Understanding why cyanobacteria are successful: their ecological strategies, unintended consequences and monitoring considerations

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USGS

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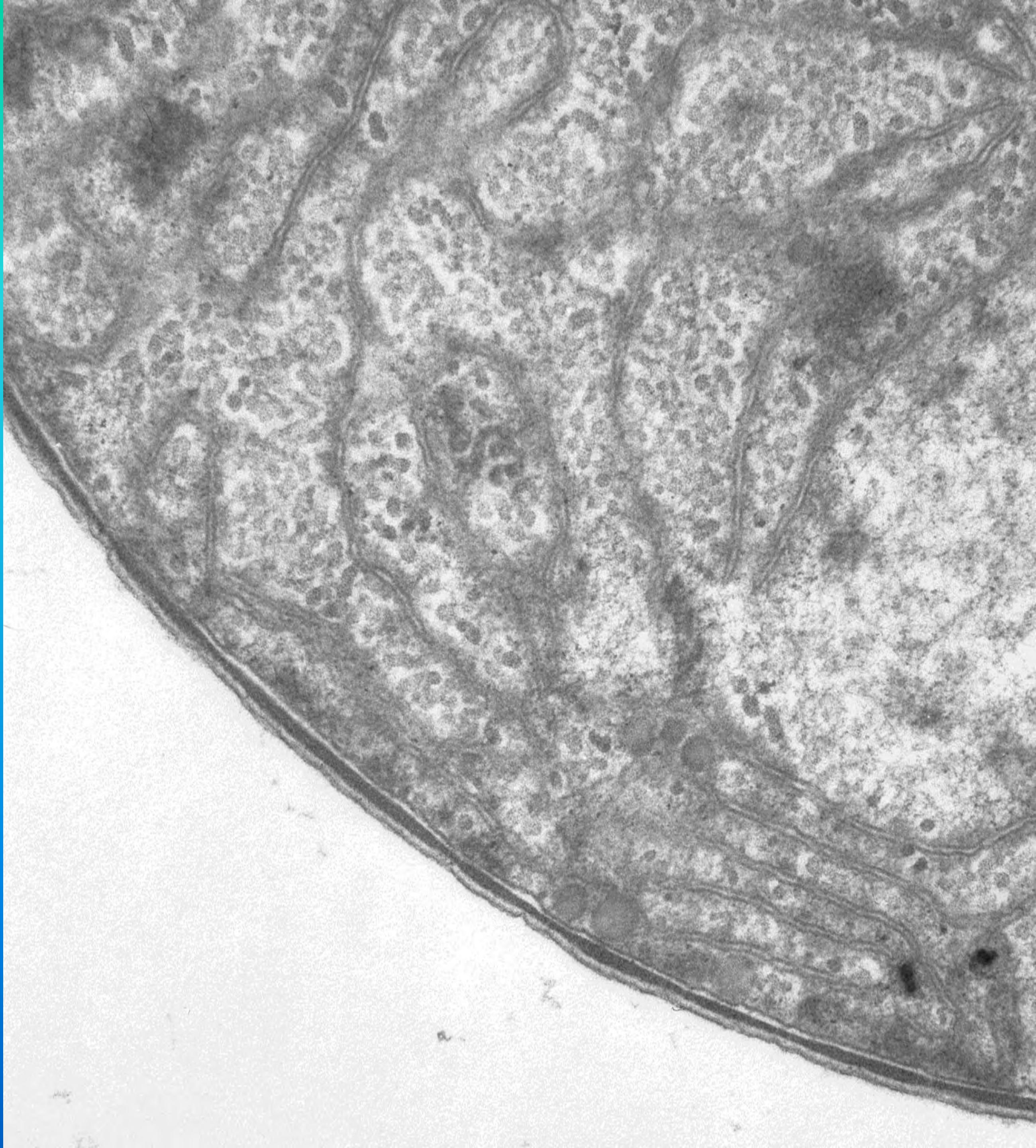
Cyanobacteria

- gram negative
- thylakoids



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Why are we concerned about cyanoHABs?

Toxicity

Hypoxia

Taste and odors

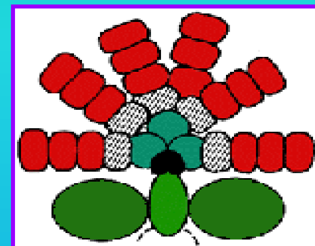
Aesthetics

Ecological strategies for cyanobacteria

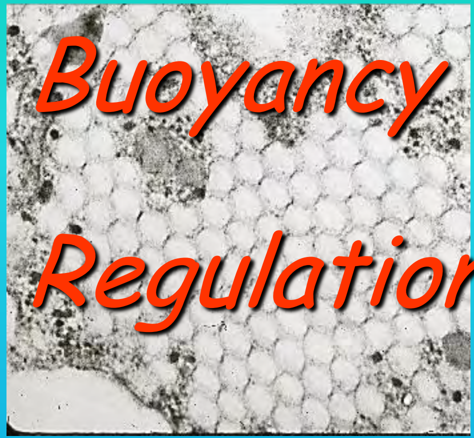
Morphology

grazing, floating

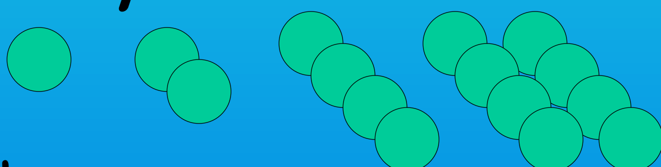
Pigments



Buoyancy Regulation

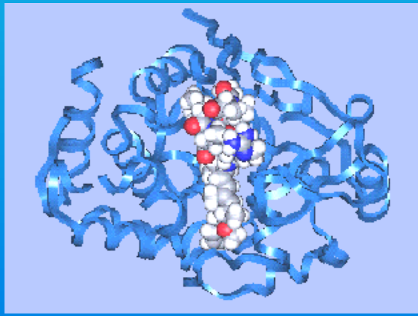


Rapid Growth

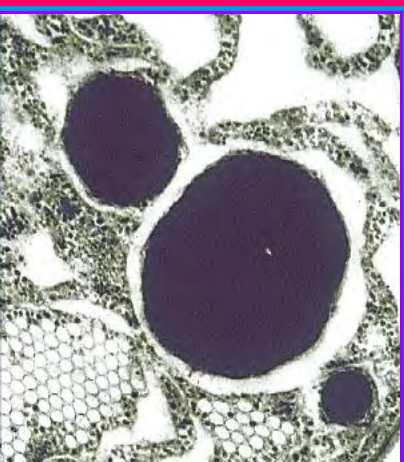


temp

Toxicity



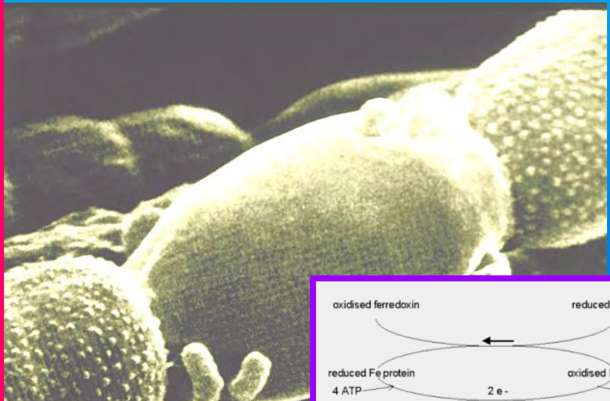
microcystin
LR complex

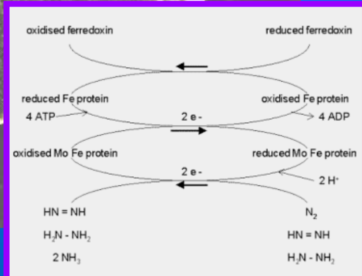


trace, P,
C, N,

Nutrient Storage

Nitrogen Fixation





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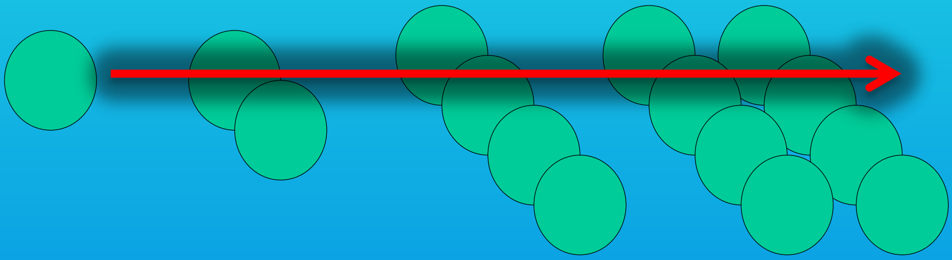
Ecological Strategies: bacteria in a eukaryotic world-thermophiles grow faster

