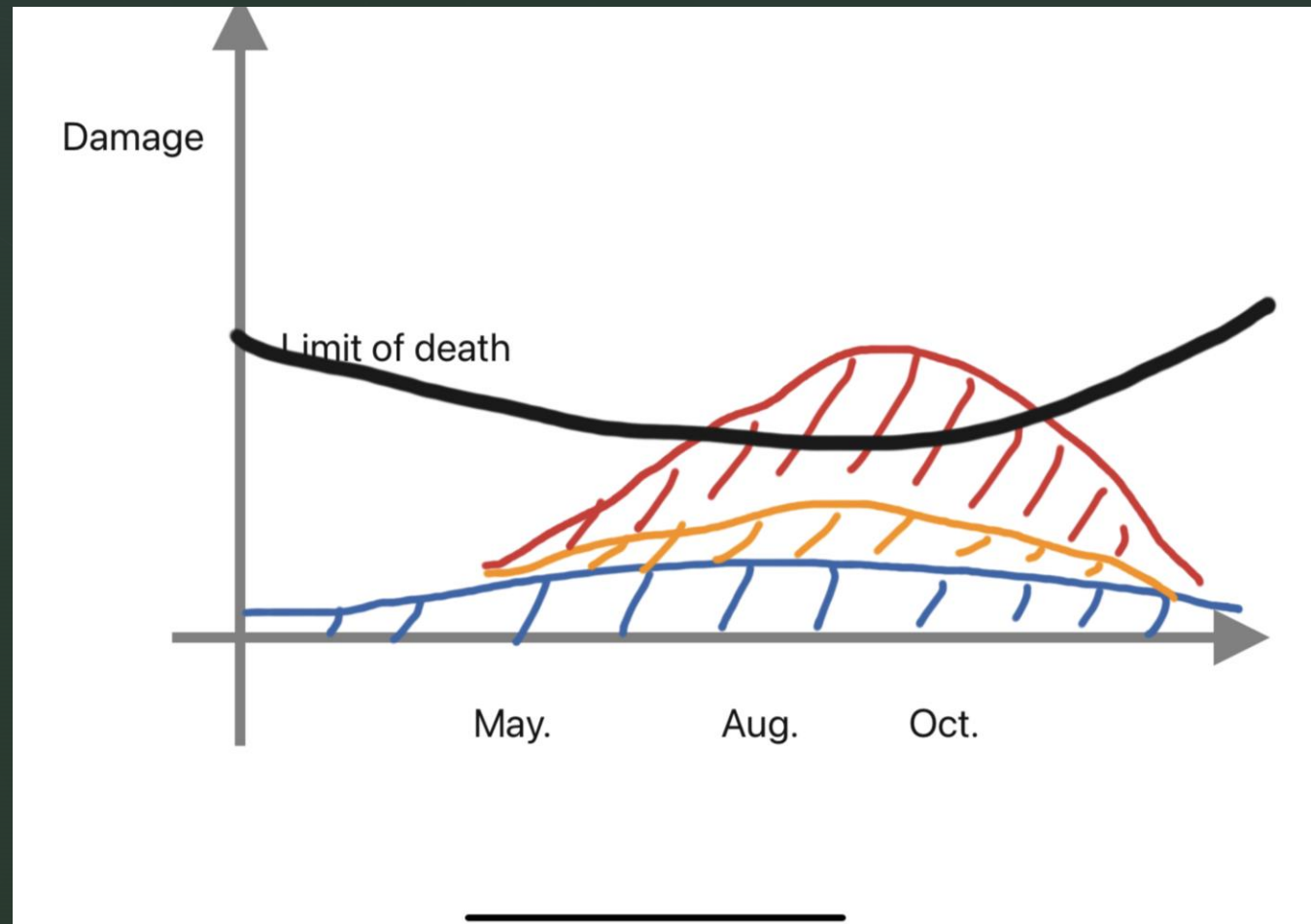


# A theoretical model for mortality, based on empirical experiences

Blue –  
amoebae

Orange -  
microsp.

