

# Climate Change and Bathing Water Quality

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WAGENINGEN UR

*For quality of life*

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## SAMENVATTING

In de nieuwe Europese richtlijn voor zwemwater, 2006/7/EG, worden twee microbiologische indicatoren, te weten intestinale enterococcon en *Escherichia coli*, gebruikt om de toestand van officiële zwembadwateren te bepalen. Ook cyanobacteriën zijn in deze richtlijn opgenomen, aangezien deze organismen een oorzaak kunnen zijn van een onvoldoende zwembadwaterkwaliteit.

Klimaatsverandering kan de zwembadwaterkwaliteit aantasten voor wat betreft zowel het aantal pathogenen en cyanobacteriën, alsook hun weerslag op de gezondheid van de mens. De verwachting is dat, vergeleken met het jaar 1990, in 2050 de winters in Nederland milder en natter en de zomers warmer zullen zijn met minder frequente, maar veel intensere regenbuien, zodat de kans op overstromingen toeneemt, en ook het groeiseizoen langer wordt.

Het is zeer waarschijnlijk dat deze veranderingen in het klimaat effecten zullen hebben op het gezondheidsrisico voor recreanten in en rond oppervlaktewateren. Daarom is een literatuurstudie gedaan naar het effect van klimaatverandering op de zwembadwaterkwaliteit en naar de toepasbaarheid van de nieuwe Europese zwembadwaterrichtlijn in de Nederlandse situatie.

Aanvoer van nieuwe pathogenen of vectoren is waarschijnlijk voor ziekten als malaria, dengue en botulisme. Veel andere ziekten kunnen ook optreden, wanneer de verspreiding van (sub)tropische pathogenen en vectoren niet meer belemmerd wordt door klimaatgrenzen of geografische grenzen, aangezien de internationale mobiliteit van juist Nederlanders tegenwoordig erg groot is. Van deze groep ziekten is het risico voor epidemieën echter niet erg groot, gezien het hoge niveau van de gezondheidszorg in Nederland.

De frequentie en reikwijdte van bloeien van schadelijke algen en cyanobacteriën zal toenemen ten gevolge van klimaatverandering. Oorzaken zijn toenemende eutrofiëring, toenemende groeisnelheden, afnemende menging van oppervlaktewateren, resulterend in anoxische diepere waterlagen, toenemende afbraaksnelheden en een verlengd groeiseizoen. Niet alleen het risico van bloeien van deze organismen zal toenemen, maar ook de hoeveelheid en het type schadelijke toxinen kan beïnvloed worden. En alhoewel de huidige maatregelen ter terugdringing van de eutrofiëring (vigerende mestbeleid, aanpassen RWZI's) uitermate succesvol zijn, kan klimaatverandering dit (deels) teniet doen.

Middels deze studie werd het duidelijk dat het voldoen aan de normen van de nieuwe Europese zwembadwaterrichtlijn niet genoeg zal zijn om een aanvaardbare kwaliteit van de zwembadwateren te garanderen, wanneer eenmaal veranderingen in het milieu als gevolg van klimaatverandering een feit zijn. De twee microbiologische indicatoren omvatten niet de risico's als gevolg van schadelijke algen of cyanobacteriën en vectoren zoals malaria en dengue verspreidende muggen. Gezien het risico voor toenemende eutrofiëring en de dominantie van algen en cyanobacteriën, die tot nu toe nog niet in de Nederlandse wateren zijn gesignaleerd, zijn er gevolgen voor de (haalbaarheid van) de doelstellingen van de Kaderrichtlijn Water en/of de maatregelen die genomen moeten worden.

## SUMMARY

In the new European bathing water directive, 2006/7/EG, two microbiological indicators, i.e. intestinal enterococci and *Escherichia coli*, are used to assess the state of the official outdoor bathing waters. In this directive also cyanobacteria are included, being a cause of insufficient bathing water quality.

Climate change will affect bathing water quality with respect to both the number of pathogens and cyanobacteria, as well as their impact on human health. It is to be expected that by 2050, compared to 1990, the winters in The Netherlands will be milder and wetter, the summers will be warmer, with less frequent, but more intensive showers, thus increasing the chance for flooding and the growing season will be longer.

It is most probable that these changes in climate will have effects on the health risk for bathing in surface waters. Therefore, a literature study was made to investigate the impact of climate change on bathing water quality and the applicability of the new European bathing water directive for The Netherlands.

