Toxic Cyanobacteria:

What should I know ?

Information for medical professionals on recreational exposure to toxic cyanobacterial cells and substances

Could my patients be ill from exposure to this ?

Cyanobacteria, commonly called „blue-green algae“, are unicellular organisms often growing in colonies or filaments. In some water-bodies, they proliferate extremely, causing high turbidity or scums as shown on these photos. They contain a large variety of bioactive substances, and toxic effects on humans are understood only for some. A few are currently studied as potential pharmaceuticals; others – the ”cyanotoxins” – are poisonous.

Umwelt Bundes Amt
Für Mensch und Umwelt
Contact: www.umweltbundesamt.de

What are Cyanotoxins ?

Heptatotoxic peptides (microcystins and nodularins) occur very frequently in eutrophic fresh or brackish waters with cyanobacterial mass developments, including the Baltic Sea. Bile acid carriers transport them primarily into liver cells, but also into intestinal cells, where they block protein phosphatases 1 and 2a.

Neurotoxins from cyanobacteria include alkaloids such as anatoxin-a (nicotonic agonist) saxitoxins (which block neuronal sodium ion channels) and an organo-phosphate (blocks acetylcholinesterase). They are found less frequently, though occasionally in concentrations high enough for acutely lethal intoxication of pets, livestock and wildlife after oral uptake.

Cylindrospermopsin is a cytotoxic alkaloid (inhibits protein synthesis) affecting liver, kidney and other organs. It also appears to occur quite frequently in some regions, though to date little is known about its distribution.

Microcystins and nodularins largely remain contained within cyanobacterial cells; the alkaloids and cylindrospermopsin are also dissolved in the water.

How Dangerous are Cyanotoxins ?

Estimates show that in extreme cases, they can be acutely lethal if a small child swallows about a cup full of ”pea soup” type highly toxic bloom.

In most cases, concentrations are not high enough for a risk of acute poisoning. However, flu-like symptoms such as abdominal pain, nausea, seizures and spasms, diarrhoea, vomiting, irritation of eyes, ears or throat may occur after recreational exposure to water with a high density of cyanobacterial cells, particularly after ingestion or aspiration of larger amounts (> 100ml) of such water.

So far, only very few cases of human intoxication have been clearly demonstrated. Fatalities have not been reported from recreational exposure (although from cyanotoxins in dialysis water), but toxicological information and animal intoxications indicate subacute poisoning to be likely. Cases may well go undetected due lack of patient reporting (due to the minor severity of symptoms) or physician misdiagnosis (due to lack of awareness of this hazard.

Dermal uptake is minor for microcystins and nodularins – dangerous exposure is oral or through aspiration. Skin irritation reported from exposure to cyanobacteria is probably due to cell wall components.

There is limited evidence for carcinogenicity of micro-cystins, nodularins and cylindrospermopsin. Health risks (e.g. liver damage) may occur by repeated exposure to subacute concentrations.

The WHO gives a provisional Guideline value for one variant of microcystins in Drinking-water. § 8 of the EU Bathing-water Directive requires assessment of risk from toxic cyanobacteria. An increasing number of countries has implemented guidance and/or regulations on toxic cyanobacteria.

Patients in my practice ?

Increasing awareness is likely to result in more patients calling into doctors’ practices after exposure to cyanobacterial scums or high cell density (“green water”), worrying about potential cyanobacterial intoxication, or with symtpoms.

What should I do ?

Diagnose and treat (see reverse page).

Inform the health and environemental department: immediate sampling (within 2-3 days) and determination of the ”algae” to which the patient was exposed, as well as toxin analysis, will be valuable both for your own diagnosis and for hazard identification towards better future risk management.

Chemical behaviour: all currently known cyanotoxins are water soluble and are adsorb to activated charcoal.

Anamnesis: exposure – when, where, for how long, what did the water look like; patient alertness; symptoms before exposure? alcohol, food and drug consumption prior to assumed exposure; other potential reasons for symptoms

Toxicity and symptoms: Noticeable acute inflammatory and irritative effects are likely to be caused by cyanobacterial cell material other than the known toxins. These are usually self-limiting without treatment. Symptoms due to the known toxins as outlined below may trigger medical consultation only if rather severe.

Exposure verification is recommended in all cases through preservation of sample of gastric material from gastic lavage (refrigerate for 1-2 days for (i) verifcation of ingestion of cyanobacterial cells by microscopy and (ii) potentially also by chemical cyanotoxin analyses.

	Peptide hepatotoxins	**Cylindrospermopsin**	**Neurotoxins**
Toxicological information	Acute LD-50 (mouse): intra peritoneal up to 50-60 µg/kg b.w., oral up to 5000 µg/kg b.w.	Acute LD-50 (mouse): intra peritoneal 2100 µg/kg b.w. oral 4400-6900 µg/kg b.w.	Acute LD-50 (mouse): Intra peritoneal 10 - 250 µg/kg b.w. Oral up to 130 - 2400 µg/kg b.w.
Visible Symptoms:	Diarrhoea, abdominal tenderness and palpation, pain	Diarrhoea, abdominal tenderness and palpation, pain	Myosis, shallow and labored breathing; potentially tingling of lips, tongue, extremities; salivation, tachycardia, possible convulsions and seizures
Diagnosis	Serum enzyme samples – GGT, GTP, ALT, AST, LDH (if possible store – deep-freeze for toxin analysis by specialised laboratory); Monitoring of: Blood glucose, fibrinogen, and prothrombin time for hepatocellular damage; Serum electrolytes, urea, creatinine, and glucose for impending renal failure		Cholinesterase activity in whole blood; for saxitoxins serum electrolyts
Confounding etiologies and symptomatologies	Death caps (*Amanita phalloides*, but not A. muscoides) / cylindrospermopsin	Gastrointestinal infection / microcystins/nodularins	*Amanita muscoides*; for Anatoxin-a ingestion of nicotin; exposure to organophosphate and carbamate insecticides (parathion, carbofuran) or chemical warfare agents (e.g. sarin, soman, tabun)
	gastrointestinal infection ; heptatitis		
Immediate treatment	Supportive care (stabilisation); depending on time after exposure forced emesis, oral or gastric application of active charcoal slurry: 240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old. If possible multiple applications		Supportive care (stabilisation); intragastral intubation with active charcoal slurry; support respiration, in case of seizures 5 mg diazepam IV. ***NOTE:*** Phenothiazines, parasympathomimetics and antihistamines are contraindicated since they have anticholinesterase activity and may potentiate toxicity.
	If silibinin is available, consider administration of 20 to 50 mg/kg/day IV		
Progression of symptoms at acute high dose	Increased serum liver enzyme values in conjunction with liver necrosis and development of hepatorenal syndrome, incl. danger of hepatorenal infarction		Recovery within hours is more likely than progression (note: this is on the animal experience only; close observation is necessary !)
Long-term treatment and surveillance	Check liver function and structure (ultrasound) Check renal function		For anatoxin-a as for organophosphates and organocarbamate pesticides or chemical warfare agents (soman, tabun, sarin) For saxitoxins not applicable. See shellfish poisoning

Further reading: Chorus I. & Bartram J. (eds., 1999) Toxic Cyanobacteria in Water – a Guide to their Public Health Consequences, Monitoring and Management. E&F Spon on behalf of WHO, London, 416 pp. Download full text: **http://www.who.int/water_sanitation_health/resourcesquality/toxicyanbact/en/index.html**