DATA Nugget

**Dangerous Aquatic Prey: Can Predators Adapt to Toxic Algae?**

Featured scientists: Michael Finiguerra and Hans Dam from University of Connecticut-Avery Point, and David Avery from the Maine Maritime Academy

*Research Background:*

**Figure 1:** Scientist Finiguerra collecting copepods at the New Jersey experimental site.

Phytoplankton are microscopic algae that form the base of all aquatic food chains. While organisms can safely eat most phytoplankton, some produce toxins. When these toxic algae reach high population levels it is known as a **toxic algal bloom**. These blooms are occurring more and more often across the globe – a worrisome trend! Toxic algae poison animals that eat them, and in turn, humans that eat these animals. For example, clams and other shellfish filter out large quantities of the toxic algae, and the toxic cells accumulate in their tissues. If humans then eat these contaminated shellfish they can become very sick, and even die.

One reason the algae produce toxins is to reduce predation. However, if predators feed on toxic prey for many generations, the predator population may evolve resistance, by natural selection, to the toxic prey. In other words, the predators may adapt and would be able to eat lots of toxic prey without being poisoned. Copepods, small crustaceans and the most abundant animals in the world, are main consumers of toxic algae. Along the northeast coast of the US, there is a toxic phytoplankton species, *Alexandrium fundyense*, which produces very toxic blooms. Blooms of *Alexandrium* occur often in Maine, but are never found in New Jersey. Scientists wondered if populations of copepods that live Maine were better at coping with this toxic prey compared to copepods from New Jersey.

Scientists tested whether copepod populations that have a long history of exposure to toxic *Alexandrium* are adapted to this toxic prey. To do this, they raised copepods from Maine (long history of exposure to toxic *Alexandrium*) and New Jersey (no exposure to toxic *Alexandrium*) in


**Figure 2:** A photograph of a copepod (left) and the toxic alga *Alexandrium* sp. (right).

the laboratory. They raised all the copepods under the same conditions. The copepods reproduced and several generations were born in the lab (a copepod generation is only about a month). This experimental design eliminated differences in environmental influences (temperature, salinity, etc.) from where the populations were originally from.

The scientists then measured how fast the copepods were able to produce eggs, also called their egg production rate. Egg production rate is an estimate of growth and indicates how well the copepods can perform in their environment. The copepods were given either a diet of toxic *Alexandrium* or another diet that was non-toxic. If the copepods from Maine produced more eggs while eating *Alexandrium,* this would be evidence that copepods have adapted to eating the toxic algae. The non-toxic diet was a control to make sure the copepods from Maine and New Jersey produced similar amounts of eggs while eating a good food source. For example, if the copepods from New Jersey always lay fewer eggs, regardless of good or bad food, then the control would show that. Without the control, it would be impossible to tell if a difference in egg production between copepod populations was due to the toxic food or something else.

**Check for Understanding:** After reading the introduction, students should be able to:

- Describe the role of algae and copepods in the aquatic food chain.
- Describe the role that toxins play in algae defense against predators.
- Understand what happens to the toxins as they move through the food chain.
- Review the themes of evolution by natural selection. How are predators able to potentially evolve resistance to toxic algae?
- Understand why it was important for the scientists to raise the copepods in the lab for a full generation before beginning the experiment. *Because the copepods were collected from two different sites, they may have been affected by environmental differences. Growing the copepods in the lab for one generation in a common environment can eliminate these differences.*
- In their own words, describe the experimental design.

*Scientific Question*: Have the copepods with a long history of eating toxic algae evolved resistance to the toxins in this food source?

*What is the hypothesis?* Find the hypothesis in the Research Background and underline it. A hypothesis is a proposed explanation for an observation, which can then be tested with experimentation or other types of studies.

*Scientific Data:*

**Use the data below to answer the scientific question:**

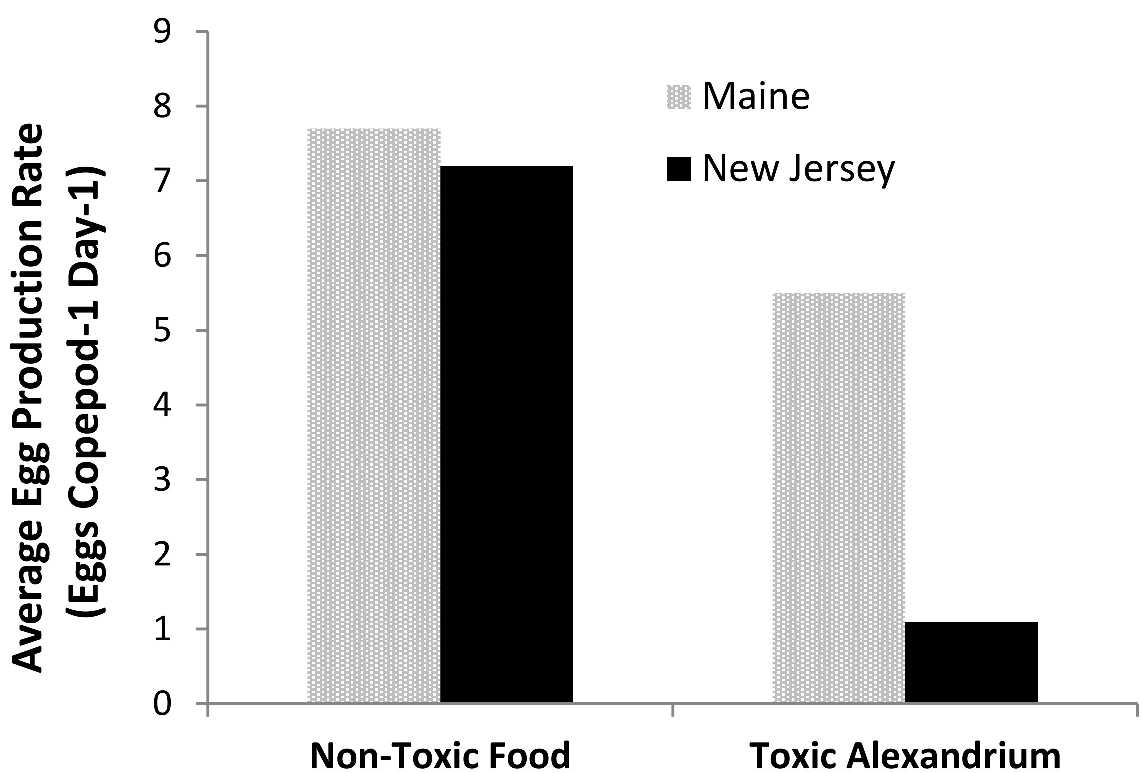
| Population | Average Egg Production Rate (Eggs/Copepod/Day) | |
|---|---|---|
| | Non-Toxic Food | Toxic Food |
| Maine | 7.7 | 5.5 |
| New Jersey | 7.2 | 1.1 |