Import of new pathogens or vectors is very likely for diseases such as malaria, dengue and botulism. Many other diseases are also to be expected as the spread of (sub)tropical pathogens and vectors will not be limited by climate boundaries, nor by geography as international mobility of particularly the Dutch is very high nowadays. Of this group of diseases the risk for epidemics, however, is not very high as health care in The Netherlands is of a high standard.

The frequency and extent of blooms of harmful algae, as well as harmful cyanobacteria, will increase as a result of climate change. Causes are increasing eutrophication, increasing growth rates, decreasing mixing of surface waters, resulting in anoxic deeper water layers, increasing rates of decomposition and a prolonged growing season. Not only the risk of blooms of these organisms will increase, but also the amount and type of harmful toxins can be affected. Although the present measures to reduce eutrophication are extremely successful, climate change could (partly) counteract this.

From this survey it became clear that meeting the standards of the new European bathing water directive will not be sufficient to guarantee bathing waters of sufficient quality once the environmental changes due to climate change are a fact. The two microbiological indicators do not cover the risks caused by harmful algae or cyanobacteria and vectors such as malaria and dengue carrying mosquitos. There are implications for the (feasibility of) objectives of the Water Framework Directive and/or the measures that need to be taken, considering the risk for increased eutrophication and the dominance of algae and cyanobacteria, hitherto unnoticed in the Dutch surface waters, that are likely to colonise these waters.

# CHAPTER 1. AIMS AND DELINEATION OF THIS REPORT

## 1.1 Introduction

On March 24<sup>th</sup>, 2006 the new European bathing water directive 2006/7/EG (Anonymous, 2007) became operational. In this directive it was laid down that by 2015 all bathing water locations have to score at least “sufficient” (art. 5, paragraph 3). This status has been defined using two microbiological indicators: 1) intestinal enterococci and 2) *Escherichia coli* (art. 2, paragraph 5). Furthermore, the directive indicates that when cyanobacterial proliferation occurs appropriate management measures have to be taken (art. 8, paragraph 2).

Climate change as predicted for The Netherlands by the KNMI, based on the 4<sup>th</sup> IPCC report, indicates, amongst others, a rising temperature and increased precipitation. At this moment it is unclear what the impact is of this climate change on organisms that have an adverse impact on bathing water quality and bather’s health.

## 1.2 Aims

In this report the following research questions will be dealt with:

### A. Health risks for bathers by climate related pathogens

With the following subquestions:

- What organisms in bathing water cause health risks and what are these risks?
- How are these pathogens related/correlated to the two microbiological indicators as mentioned in the EU Bathing water directive (intestinal enterococci, *Escherichia coli*)?

### B. The Cyanobacteria issue

With the following subquestions:

- What are the effects of climate change on the occurrence of cyanobacteria nuisance to bathers, what is the relation with climate (temperature and CO<sub>2</sub> – increase in the atmosphere), and which bloom forming cyanobacteria benefit in particular from the predicted changes?
- What is then the relative importance of climate change with respect to nutrients and/or other polluting substances? Does this result in extra efforts in terms of the objectives (e.g. phytoplankton abundance and species composition and supporting physico-chemical elements such as nutrients) of the Water Framework Directive?

## 1.3 Outline

*Chapter 2* summarizes the most important outcomes of the 4<sup>th</sup> assessment report of the Intergovernmental Panel on Climate Change (IPCC) and gives an overview of the most relevant trends in climate change for The Netherlands. In *Chapter 3* the EU directives for bathing water are dealt with. The organisms that contribute to the health risks for bathers are dealt with in *Chapter 4*, where special attention is given to pathogens, particularly their relation to the indicators of the EU bathing water directive. *Chapter 5* deals with the issue of cyanobacteria. *Chapter 6* summarizes the conclusions and recommendations.

## CHAPTER 2. CLIMATE CHANGE

### 2.1 Global change

The IPCC (2007) predicted average global temperatures to increase by 1.1 and 6.4 °C by the end of this century (2100), compared to 1990; the highest probability is in the range 1.8 - 4 °C. By 2050 the global increase will be 1 – 2 °C (highest probability). With respect to a possible change in atmospheric circulation the models used did not come up with a high probability.

### 2.2 The Netherlands

Combining global and regional climate model results (Lenderink *et al.*, 2007) and using the General Circulation Model (GCM) simulations from IPCC (2007), the KNMI came up with predictions of, among others, future temperature and precipitation patterns for The Netherlands (up to 2050) (Hurk *et al.*, 2006). Four scenarios were used (Table 1), i.e. two scenarios using the 1 °C increase (G and G<sup>+</sup>) and two using the 2 °C increase (W and W<sup>+</sup>). Furthermore, for each pair of temperature scenarios a scenario with unchanged atmospheric circulation was used (G and W) and a scenario with changed atmospheric circulation (G<sup>+</sup> and W<sup>+</sup>).