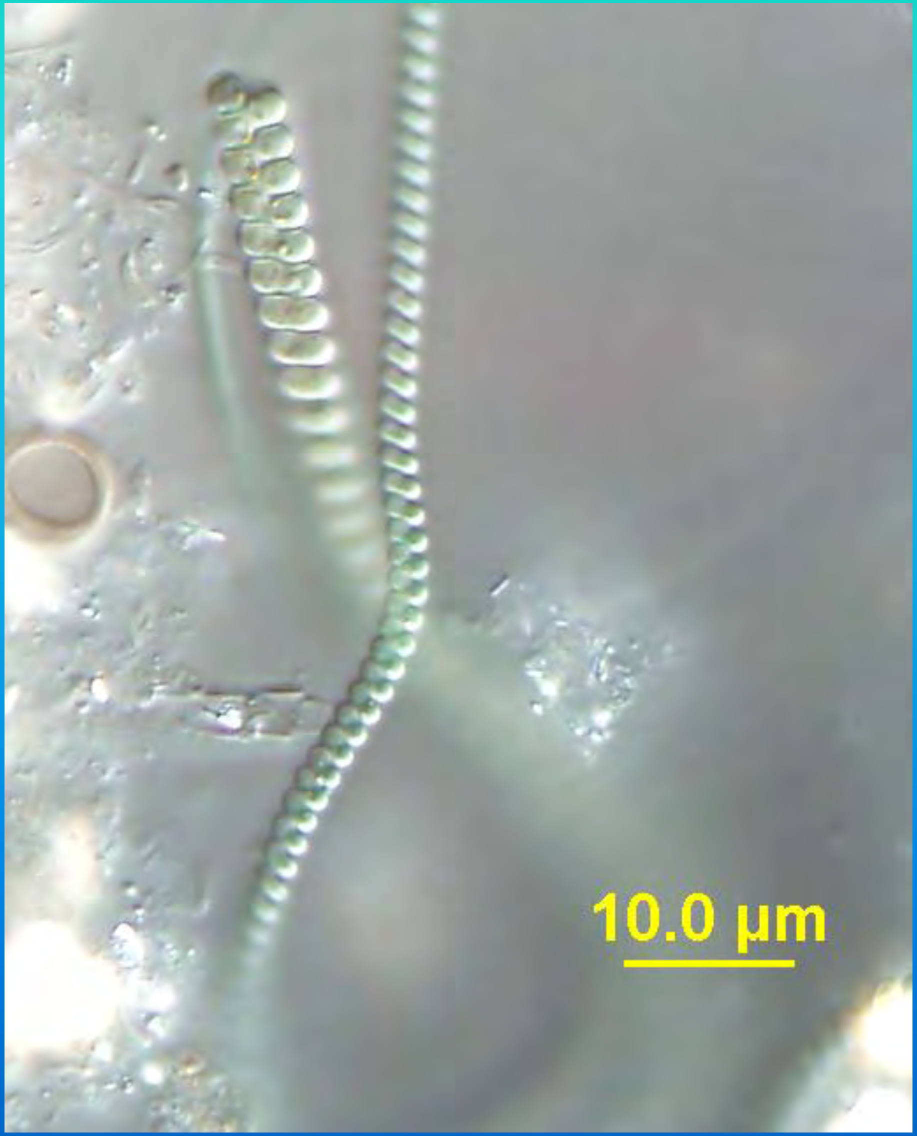
Rapid Growth

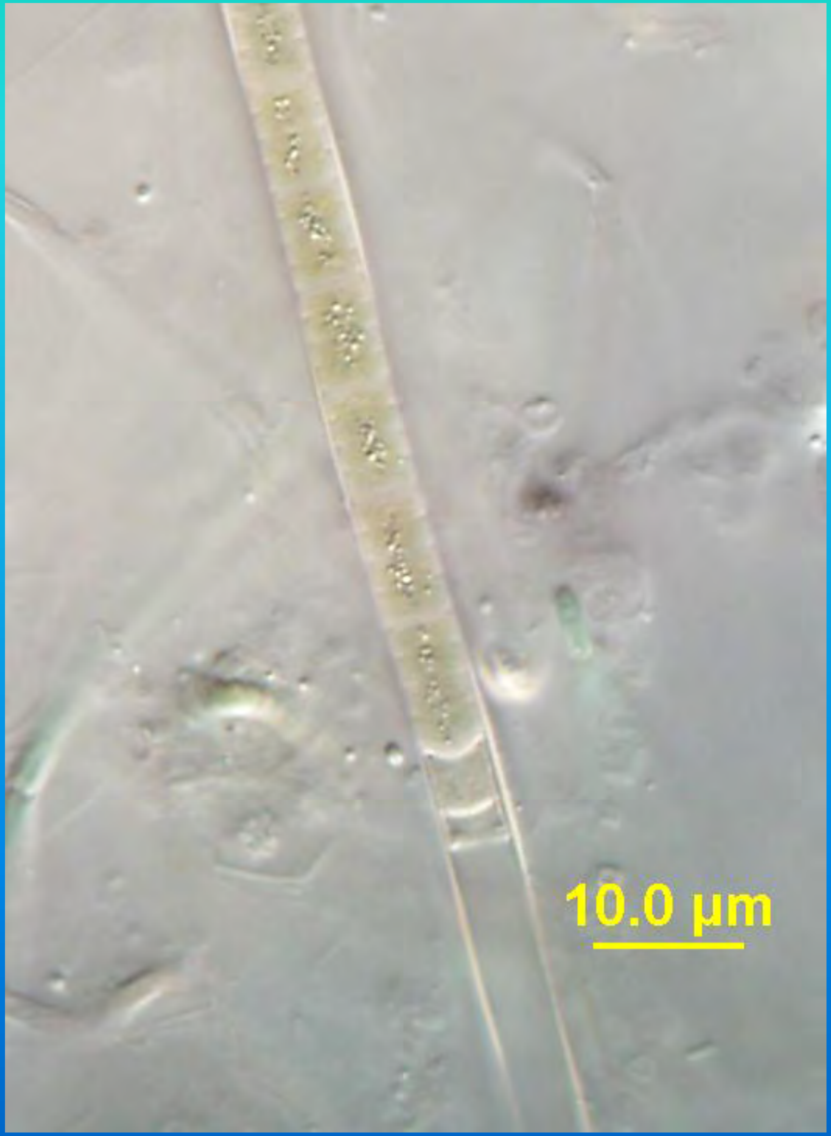


temperature

3 “doublings” or divisions

Ecological Strategies: Motility





Ecological Strategies: morphology for staying in the water column

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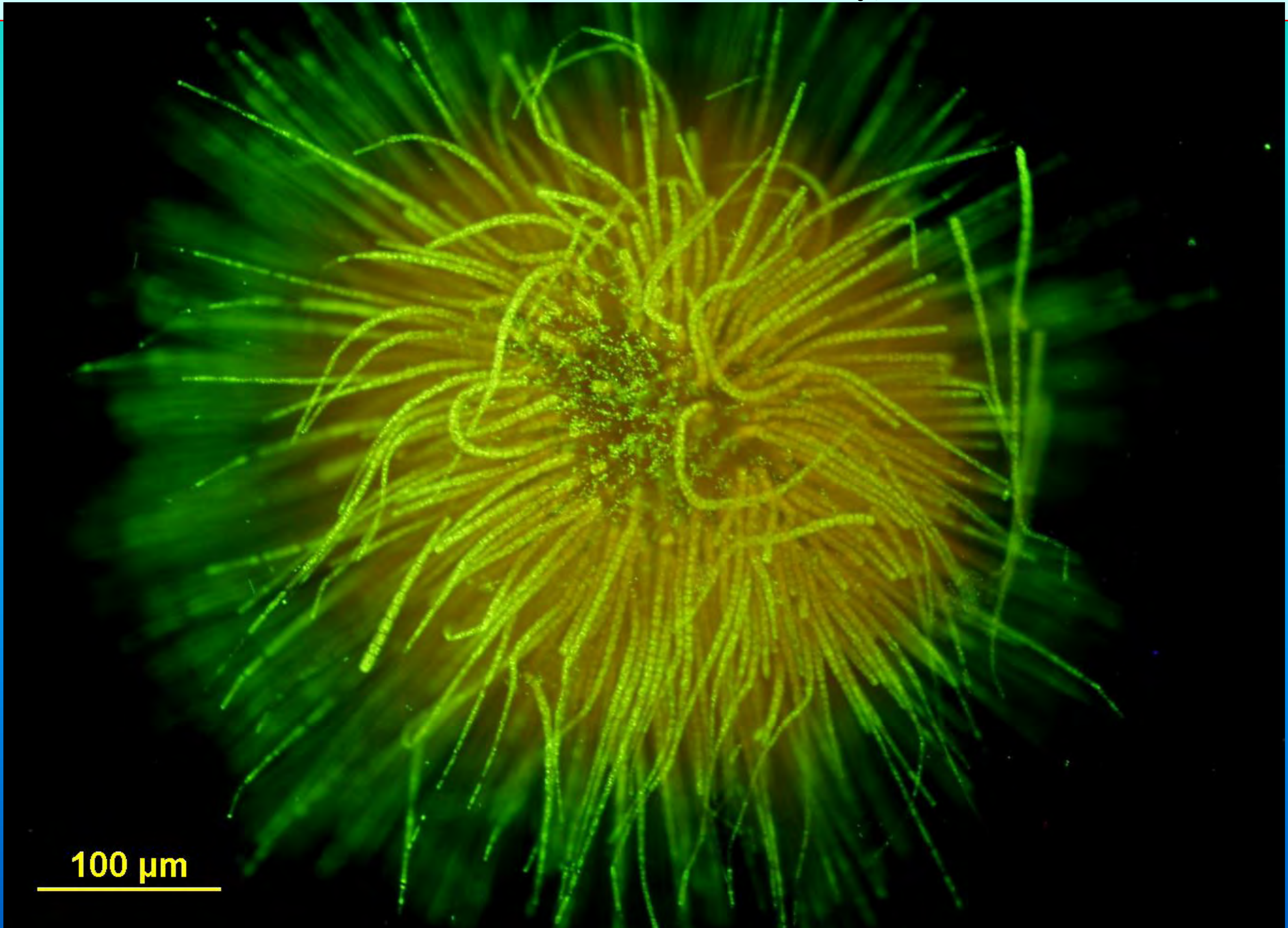
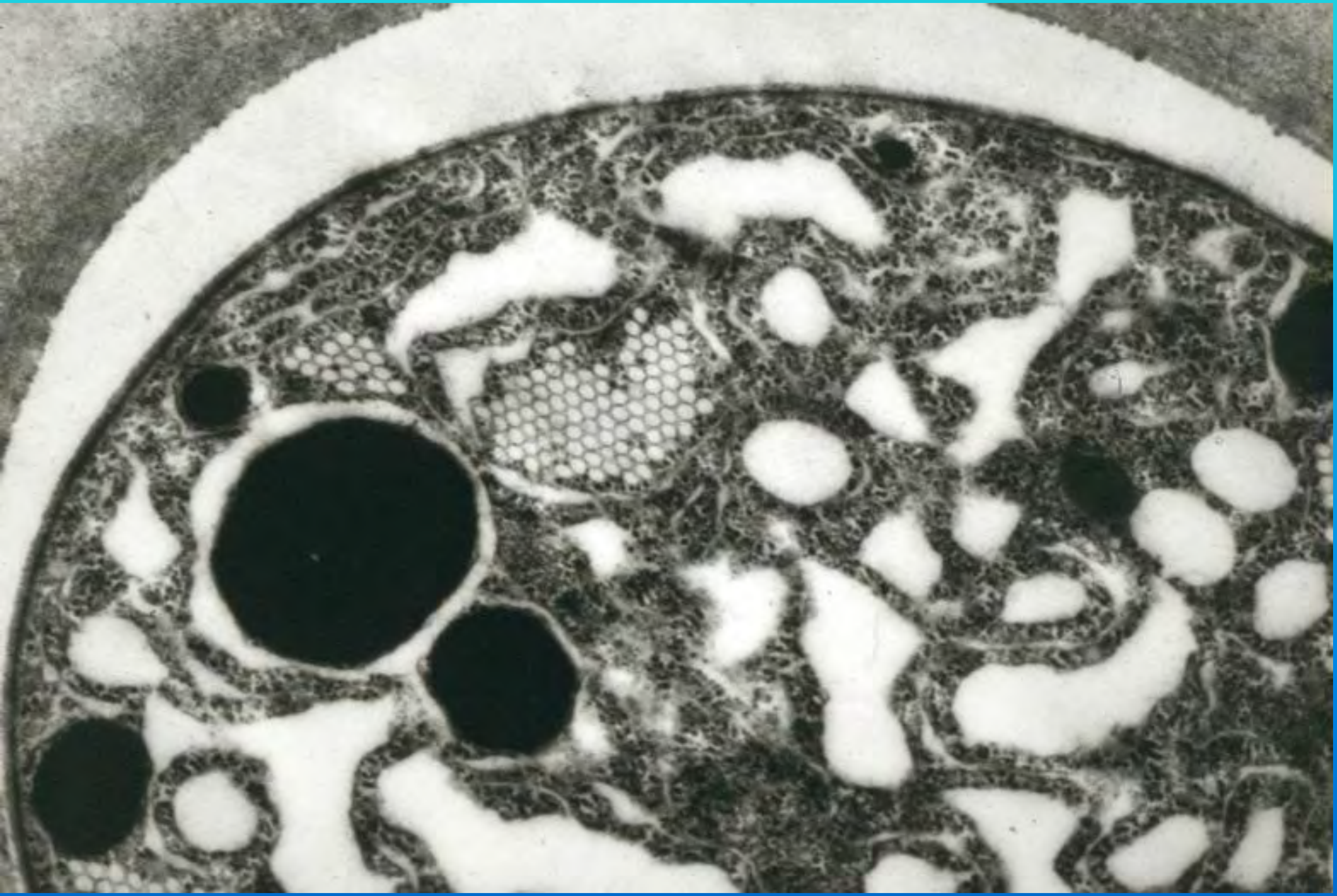