What data will you graph to answer the question?

Independent variables: Population (Maine or New Jersey) and food type (toxic or non-toxic)

Dependent variable: Average Egg Reproduction Rate

*Draw your graph below:* Identify any changes, trends, or differences you see in your graph. Draw arrows pointing out what you see, and write one sentence describing what you see next to each arrow.



*Interpret the data*:

Make a claim that answers the scientific question.

Copepods from Maine, with a long history of exposure to toxic algae, have evolved resistance to their toxins, while copepods from New Jersey, without a history of exposure, have not.

What evidence was used to write your claim? Reference specific parts of the table or graph.

When fed non-toxic food, Maine copepods produce 7.7 eggs per day, while New Jersey copepods produce 7.2 eggs per day. When fed toxic food, Maine copepods produce 5.5 eggs per day, while New Jersey copepods produce only 1.1 eggs per day.

Explain your reasoning and why the evidence supports your claim. Connect the data back to what you learned about predators evolving resistance to toxic algae.

First, let's look at the egg production rate data on a non-toxic diet. This will help us determine if there are any differences in how many eggs are laid by the two copepod populations under normal conditions. The results show no difference. This non-toxic diet served as a control; now we can be sure if we see a difference in egg production rate on toxic food, it is not from a natural difference between the two populations.

Next, let us analyze the toxic diet and see what the data can tell us regarding adaptation. Recall, if copepods are adapted, then the toxins' negative effects are reduced. This would result in the adapted population being able to lay more eggs on a toxic diet compared to populations that are not adapted. The data align with our predictions, supporting our hypothesis. Copepods from Maine, with the long history of exposure to toxic *Alexandrium*, had much higher egg production rates compared to copepods from New Jersey, which have no experience with toxic *Alexandrium*. Maine copepods appear to have adapted to the toxins, while New Jersey copepods did not.

**Teacher Note:** It appears that adaptation may not make Maine copepods completely resistant to the toxins; compared to the toxin-free control where they produced 7.7 eggs per day, their egg production dropped to 5.5 eggs per day when fed a toxic diet.

This last finding was not statistically significant in the study, and further experimentation did not find evidence for it. However, this is an interesting point to discuss with the class and students could be asked to design an experiment that might test whether copepods with a history of exposure to toxins are fully adapted to them.

What do the data from this study tell us about Michael and Hans' hypothesis?

The data support the hypothesis that, after feeding on toxic algae for many generations, the predator populations in Maine have evolved resistance to toxic prey. Unlike populations in New Jersey that have not been exposed to the toxic algae, Maine populations were able to keep egg production high even when fed a toxic diet. However, Maine populations are still not completely resistant to the toxin and produce slightly fewer eggs than when fed a control toxin-free diet.

*Your next step as a scientist:* Science is an ongoing process. What new question do you think should be investigated? What future data should be collected to answer your question?

See following Teacher Note.

**Teacher Note:** Student responses may vary, and they will probably generate a wide diversity of questions for in this system. You can have a class discussion where you jot down all the questions up on the board. Be prepared to ask your students to clarify or justify another student's response in a class discussion. Do students see any ways to improve each other's questions? Are some questions untestable? Remember, if your class wants to send their questions about the study system to Michael and Hans, the scientists studying toxic algae, they can email them to datanuggetsk16@gmail.com!

In the research shared in this Data Nugget, Michael and Hans showed evidence that historical exposure to toxic phytoplankton led to adaptation in copepods. Their next steps are to investigate the molecular mechanisms underlying adaptation to toxic *Alexandrium*. *Alexandrium* toxins act by blocking cellular sodium channels, which causes muscle paralysis. That is why the toxins are also known to cause Paralytic Shellfish Poisoning. Clams ingest toxic *Alexandrium*, humans eat contaminated clams, and the toxins paralyze breathing muscles and humans can die from asphyxiation. In clams, adaptation to *Alexandrium* toxins is due to a mutation in the sodium channel that keeps the toxin from blocking the channel. Adapted clams can ingest vast quantities of toxic food and not be poisoned because the toxin cannot block their sodium channel. However, these clams also retain very high concentrations of the toxins in their bodies, making them more deadly to humans who consume them. Research is currently underway to see if copepods have a similar mutation in their sodium channel.