Red -  
epitelio



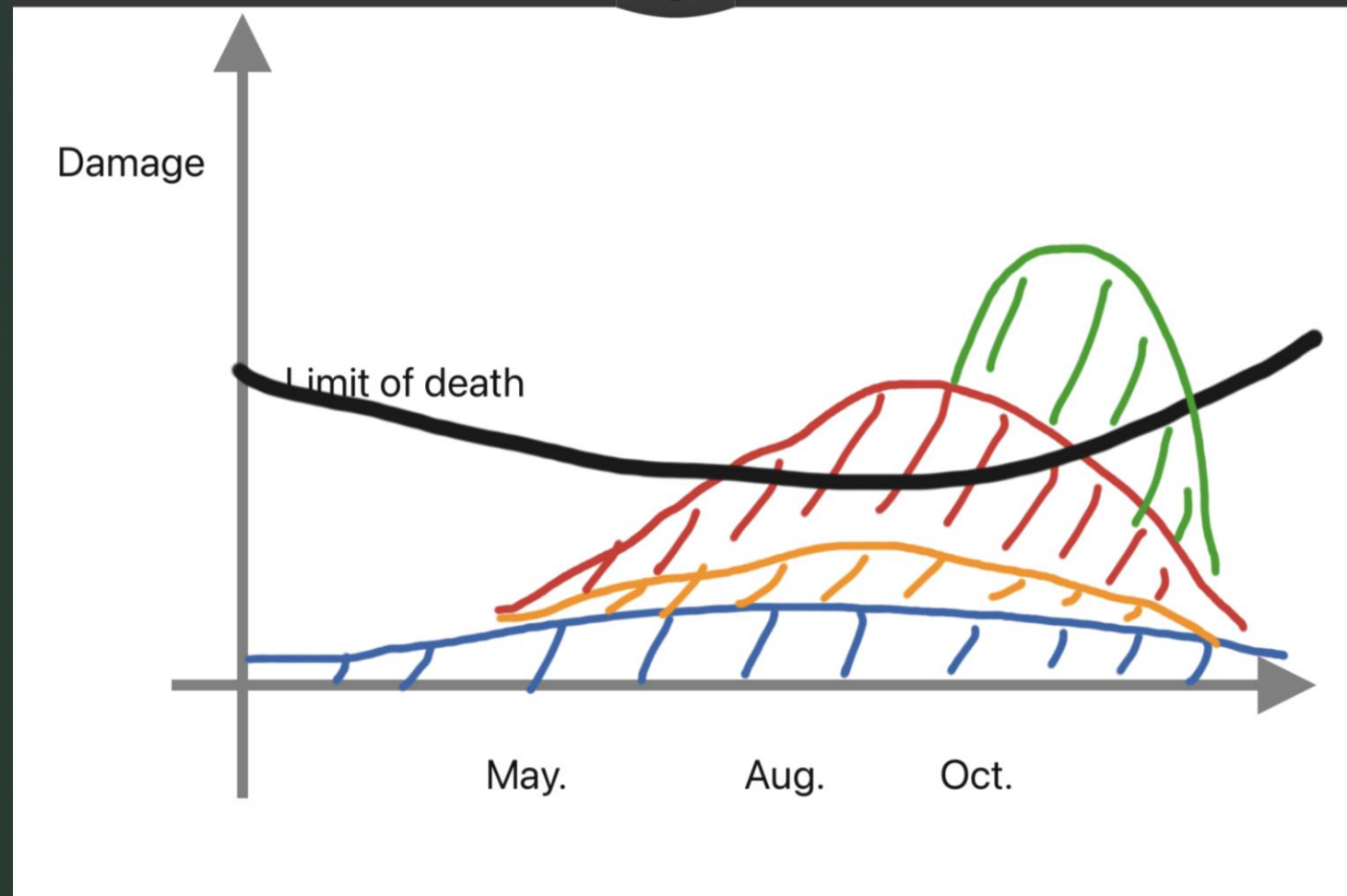
# A theoretical model for mortality based on empirical experiences

Blue –  
amoebae

Orange -  
microsp.