Table 1. The four scenarios as used for The Netherlands (Hurk *et al.*, 2006).

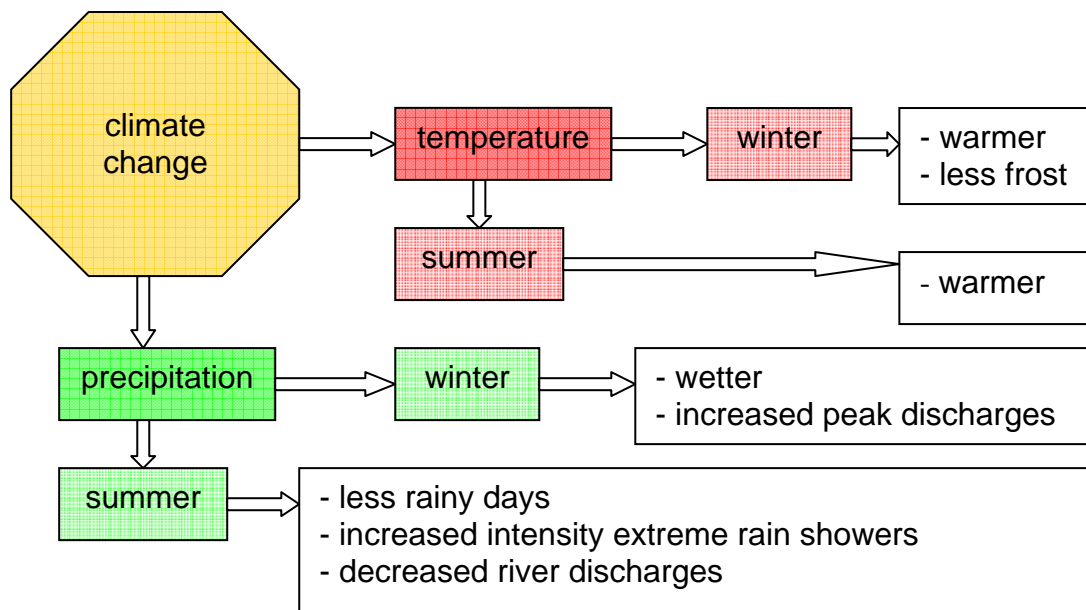
| Scenario       | Global temperature increase in 2050 | Change of atmospheric circulation |
|----------------|-------------------------------------|-----------------------------------|
| G              | + 1 °C                              | weak                              |
| G <sup>+</sup> | + 1 °C                              | strong                            |
| W              | + 2 °C                              | weak                              |
| W <sup>+</sup> | + 2 °C                              | strong                            |

In the two scenarios with a strong change in atmospheric circulation in Western Europe, the winters are mild and wetter due to more western winds and the summers are warmer and dryer resulting from more eastern winds.

All four scenarios indicated the following trends (Bresser *et al.*, 2005, Dorland en Jansen, 2007):

- Temperature in The Netherlands will continue to rise. Mild winters and hot summers will become more common.
- On average winters will become wetter (4 - 14 % in 2050) and extreme precipitation amounts will increase (4 - 14 % in 2050).
- The intensity of extreme rain showers in summer will increase (5 - 27 % in 2050). However, the number of rainy days in summer will decrease.
- The calculated change in wind is small compared to the natural fluctuations.
- The sea level will continue to rise, however at a lower rate than predicted earlier.
- The expected increase in winter precipitation will be accompanied by increased peak discharges.
- More precipitation in the Alps will occur in the form of rain rather than snow. This increases the Rhine discharge in winter.
- Summertime river discharges will decrease.

In *Figure 1* the most relevant outcomes in the light of this study are summarized.



*Figure 1. Predicted temperature and precipitation patterns for The Netherlands by the year 2050.*

As both summer and winter will be warmer, the growing season will be prolonged. As the winters will be wetter and the summer rains, although less frequent, will be more intensive, an increased run-off is to be anticipated.

## CHAPTER 3. BATHING WATER DIRECTIVES

Recreation on and in surface waters can be a serious source of health complaints. Apart from injuries, contact with water or with pathogens or their vectors could imply all forms of health problems (Schets en De Roda Husman, 2004, 2005a, Craun *et al.*, 2005). Therefore, in 1976, the European Bathing Water Directive 76/160/EEG (Anonymous, 1976) became operational. The idea behind this directive was to improve the water quality as well as to protect public health against possible risks of bathing in surface waters. All EU member states had to monitor the official bathing localities and to comply with the quality standards as laid down in the directive. The microbiological parameters total coliforms and faecal coliforms had to be monitored biweekly, whereas faecal streptococci, *Salmonella* and enteroviruses had to be measured in case the locality was suspected.