Ecological Strategies: internal structures for optimizing placement in the water column

Gas Vesicles: *Buoyancy regulation and vertical migration*

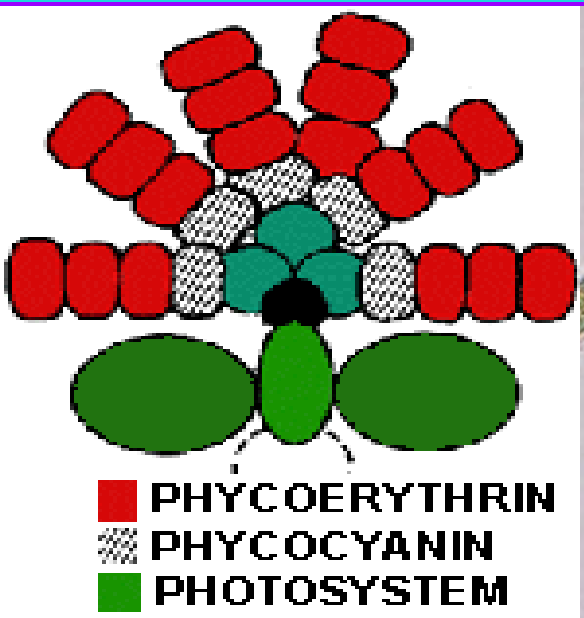


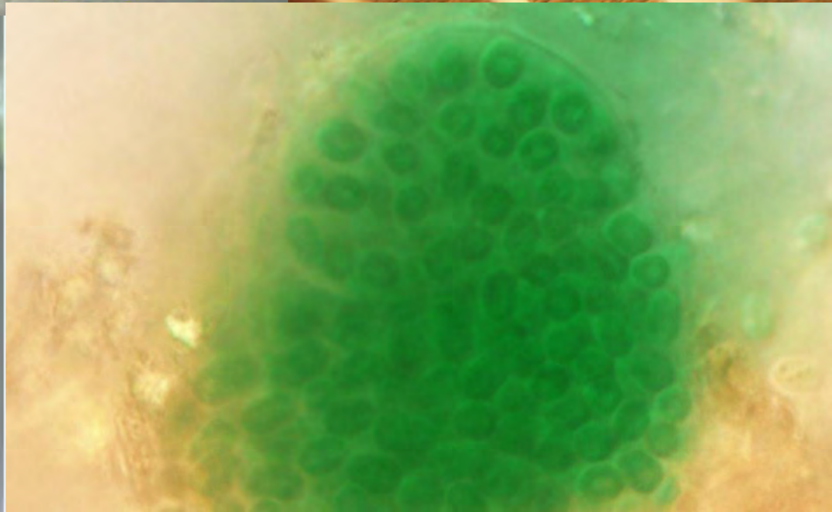
Low light

$(C_6H_{12}O_6)n$

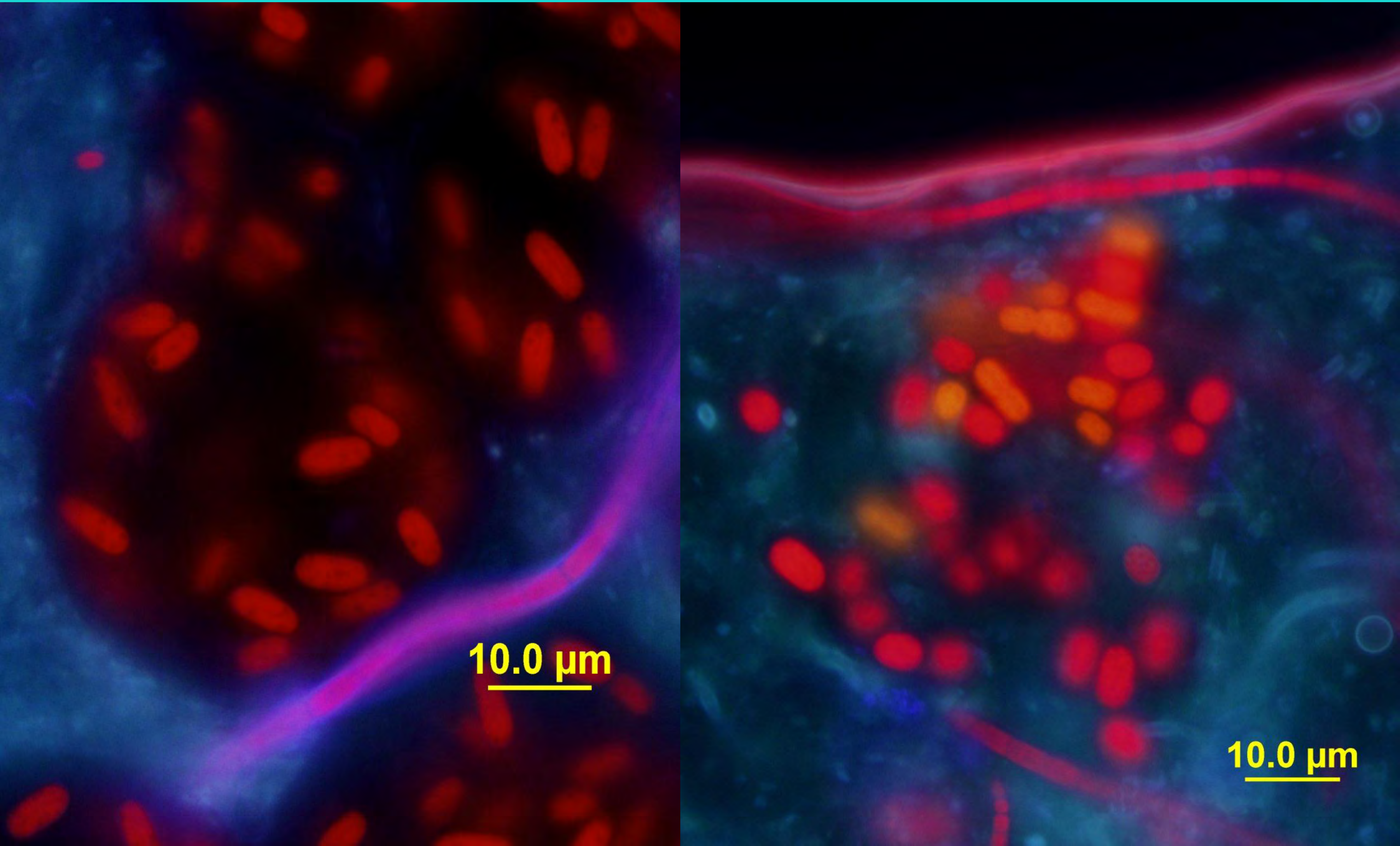
Nutrients scavenged whilst near lake sediments or thermocline