Red –  
epitelio

Green –  
pox in  
october

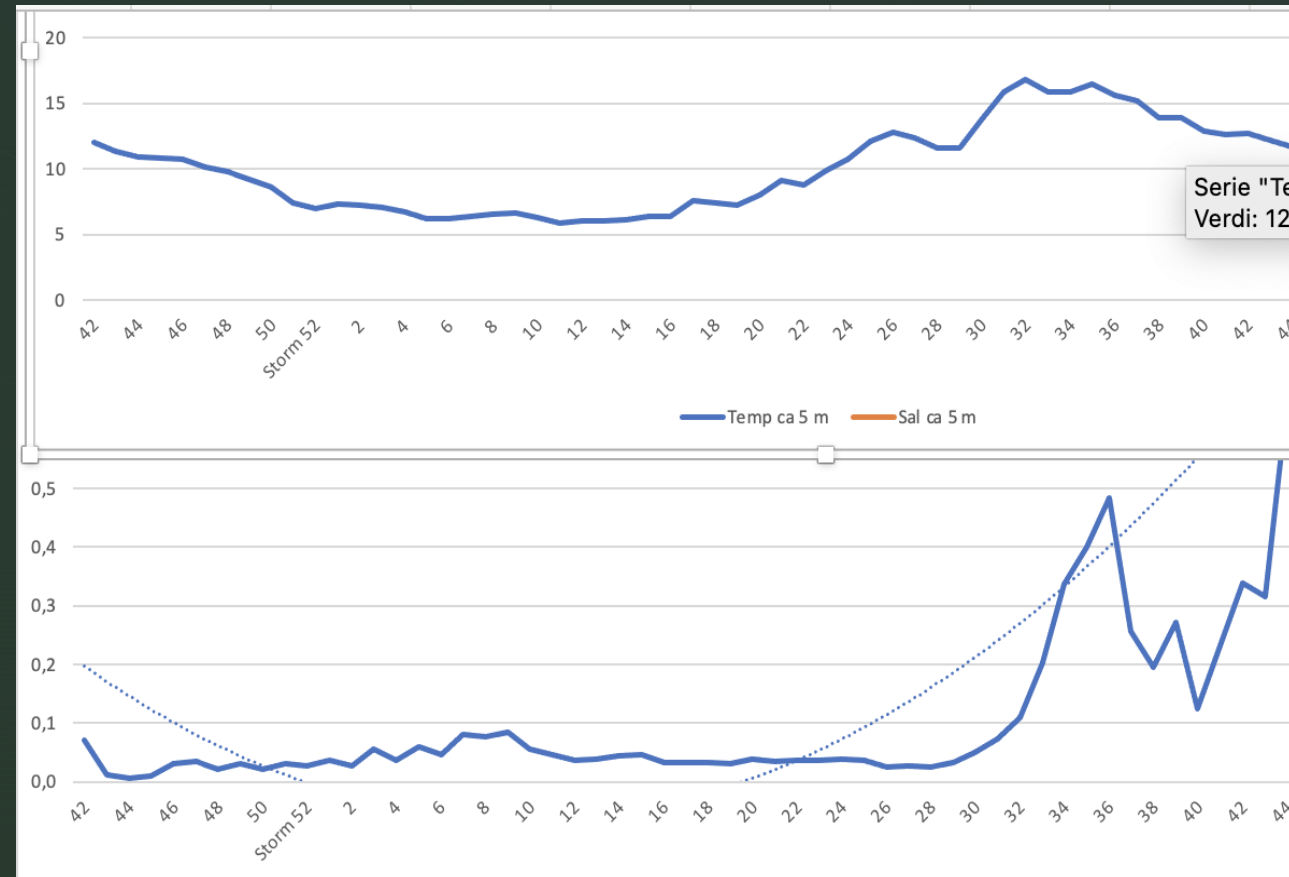


# Cases – location A – coastal – autumn outset 2018

Week 36:  
Epiteliocystis,  
AGD, pox – not  
a clear picture

Week 44:  
Slim fish dies.

Low acute  
mortality, but  
some slim losers  
die afterward.