Since 1976 water recreation expanded and not only bathers are using surface waters, but also windsurfing, canoeing, waterskiing and other water related sports became very popular. This led to the release of an improved directive for bathing waters, 2006/7/EG (Anonymous, 2006). This directive also had to comply with the Water Framework Directive, 2000/60/EG (Anonymous, 2000a), which became operational in 2000.

Monitoring indicated that microbiological pollution is the most important limitation for reaching a good bathing water quality. The microbiological parameters in the Bathing Water Directive of 1976, i.e. total coliforms, faecal coliforms and faecal streptococci were abolished and intestinal enterococci as well as *Escherichia coli* have been selected as microbiological parameters in the new directive, as many comprehensive epidemiological studies showed that these two parameters indicated a far better correlation between faecal pollution of bathing waters and health risks (Kay *et al.*, 1994, Fleisher *et al.*, 1996, Asperen *et al.*, 1998). In the new directive it is laid down that by 2015 all bathing water locations have to score at least “sufficient” (art. 5, paragraph 3). This status has been defined using the two forementioned microbiological indicators.

Despite the improvement in the control of bathing waters as laid down in the Bathing Water Directive 2006/7/EG, the therein mentioned two microbiological indicators are not sufficient to protect the bather’s health as there is no unambiguous relation between the presence of these indicators and the presence of pathogen microorganisms in the water (Wade *et al.*, 2003). For instance, Ruiters *et al.* (2004) found no relation between *E. coli* and *Campylobacter* in six bathing locations. So, if bathing water meets the microbiological standards of the new bathing water directive, still pathogenic bacteria, protozoa or viruses could be present. Furthermore, directive 2006/7/EG does not include guidelines to reduce the incidents associated with skin complaints, apart from the inclusion of cyanobacteria in the directive. To reduce the number of skin complaints additional standards are needed with respect to other organisms that cause these complaints (Leenen en Roda Husman, 2004).



# CHAPTER 4. ORGANISMS THAT CAUSE HEALTH RISKS FOR BATHERS

## 4.1 Taxa involved and their characteristics

There are many potential hazards that are encountered by bathers and other recreational users of coastal and inland fresh waters. The World Health Organisation distinguished: physical hazards, cold, heat and sunlight, water quality, contamination of beach sand, algae and their toxic products, chemical and physical agents and dangerous aquatic organisms (WHO, 2003). Although the Bathing Water Directive is restricted to the quality of the water itself, the surroundings, such as beach, meadow and forest, are also important potential sources of disease bringing organisms to the bathers. In other words, the water is indissolubly connected to its surroundings.

Contamination with pathogens takes place through vectors and hosts such as the bathers themselves, or animals that visit the bathing place, but also through faecal pollution by birds, not only of the water itself, but also of the surrounding areas that give access to the water body. Therefore, here an overview is given of both vectors and pathogen organisms, as encountered in the surface waters as well as in their direct surroundings (Table 1). The distribution of these organisms is predominantly worldwide, with a few typically occurring in the tropics (hepatitis viruses and *Schistosoma*). However, due to the enormously increased mobility of people all these organisms can be transported to The Netherlands, either by their vectors or by humans themselves, or, as in the case of the toxic dinoflagellate *Pfiesteria piscida* by ballast water. Table 1 summarizes most important characteristics of the pathogens; the last column indicates whether the risk of infection with this disease is likely to increase in The Netherlands, due to climate change

Many pathogens are not waterborne themselves, but develop in hosts that are, during part of their life, living in water. A well-known example of these vector-borne pathogens is malaria, where the adult mosquito *Anopheles* is the carrier of the pathogen *Plasmodium*. *Anopheles* lives in freshwaters throughout its larval and pupal life stage. In Table 2 the most important pathogens belonging to this group are summarized. The last column of this table indicates whether the risk of infection with this disease is likely to increase in The Netherlands, due to climate change.

Diseases that are normally mild and self-limiting in the general population can have severe manifestations in susceptible individuals. The immune system of these victims can be affected by other diseases, age, medications taken, pregnancy, nutritional status, genetics and other factors (Carr and Bartram, 2004). Furthermore, sequelae symptoms may be completely different from the symptoms of the acute illness and may occur even if the immune system successfully manages to eliminate the primary infection (Pond, 2005).