Ecological Strategies: complimentary pigments for maximizing photosynthesis

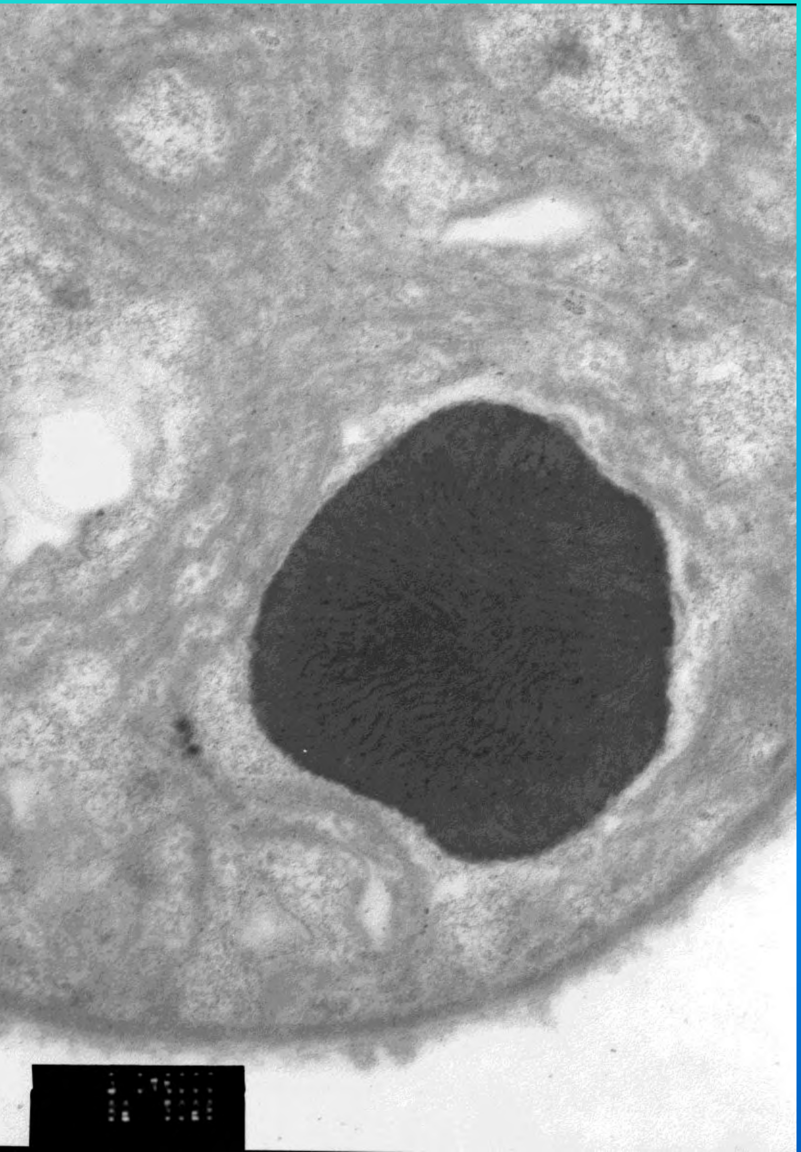




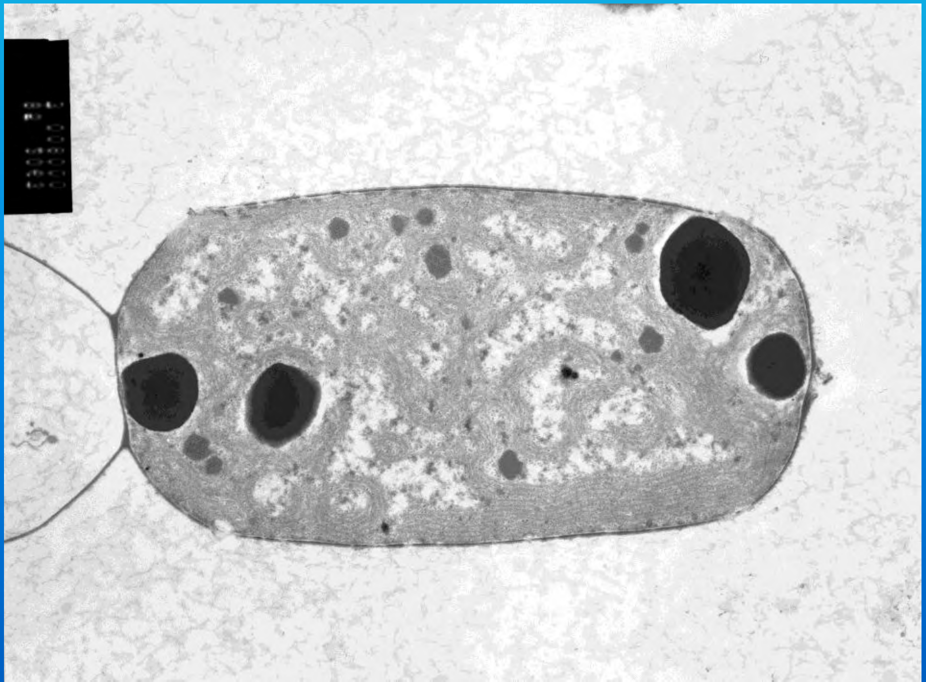
Ecological Strategies: complimentary pigments for maximizing photosynthesis



Ecological Strategies: luxuriant nutrient uptake and storage & metal sequestration

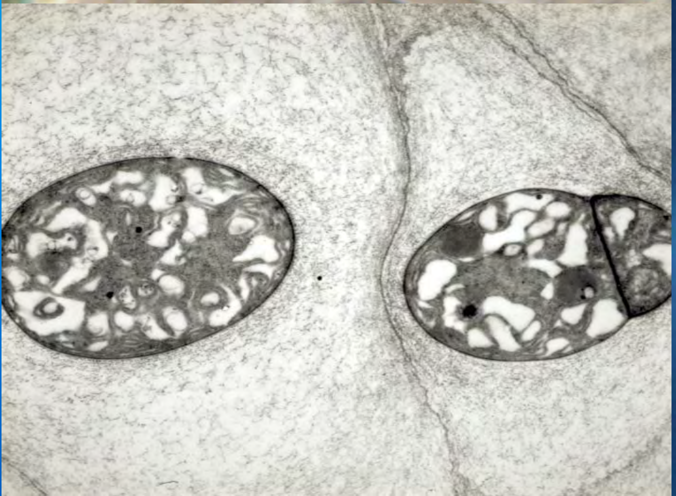


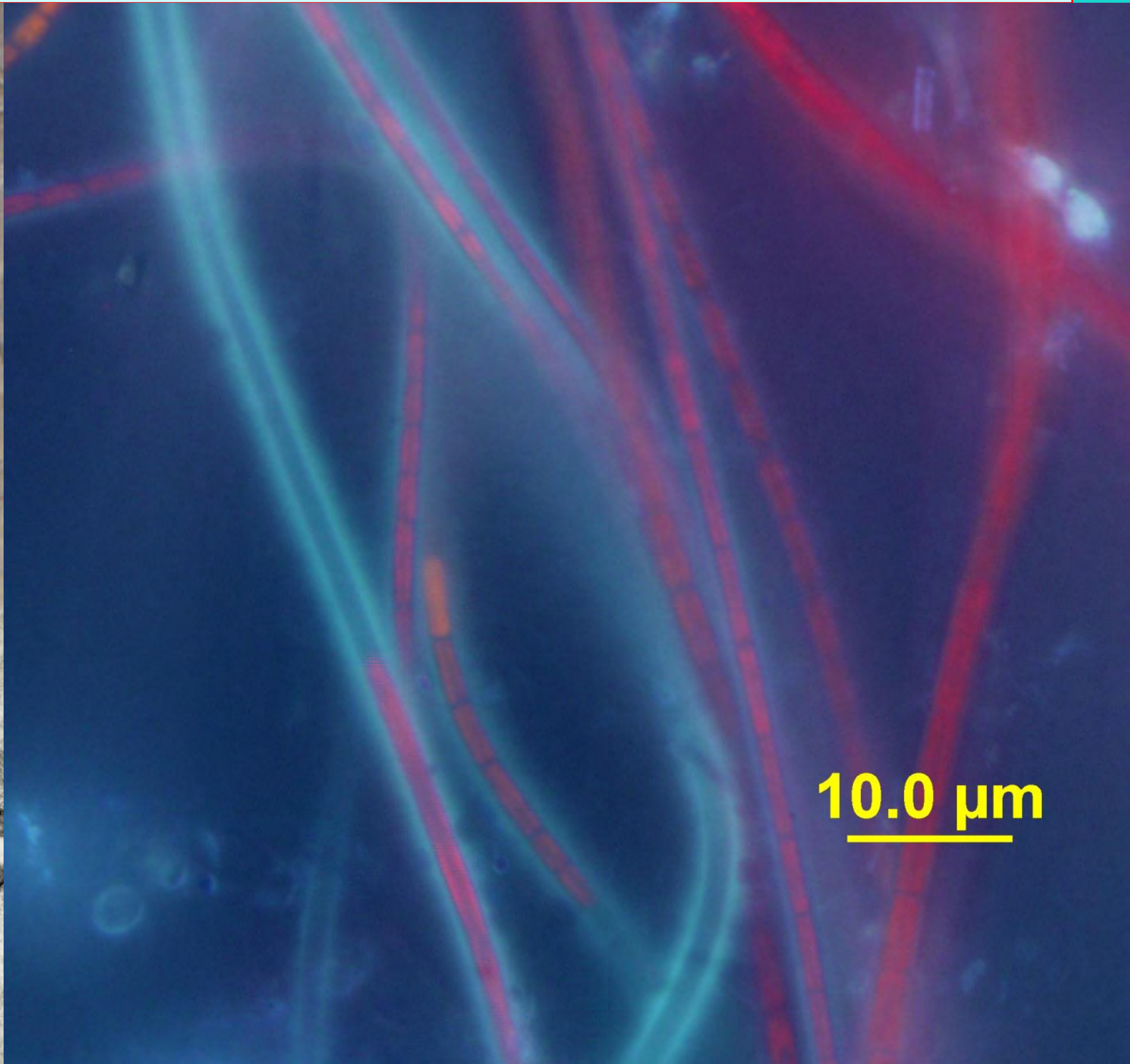
- Contain protein, lipids, polyP
- Na, Mg, Ca, K, Mn, Fe, Cu