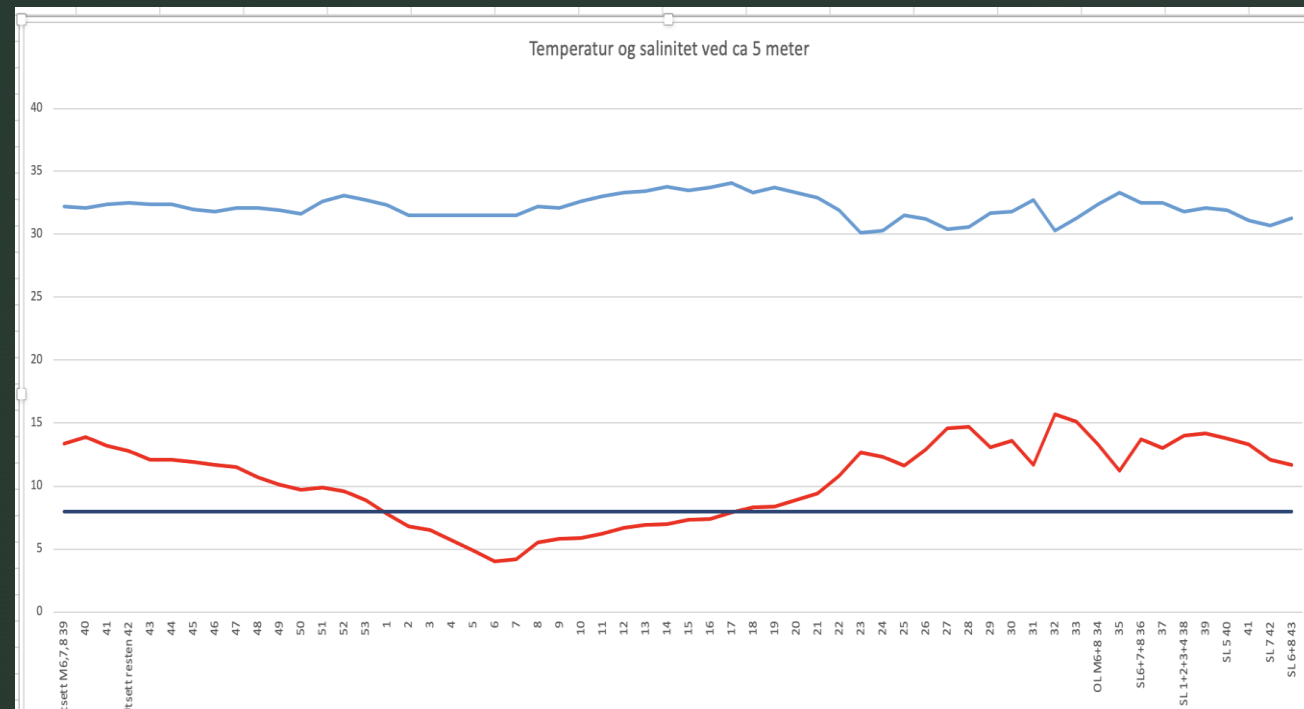




# Cases – location A – coastal – autumn outset 2020

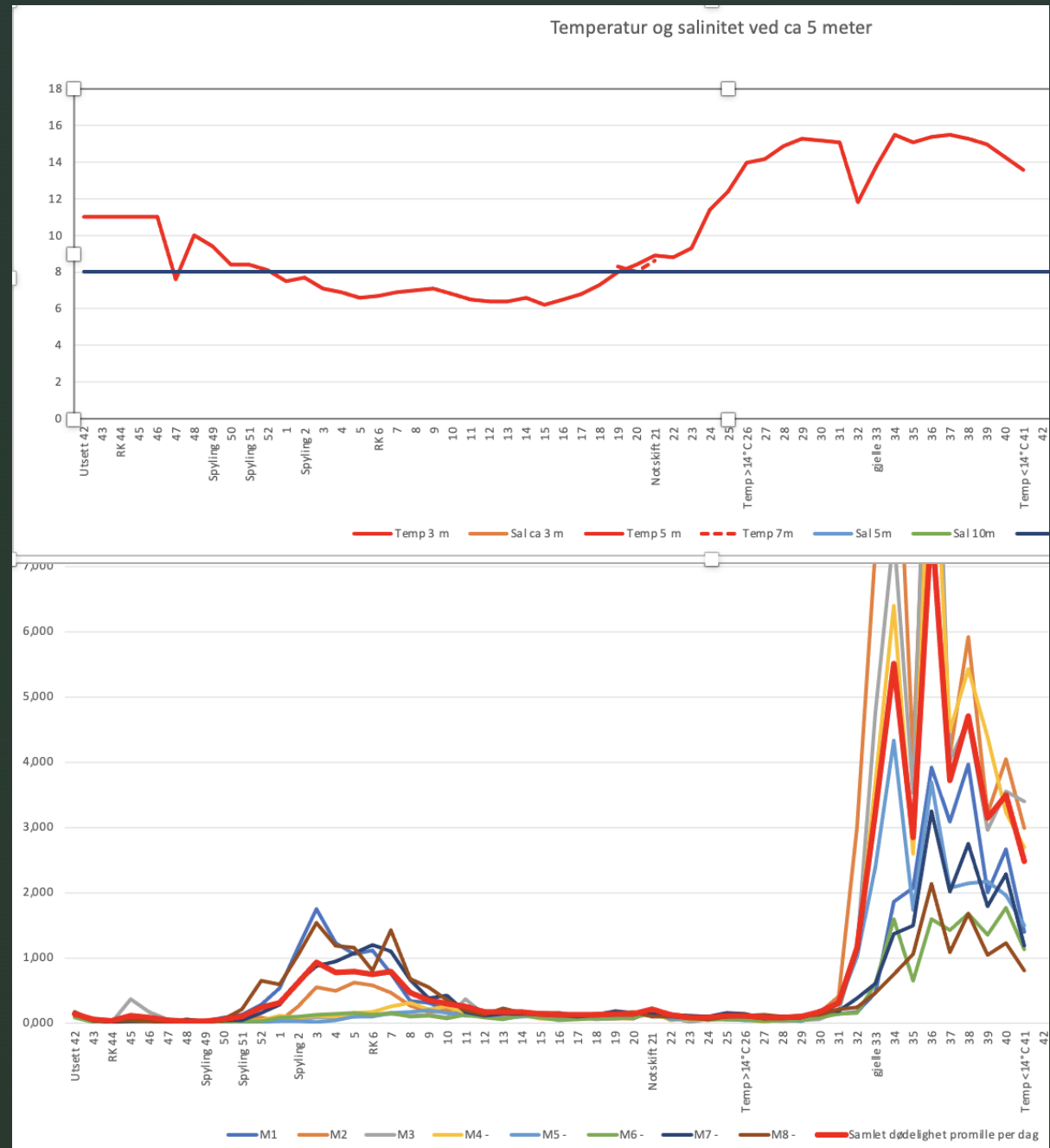
No mortality  
diagnosed to gill  
diseases this year.

Is it due to  
relatively low  
temperatures at  
the site? Less  
algae and  
jellyfish? Low  
temperature is  
often because of  
wind from north.