Table 1. Overview of most important organisms that are a serious health risk for bathers in The Netherlands (Pond, 2005, Giessen et al., 2004). The last column indicates the risk of infection in The Netherlands as a result of the anticipated climate change (G+ and W+ scenarios). 0 = no increased risk, ++ = slightly increased risk, +++ = increased risk.

| organism                              | disease*                                       | oxygen        | temp. (max.)      | fresh/marine   | dry/wet      | vector          | increased risk |
|---------------------------------------|--|---------------|-------------------|----------------|--------------|-----------------|----------------|
| <b>Bacteria</b>                       |  |               |                   |                |              |                 |                |
| <i>Escherichia coli</i> O157          | abdominal cramping, bloody diarrhoea           |               | < 5 19.3-41.0 (b) | fresh          | wet          | cattle          | 0              |
| <i>Legionella pneumophila</i>         | Legionnaires' disease                          | aerobic       | 25- 35 (60)       | fresh/brackish | wet          | free-living     | ++             |
| <i>Leptospira icterohaemorrhagiae</i> | leptospirosis (Weil's disease)                 | aerobic       | 28-30; < 42       |                | wet          | rats            | ++             |
| <i>Listeria monocytogenes</i>         | listeriose (meningoencephalitis)               | anaer. + aer. | 3-42              |                | wet/dry      |                 | 0              |
| <i>Mycobacterium avium</i>            | lung damage                                    | low           | up to 45          |                | wet/dry      | animals, humans | ++             |
| <i>Salmonella</i> spp.                | (para)typhus                                   | fac. anaer.   |                   | fresh/marine   | wet          | animals, humans | 0              |
| <i>Shigella</i> spp.                  | dysentery                                      | fac. anaer.   | 12-37 (20)        |                | wet          | man, gorilla    | 0              |
| <i>Vibrio cholerae</i>                | diarrhoea                                      | fac. anaer.   |                   | fresh          | wet/dry      | free-living     | ++             |
| <i>Vibrio vulnificus</i>              | necrotising wound, infections, gastroenteritis | fac. anaer.   |                   | marine         | wet          | free-living     | ++             |
| <b>Algae</b>                          |  |               |                   |                |              |                 |                |
| <i>Pfiesteria piscicida</i>           | skin irritation, nervous system problems       | aerobic       |                   | marine         | wet          | free-living     | ++             |
| <b>Protozoa</b>                       |  |               |                   |                |              |                 |                |
| <i>Brucella</i> spp.                  | brucellosis                                    |               |                   |                |              | cattle          | 0              |
| <i>Clostridium botulinum</i>          | paralysis                                      | low           | > 20              | fresh/marine   | wet          | birds           | +++            |
| <i>Cryptosporidium parvum</i>         | diarrhoea                                      |               |                   | fresh/marine   | wet-oocysts  | mammals         | ++             |
| <i>Giardia duodenalis</i>             | diarrhoea                                      |               |                   |                | wet-oocysts  | animals, humans | ++             |
| Microsporidia                         | infection digestive tract                      |               |                   |                | wet-spores   | animals, humans | 0              |
| <i>Naegleria fowleri</i>              | meningoencephalitis                            |               | 25-35             | fresh warm     | wet-sediment | free-living     | ++             |
| <b>Viruses</b>                        |  |               |                   |                |              |                 |                |
| Human adenovirus                      | upper respiratory tract                        |               |                   | fresh/marine   |              | humans          | ++             |
| coxsackievirus                        | gastro-enteritis                               |               | -20-70            | fresh/marine   |              | humans          | ++             |
| echovirus                             | gastro-enteritis                               |               |                   | fresh          |              | humans          | ++             |
| hepatitis A                           | jaundice                                       |               |                   |                |              | humans          | ++             |
| hepatitis E                           | jaundice                                       |               |                   |                |              | humans          | ++             |

\* only the most important diseases

(a) Raghubeer, E.V. and J.R. Matches, 1990.

(b) Ruiter, H. et al., 2004.