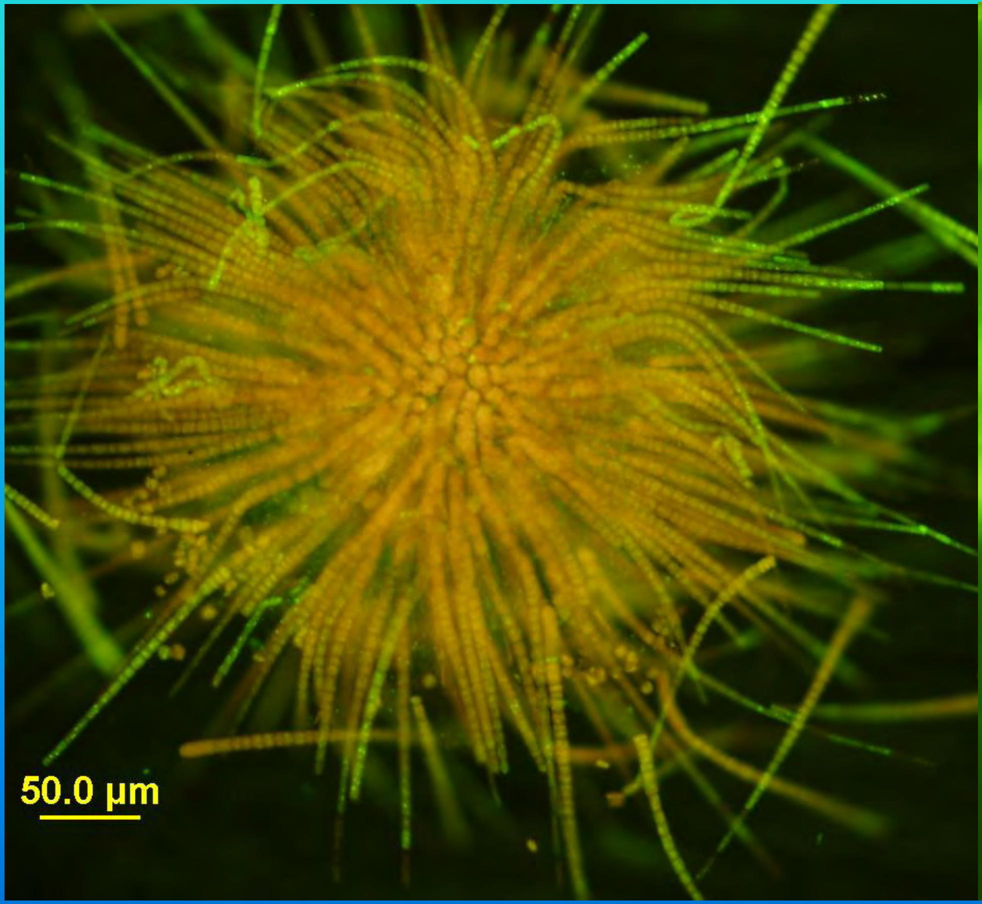
Ecological Strategies: make your own nitrogen source

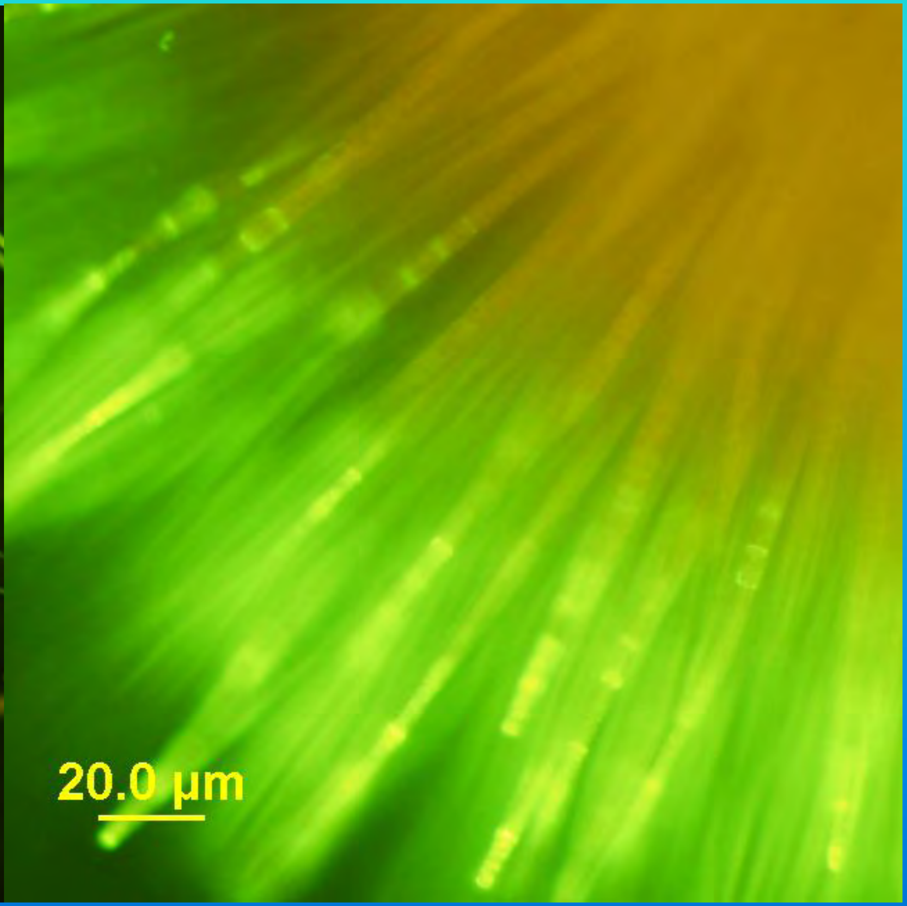
Ecological Strategies: desiccation tolerant (polysaccharide sheath-often pigmented)



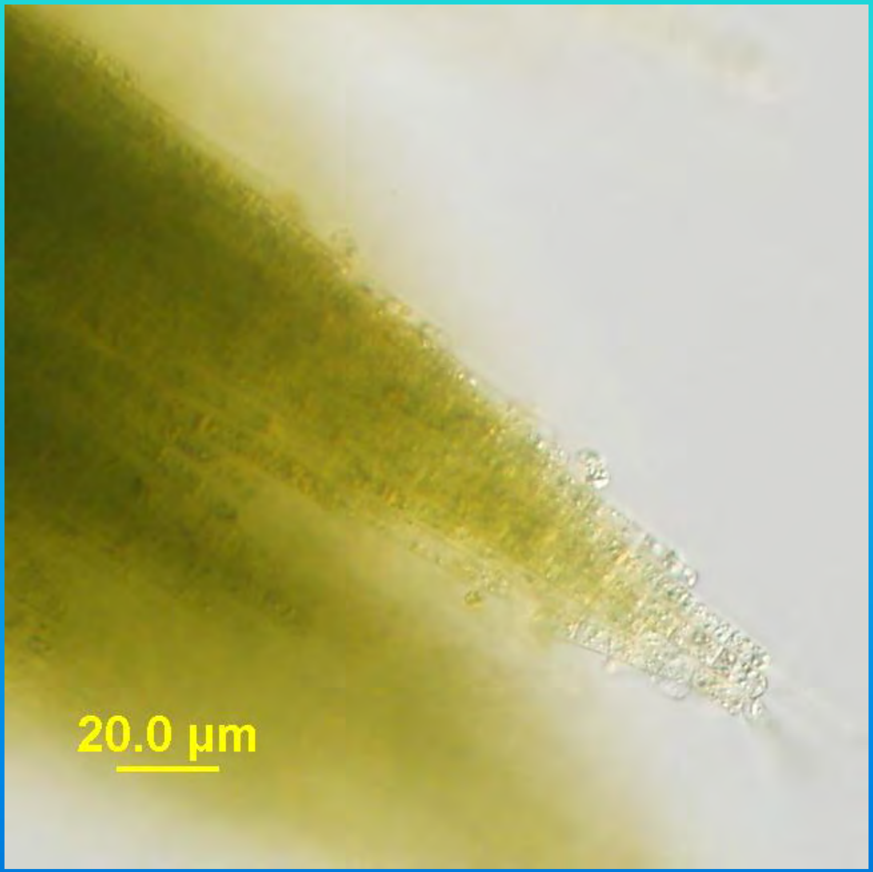


Ecological Strategies: morphology to prevent grazing





Ecological Strategies: morphology to prevent grazing



Ecological Strategies: Toxins, chemical defense?