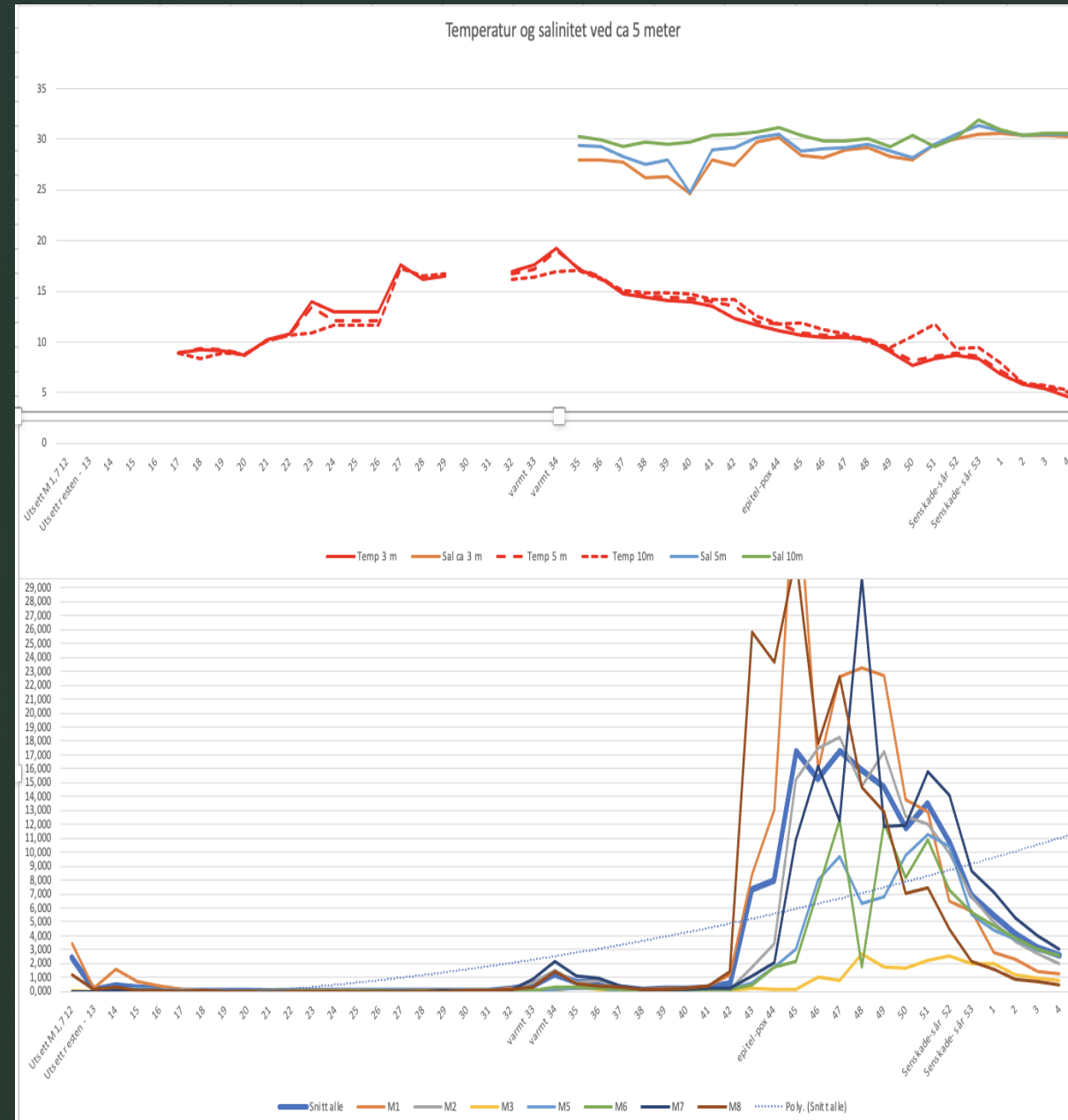
# Cases – location A – coastal – autumn outset 2022

High mortality that started early and increased rapidly. Mostly there were findings of epitheliocystis, but also some microsporidia, small amounts of amoebae, and pox. Many others in the region are facing gill issues this year. Could algae or jellyfish have triggered the disease? Wind direction this summer – mostly south and west, less north? First and most in the nets closest to the shore, including the shallowest nets?



# Cases – location B – outer fjord – spring outset 2020

Some mortality associated with high water temperature in mid-August. A slight increase in mortality due to epitheliocystis and AGD was observed in early October. Short oxygen drop in week 42. Triggering factor? Rapid and very strong increase afterward, where pox was also found. Duration of mortality peak approximately 8 weeks, with a tail of losers dying afterward. Note net 3 from another smolt facility.



# Can we treat gill disorders?

- AGD can be treated with freshwater or H<sub>2</sub>O<sub>2</sub>.
- Other pathogens have traditionally not been treated.
- Some facilities that have frequently treated against sea lice with freshwater, believe they have seen some reduction in gill problems (I do not have personal experience with this).
- Based on this feedback, some have tried freshwater on fish with extensive gill issues this fall (including AGD), but from what I've heard, the results haven't been very positive.
  - Do amoebae or *Tenacibaculum* dominate in those where the treatment is effective?
  - Will regular monitoring of smaller quantities of amoebae limit the entry point for epitheliocystis and microsporidia?
  - Will cooling the treatment water on a wellboat (4-5 hours retention time) have a slowing effect on epitheliocystis with regular repetitions?
- Do we have enough experiences to conclude anything at all??