Table 2. Vector-borne pathogens transmitted by waterborne organisms (compiled from WHO, 1990; Chan *et al.*, 1999)  
 0 = no increased risk, ++ = slightly increased risk, +++ = increased risk in The Netherlands.

| pathogen               | disease                        | vector                | presence     | increased risk |
|------------------------|--------------------------------|-----------------------|--------------|----------------|
| <i>Plasmodium</i> spp. | malaria                        | <i>Anopheles</i> spp. | (sub)tropics | +++            |
| dengue virus           | dengue                         | <i>Aedes aegypti</i>  | (sub)tropics | +++            |
| Trematodes             | schistosomiasis<br>(bilharzia) | snails                | (sub)tropics | ++             |
| West-Nile virus        | West-Nile virus                | mosquitos             | tropics      | ++             |
| Nematodes              | lymphatic filariasis           | mosquitos             | (sub)tropics | 0              |

## 4.2 Future risks related to climate change

Exposure to pathogens occurs either via drinking water (associated with faecal contamination), seafood and fresh produce or via direct contact with the pathogens and vectors themselves. The impacts of climate change on the epidemiology of waterborne diseases are through increased flooding, heavy rainfall events and increased temperature. In developing countries increased risk of infection is associated with flooding, but not in the West, unless water sources are compromised (Hunter, 2003). Guber *et al.* (2001) listed a range of possible mechanisms whereby rainfall can impact on the risk of transmission of vector-borne diseases. Increased surface water areas can provide breeding sites for vectors and can increase vegetation, thus allowing expansion in population of invertebrate host, while flooding may eliminate habitats for both vectors and hosts and it may force vertebrate hosts into closer contact with humans. Temperature can effect both the distribution of the vector and the effectiveness of pathogen transmission through the vector. Temperature impacts on the risk of transmission of vector-borne diseases are (Gubler *et al.*, 2001): changes in a) survival of the vector, b) rate of vector population growth, c) feeding behaviour, d) susceptibility of vector to pathogens, e) incubation period of the pathogen, f) seasonality of vector activity and g) seasonality of pathogen transmission.

Distribution patterns of pathogens and vectors are largely influenced by humans. Apart from climate change, important factors are trade, tourism, migration and the changing of the environment. Ecological changes, such as encroachment of forests and destruction of natural areas may increase human contacts with animals, carrying microbes that may adapt to humans, leading to new diseases. In contrast, in several European countries with nature restoration programmes, opportunities for sylvatic animals (vertebrates and arthropods) increase and so do opportunities for the spreading of zoonotic pathogens (Giessen *et al.*, 2004). Through migration and also tourism pathogens as well as vectors are easily brought from one country to the other; this became particularly a problem for The Netherlands during the last decade in which not only

immigration increased, but travelling to the most exotic places in the tropics gave an increased probability of infection with harmful pathogens, unknown to The Netherlands or eradicated there for many years. That no serious outbreaks occurred is the result of the high level of health care. Trade as a form of spreading pathogens is an ever increasing risk. Ballast water, trade with plants and animals, but also accidentally transport of pathogens via contaminated cargo ensure an international exchange of pathogens. All these means of distribution facilitated by humans are ongoing for decades although most of the imported exotic pathogens did not lead to serious epidemics as the environmental situation in The Netherlands was not optimal or even hostile.

However, climate change will lead in The Netherlands to a more favorable environment for many (sub)tropical pathogens or their vectors. It is to be expected that tourism will increase as there will be both a shift from the Mediterranean tourist centre (too hot in summer) to the more northern countries as well as an increase in the number of Dutch recreants that will stay in The Netherlands; this increase most probably will outweigh the expected deterioration of the water quality (Bresser *et al.*, 2005). Heavy rainfall events are likely to lead to a marked decline in the microbiological quality of inland and marine recreational waters as a result of increased runoff. Higher temperatures may add to an increased risk of eutrophication of surface waters. Also the onset of eutrophication, as visualised through algal and cyanobacterial blooms or cover with duckweeds, will be earlier in spring and these blooms will last longer. There will be a cascade of detrimental effects for the ecosystems in question. For instance, anoxic situations will prevail and start earlier in the year, thus providing ideal circumstances for many groups of pathogens. Eutrophication in combination with higher temperatures will lead to more vegetation in the water. This will increase the accumulation rate of sediment. If the frequency of dredging will not be increased, the average ratio water/sediment will decrease, thereby decreasing the water quality even further and the possibility of survival of coliforms. It is assumed in general that dredging has a positive effect on the water quality.

The change in climate will also create ideal situations for pathogens originating from (sub)tropical areas. As dispersal is nowadays not limited and even is facilitated by the increasing numbers of Dutch going to (sub)tropical regions for their holidays, inoculation with non-endemic pathogens will be a constant factor. Serious diseases as malaria and dengue will become a possible threat for The Netherlands. The same is true for diseases caused by fungi and viruses.

We will envisage changes in migration patterns of birds. Already now we observe an increase in the populations of geese (f.i. *Branta bernicla*) that do not leave The Netherlands anymore. The nutrient enrichment of our surface waters nearby the resting places of these birds is a growing concern. Passing birds like geese are also a source of coliforms to be found in surface water during winter (Harmsen *et al.*, 2002).