Only a few examples:

- increase in cell toxin content when exposed (Jang et al. 2007)
- some zooplankton inhibit or cease feeding (Haney 1987; Lampert 1987)
- some zooplankton exhibit physiological resistance (Kurmayer and Juttner 1999)
- some just avoid the toxin producers

Key toxin-producing organisms: a diverse group

Unicellular forms

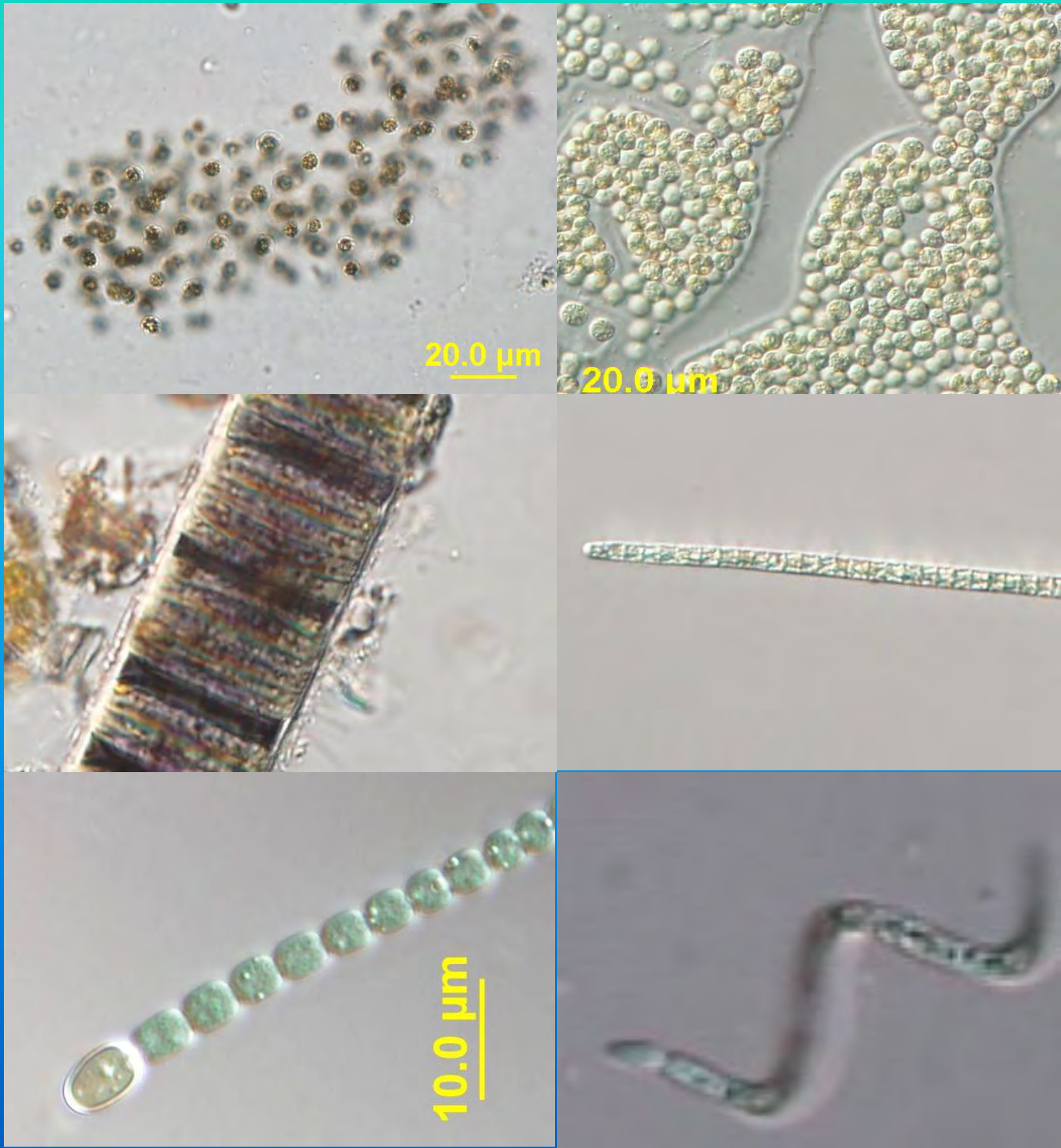
Microcystis

Filamentous

Lyngbya,
Oscillatoria
Planktothrix

Filamentous

Anabaena
Aphanizomenon
Cylindrospermopsis
Nodularia



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Occurrence and health significance of cyanotoxins

- Microcystins - most common, widespread poisonings
- Anatoxins - common; many animal poisonings
- Cylindrospermopsins - common; poisonings Australia
- Nodularin – world-wide in brackish water
- Lyngbyatoxins - probably in continental US;
  poisonings in South & Central Pacific
- Saxitoxins - sporadic; animal deaths
- *beta*-methylamino-L-alanine- BMAA - world wide;
  potential major health significance
- LPS - world wide; health significance unclear

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Cyanotoxins are highly potent

Compounds & LD_{50} (ug/kg)

Saxitoxin	9	Ricin	0.02
Anatoxin-a(s)	20	Cobra toxin	20
Microcystin LR	50	Curare	500
Anatoxin-a	200-250	Strychnine	2000
Nodularin	50		
Cylindrospermopsins	200		

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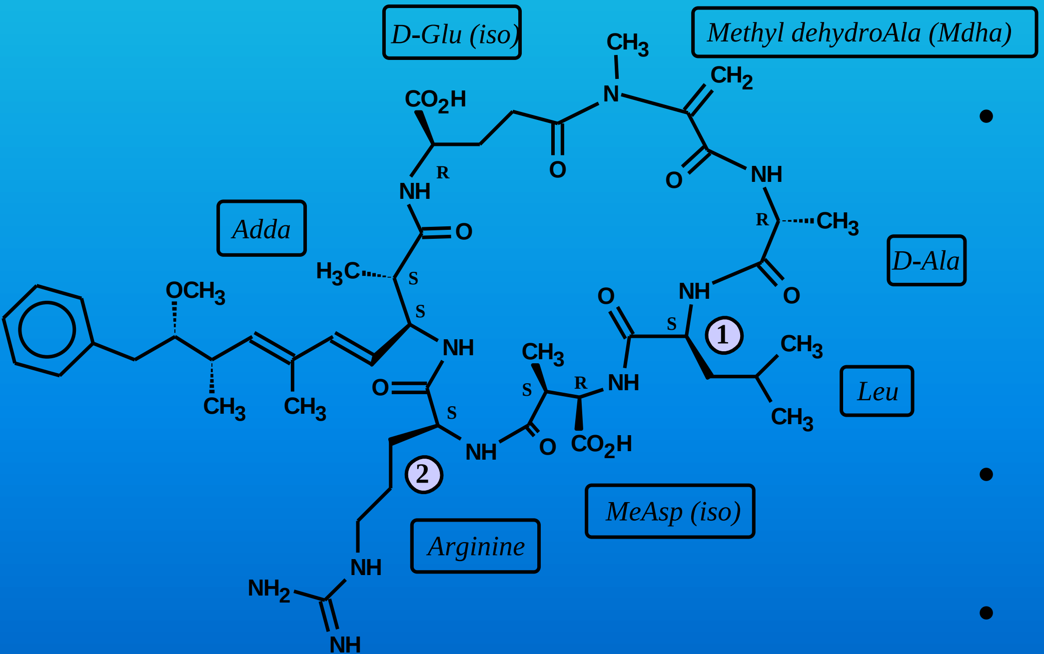
Microcystins

- *Microcystis aeruginosa*
- non-N fixer
- Very common
  - Also produced by a number of other species.

- Peptide Toxins:
  - 90+ structural variants
  - + 200 others related compounds: nodularins, anabaenapeptins, etc.

- Microcystins are hepatotoxic
  - LD-50: 25-60 μg kg^{-1}
- Called **“fast death factor”**
  - Potent tumor promotor



