

Blue light culprit in red tide blooms

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Each year, phytoplankton blooms known as "red tides" kill millions of fish and other marine organisms and blanket vast areas of coastal water around the world. Though the precise causes of red tides remain a mystery, a team of researchers in the United States and Spain has solved one of the main riddles about these ecological disasters by uncovering the specific mechanism that triggers phytoplankton to release their powerful toxins into the environment.

"Previous theories about how phytoplankton release toxins proposed a rather awkward, untested 'exudation' mechanism," said researcher Pedro Verdugo of the University of Washington in Friday Harbor. "The true mechanism has been a very exciting riddle to crack and it provides a handle on understanding the development of huge [phytoplankton blooms](#), eventually affecting several square miles of the ocean's upper surface."

Verdugo and his colleagues, Kellie L. Vigna also of the University of Washington and Ivan Quesada of the Universidad Miguel Hernandez in Alicante, Spain, will present their research at the 56th Annual Meeting of the Biophysical Society (BPS), held Feb. 25-29 in San Diego, Calif.

Red tides appear when naturally occurring algae – including *Karenia brevis* – multiply very rapidly, becoming so concentrated that the ocean surface takes on a reddish hue. *Karenia* produces brevetoxin, a powerful neurotoxin that binds to nerve and muscle cells, leading to substantial marine life mortality and human morbidity. The blooms are triggered by some as yet unknown fluctuations in ocean temperature, salinity, and available nutrients.

The researchers discovered that *Karenia* and other unicellular microalgae function very much like the secretory cells we have in our bodies. Namely, they store inside membrane-lined microscopic vesicles their active chemicals – such as hormones, antibacterial products, and, in *Karenia's* case, toxins. When properly stimulated, these cells release their cargo by a process known as exocytosis.

Secretory cells store high concentrations of active chemicals in their vesicles by "caging" them in a gel matrix, as Verdugo's lab discovered more than a decade ago. This trick offers a clever thermodynamic advantage as storage across membrane-lined vesicles would otherwise require large amounts of osmotic work. According to the researchers, these microscopic gels found inside virtually all secretory vesicles remain in a condensed gel phase – with their cargo virtually immobilized – until they are released from the cell, when they undergo drastic swelling and release their payload. "Swelling results from a polymer gel phase transition, a characteristic property of both natural and synthetic polymer gels, which has been further applied in our lab to engineer high payload drug delivery vesicles," said Verdugo.

The cargo in phytoplankton vesicles are toxins. They are caged in a gel matrix made up of a biopolymer very similar to alginate, one of the constituents of [algae](#) cell walls. The researchers discovered that phytoplankton release their toxin-loaded gels when exposed to sunlight, particularly the blue portion of the spectrum.

"We do not know why phytoplankton respond to [blue light](#), but it might be associated with the fact that blue light penetrates deeper in seawater," said Verdugo. "Often, plants and animals release toxins as a defense mechanism. Whether this is the case in phytoplankton remains speculative. However, blue light stimulation implies that these cells must have a photoreceptor – most likely associated with the cell structures known as chloroplasts, which are responsible for photosynthesis. This is

in fact one of the riddles we'll tackle next."

These observations support the notion that *Karenia brevis* functions as a typical secretory cell, which the researchers believe opens the way to a better understanding of red tide bloom dynamics.

More information: The presentation, "Exocytic mechanisms of storage and release of brevetoxin in the dinoflagellate *Karenia brevis*," is at 1:45 p.m. on Monday, Feb. 27, 2012, in the San Diego Convention Center, Hall FGH. ABSTRACT: <http://tinyurl.com/6nuttzl>

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