Below the health risks of pathogens, mentioned in Table 1 and 2, are explained further.

*a) Pathogens that are likely to have an increased health risk due to climate change:*

Botulism already exists in The Netherlands for decades. Of the six types only a few types are human related. *Clostridium botulinum* has a high growth rate at temperatures above 20 °C and is then fatal to aquatic animals (mostly water fowl). If the dead birds are not removed immediately, they form an infection burden to bathers, divers and other water recreants. It is to be expected that botulism will increase due to climate change.

Malaria was endemic in The Netherlands till after World War II; since 1970 The Netherlands are malaria free (Kaaden, 2003). Nowadays, malaria in The Netherlands is an import disease, with slightly increasing numbers of infected Dutch (Eerden *et al.*, 2002, 2003). It is to be expected that malaria infections in The Netherlands will increase, due to both increased temperature and rainfall. Bouma and Kaay (1996) and Bouma *et al.* (1997) indicated a link between increased malaria risk and El-Niño; this relationship was partly due to increased temperature and increased rainfall, leading to increased mosquito breeding sites. For the UK it is expected that climate change will not result in an increased risk of malaria outbreaks (Department of Health, 2001/2002), unless new vector species become established in Europe (Department of Health and Health Protection Agency, 2007).

For dengue the situation is the same. The vector, *Aedes aegypti*, has reached Europe (Italy, Brussels) and has even been identified in pools in Schiermonnikoog. The mosquito is imported with bamboo shoots from China (Reinhold, 2007).

*b) Pathogens that are likely to have a slightly increased health risk due to climate change:*

Legionellosis caused by the bacterium *Legionella pneumophila* was the cause of over 200 infected visitors of a flower exhibition in 1999 in Bovenkarspel, The Netherlands. This was the first serious case of this disease that led to the death of 32 visitors. The bacteria are always present but thrive at temperatures above 25 °C (up to 60 °C). However, infection is only through water vapour.

Leptospirosis is a disease caused by bacteria. *Leptospira icterohaemorrhagiae* is the cause of Weil's disease, and although the bacterium is widespread in The Netherlands during late summer, there are not that many cases reported (30 serious cases per year). However, a lengthening of the growing season and the increase in temperature could drastically increase the number of infections.

Cholera in The Netherlands is an import disease, at least since the last pandemic of 1958. However, a recent case of infection with *Vibrio cholerae* indicated that the risk of infection with these pathogens is still present. *Vibrio spp.* thrive at higher temperatures in surface waters and have been found in the Baltic Sea and even Lake IJsselmeer is now suspected. Thus, climate change could trigger the return of *Vibrio*-related diseases (Muijsken en Menger, 2007).

Although *Mycobacterium spp.* are widespread in surface waters, human mycobacterial diseases associated with their use are rare. Apart from superficial infections, the risk of more serious diseases in people with a weakened immune system should not be underestimated (Leoni *et al.*, 1999). Temperature increase and lengthening of the growing season will increase the number of infections.

*Pfiesteria piscicida* is a marine dinoflagellate producing toxins that seriously affect human health (Glasgow *et al.*, 1995). It is not clear whether this organism should be regarded as a true pathogen (it has an amoeboid phase in its life cycle). This organism was first detected in estuarine areas in North Carolina in the 1980s and 1990s and later at Chesapeake Bay and off Mexico. The effects on human are diverse and vary from skin irritation to defects in the neural system (Leenen, 1998). The species thrives in areas where the salinity is low (estuaries) and pollution high. Jacobsen *et al.* (2002) reported the species from Norway, indicating that *Pfiesteria* crossed the Atlantic and could become a serious threat to the Dutch coast. It is therefore that climate change, i.e. increased runoff of nutrients, could contribute to the occurrence and abundance of this species.

*Cryptosporidium parvum* as well as *Giardia duodenalis* are found in surface waters, where they arrive through pollution with sewage water, via faeces from infected animals or via runoff from manure-enriched agricultural areas. The oocysts are widespread. Both pathogens are strongly associated with each other.