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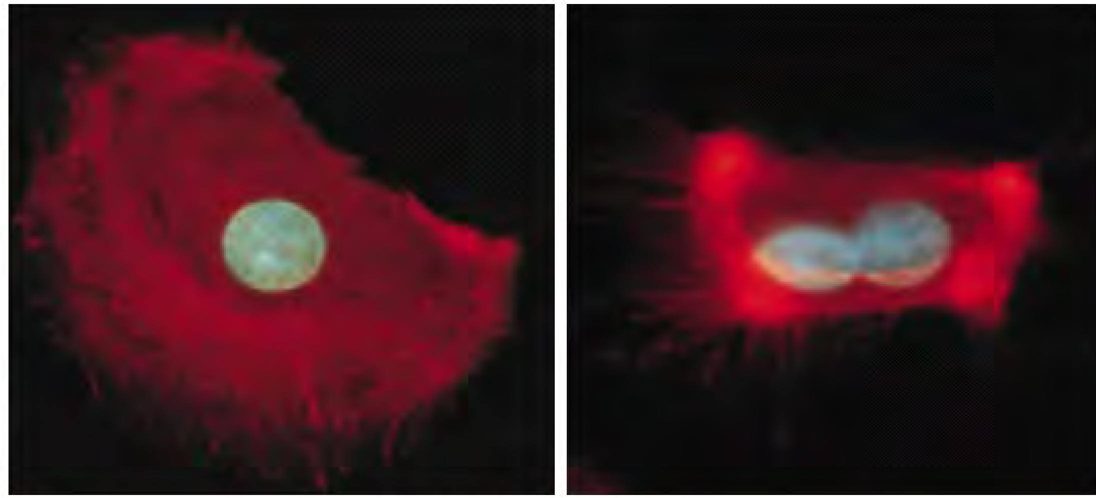
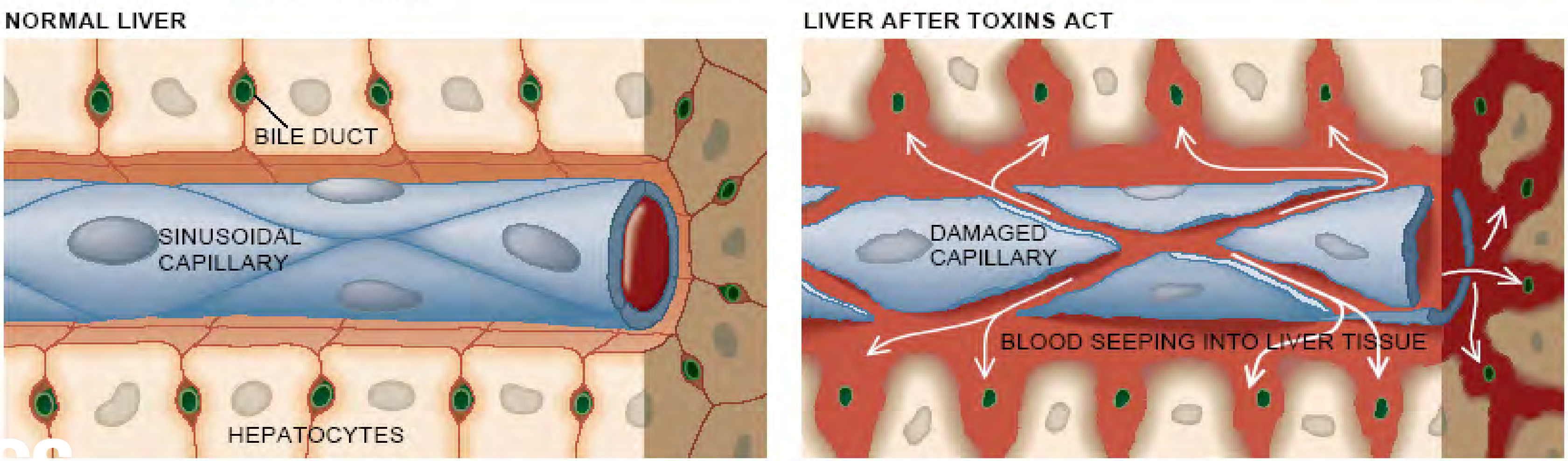
Microcystin exposure: response

- Uptake by bile acid transporter
- Inhibit protein phosphatases 1 and 2A
- Affects cytoskeleton, cell cycle, general metabolism, apoptosis

Hepatotoxicity

MICROFILAMENTS (*red threads in micrographs*), structural components of cells, are usually quite long, as in the rat hepatocyte at the left. But after exposure to microcystins (*right*), microfilaments collapse toward the nucleus (*blue*). (This cell, like many healthy hepatocytes, happens to have two nuclei.) Such collapse helps to shrink hepatocytes—which normally touch one another and touch sinusoidal capillaries (*left drawing*). Then the shrunken cells separate from one another and from the sinusoids (*right drawing*). The cells of the sinusoids separate as well, causing blood to spill into liver tissue. This bleeding can lead swiftly to death.





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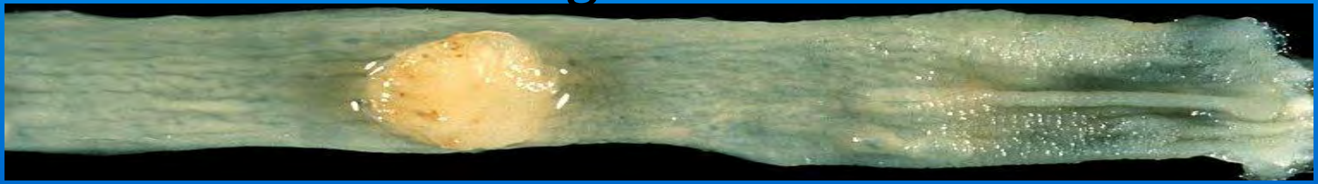
Wayne Carmichael ISOC-HAB Ch. 4, Scientific American, January, 1994

Microcystin exposure: tumor promotion

- Epidemiology in China:
  - Contaminated drinking water ↔ primary liver and colon cancer.
- Injection of toxin ± initiator:
  - Increased size/number of liver cancer precursors.
- Oral *M. aeruginosa.* extract:
  - Skin papillomas larger/heavier
  - No effect on duodenal tumours or lymphoma.

Colon cancer precursors larger

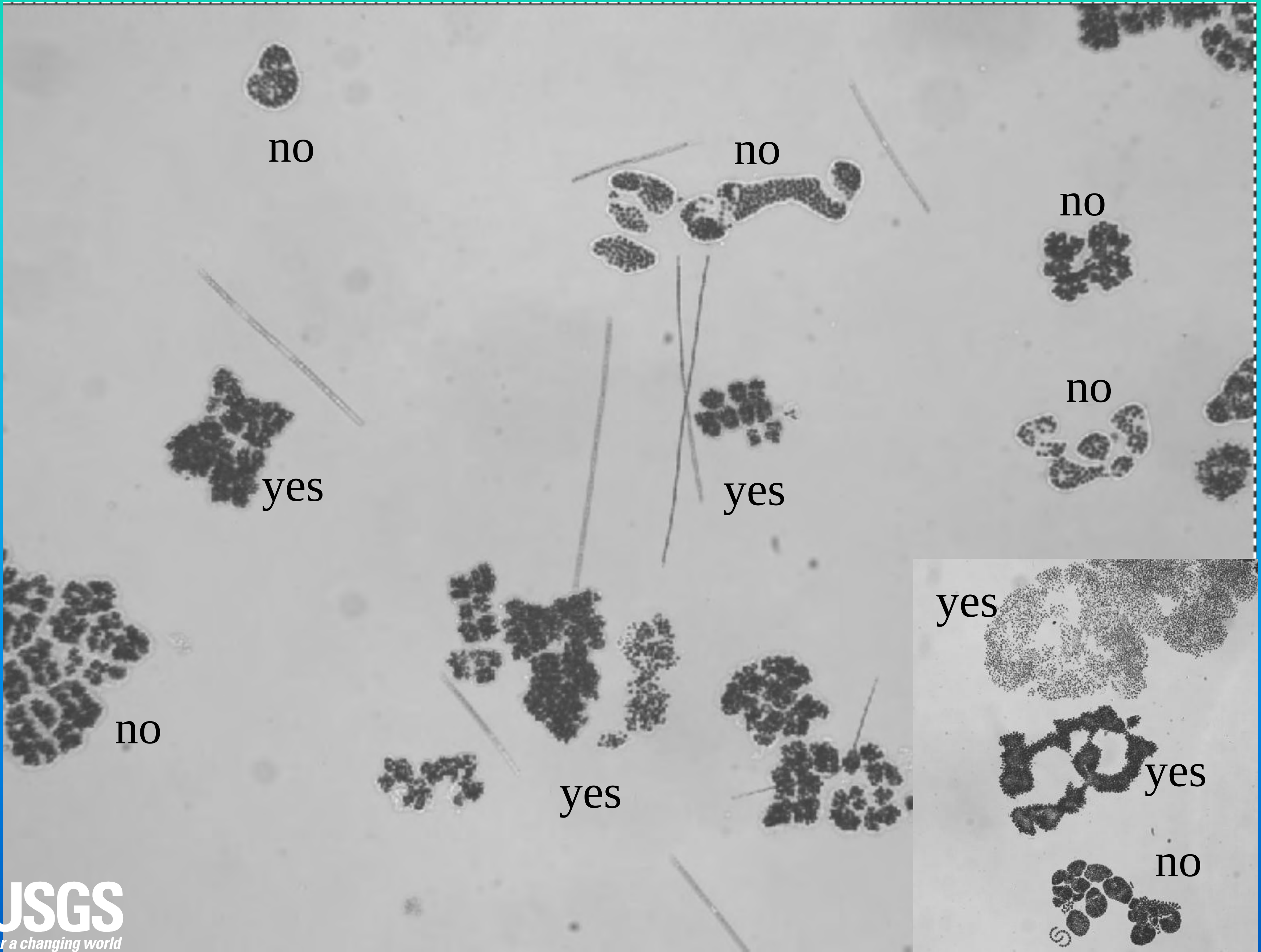


Andrew Humpage ISOC-HAB Ch. 16

Microsystin-producing strains include:

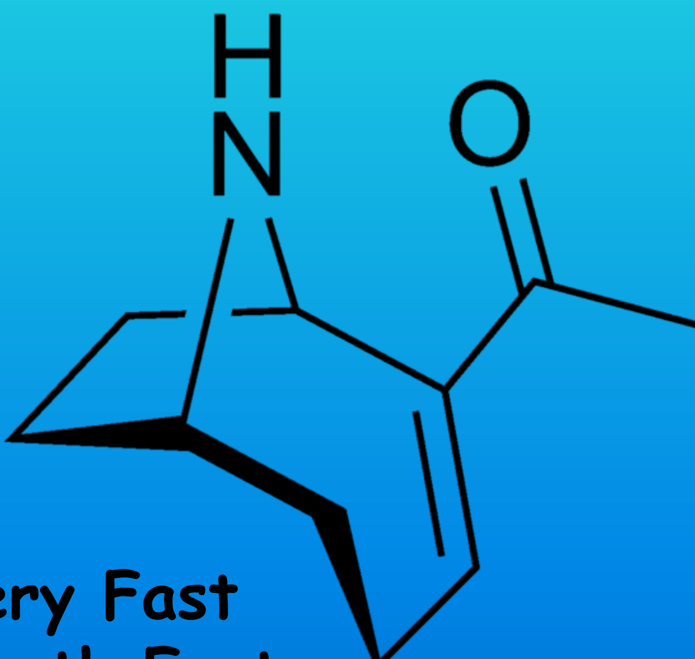
- *Microcystis aeruginosa*
- *M. wesenbergii*
- *M. botrys*
- *Oscillatoria limosa*
- *Anabaena flos-aquae*
- *A. lemmermannii*
- *A. circinalis*
- *Planktothrix agardhii*
- *P. mougeotii*
- *Nostoc spumigena*
- *N. species*
- *Anabaenopsis millerii*
- *Haphalosiphon hibermicus*
- *Gloeotrichia sp.*

Can not use taxonomy to predict toxicity



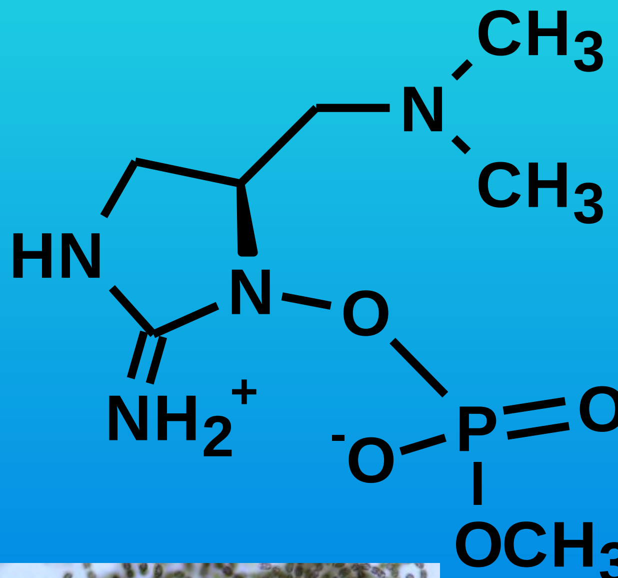
Anatoxins

Anatoxin-a
actylcholine agonist

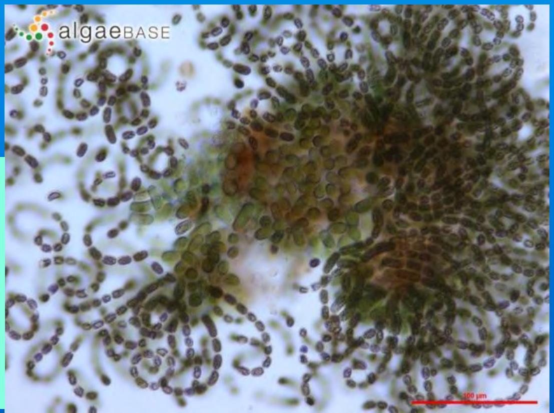


Very Fast
Death Factor

Anatoxin-a(S)
acetylcholinesterase inhibitor



Anabaena flos-aquae & lemmermannii



Anatoxin-producing strains include:

- *Anabaena circinalis*
- *A. flos-aquae*
- *A. planctonica*
- *Planktothrix sp.*
- *Aphanizomenon flos-aquae*
- *A. ovalisporum*
- *Cylindrospermopsis raciborskii*

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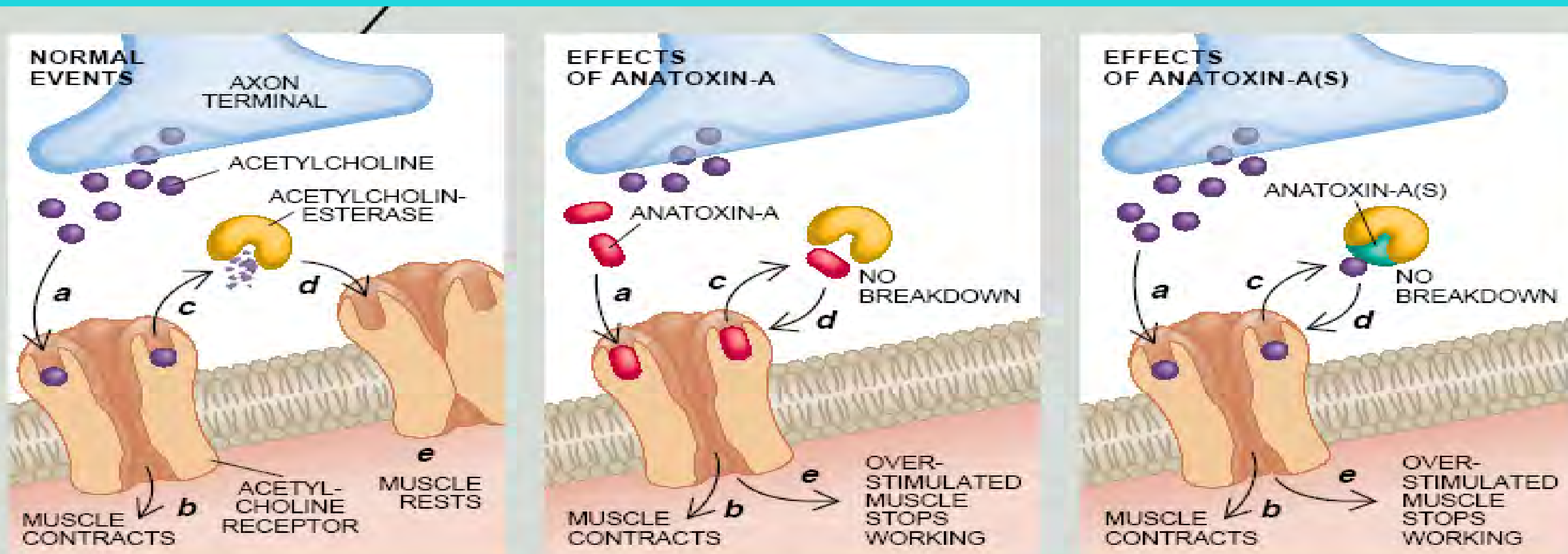
Anatoxin-a and a(s)

Anabaena

Anatoxin-a: Acetylcholine receptor agonist
Anatoxin-a(s): Acetylcholinesterase inhibitor

Neurotoxicity

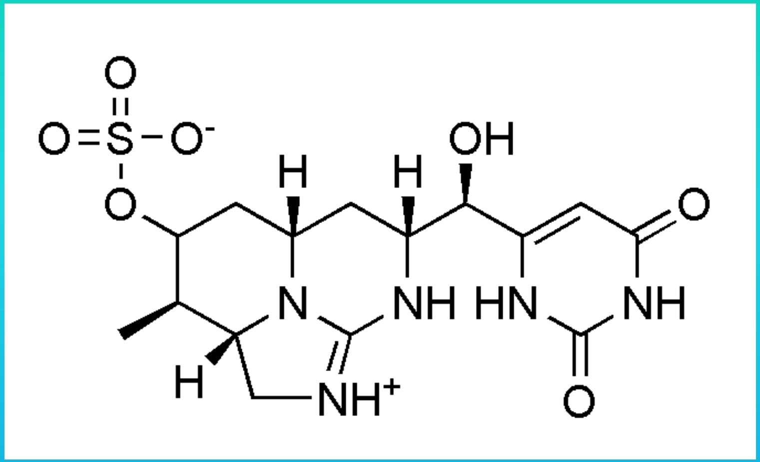


Anatoxin-a and anatoxin-a(s) (*center and right panels*) overexcite muscle cells by disrupting the functioning of the neurotransmitter acetylcholine. Normally, acetylcholine molecules (*purple*) bind to acetylcholine receptors on muscle cells (*a in left panel*), thereby inducing the cells to contract (*b*). Then the enzyme acetylcholinesterase (*yellow*) degrades acetylcholine (*c*), allowing its receptors and hence the muscle cells to return to their resting state (*d* and *e*). Anatoxin-a (*red in center panel*) is a mimic of acetylcholine. It, too, binds to acetylcholine receptors (*a*), triggering contraction (*b*), but it cannot be degraded by acetylcholinesterase (*c*). Consequently, it continues to act on muscle cells (*d*). The cells then become so exhausted from contracting that they stop operating (*e*). Anatoxin-a(s) (*green in right panel*) acts more indirectly. It allows acetylcholine to bind to its receptors and induce contraction as usual (*a* and *b*), but it blocks acetylcholinesterase from degrading acetylcholine (*c*). As a result, the neurotransmitter persists and overstimulates respiratory muscles (*d*), which once again eventually become too fatigued to operate (*e*).

Wayne Carmichael ISOC-HAB Ch. 4, Scientific American, January, 1994

Cylindrospermopsin

Cylindrospermopsis



Alkaloid Toxin

- Gastrointestinal effects
- Hepatotoxicity
- Liver necrosis
- Kidney effects
- Inhibition of protein synthesis

- Covalently modify DNA and/or RNA
- Resistant to degradation by pH and temp-persistent

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WARNING
LIFEGUARD,
BACTERIOLOGIST,
MICROBIOLOGIST,
& TOXICOLOGIST
NOT ON DUTY
SWIM AT YOUR
OWN RISK

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