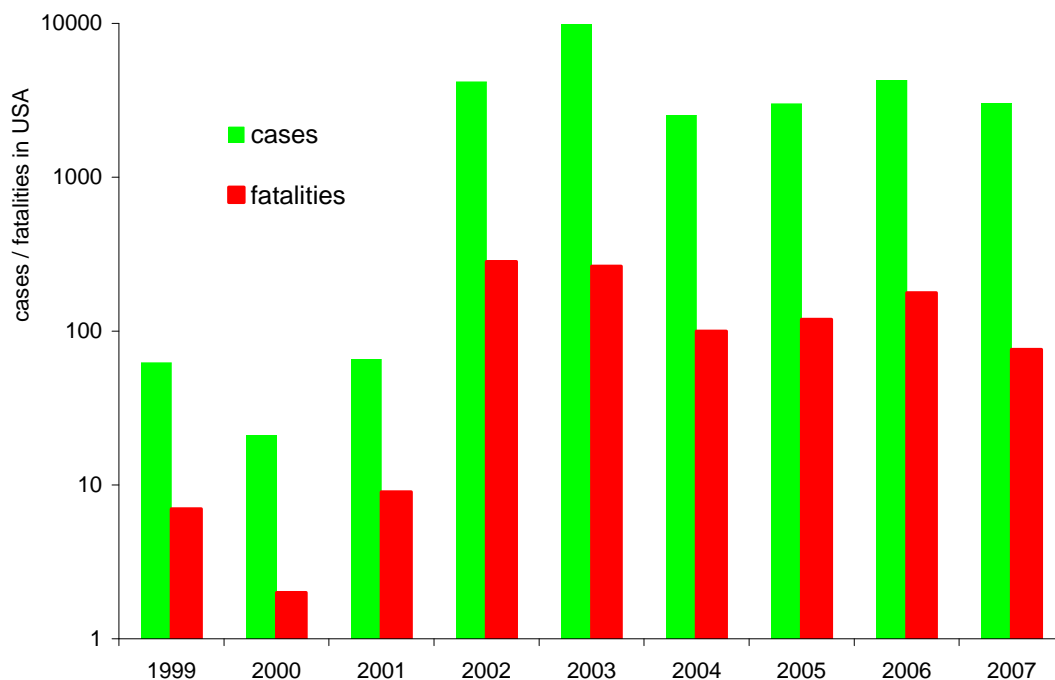
The protozoan *Naegleria fowleri*, is an amoeba that thrives in warm, shallow freshwaters. The organism enters the body via the nose and thus is a risk for bathers. The number of infections, however, is very low and no cases from The Netherlands are known. As the protozoan growth rate increases with increasing temperatures (> 25 °C), there will be a risk in future.

Viruses (human adenovirus, coxsackievirus, echovirus, hepatitis A and hepatitis E virus) are most likely associated with bathing waters, contaminated with faeces or nasal excretates (coxsackievirus) from infected individuals (Pond, 2005). Increase of infections is likely at higher temperatures.

In France swimmers itch (cercarial dermatitis; *Trichobilharzia*) increased 10-fold in 10 years time (Sluifers, 2004). Also in The Netherlands swimmers itch is the most occurring health problem associated with recreational bathing (Leenen en Roda Husman, 2004). The most probable cause of this increase is the early start of the growing season (3-4 weeks earlier). However, also the improved water

quality in surface waters is an important driver as the increased transparency enabled the growth of submerged waterplants that are perfect habitats for snails, the hosts of the cercaria. The increased occurrence of cercarial dermatitis could also be linked to the prolonged stay of migratory birds, especially ducks (Anatidae), carrying the larvae as intermediate hosts (Bayssade-Dufour *et al.*, 2002).

The West Nile virus was first detected in Uganda in 1937. Nowadays it is most commonly found in Africa, West Asia, Europe and the Middle East. It caused an epidemic in New York during the summer of 1999. Since then it regularly reappears with increasing numbers of infected people and fatalities (*Figure 2*). The vectors of this virus are several species of mosquitos, predominantly *Aedes aegypti*. Both increased temperature and rainfall have a positive effect on its distribution.



*Figure 2. The development of the West Nile virus in the USA (data from CDC, 2007).*

### 4.3 Relation with indicators from the EU directive

In the new EU directive for bathing water quality (2006/7/EG, Anonymous, 2006) intestinal enterococci and *Escherichia coli* are the two microbiological indicators for assessment of the quality of bathing waters. Most authors agree that the replacement of the three indicators in the old directive (76/160/EEG, Anonymous, 1976), i.e. total coliforms, faecal coliforms and faecal streptococci, was a good move (Kay *et al.*, 1994, Fleisher *et al.*, 1996, Asperen *et al.*, 1998). However, apart from questions raised with respect to the methodology (Chawla and Hunter, 2005; Kinzelman *et al.*, 2003; Schets *et al.*, 2006), also the use of only these two indicators is questioned (Wiedenmann *et al.*, 2006; Leenen en Roda Husman, 2004; Schets *et al.*, 2006). This is mainly due to the fact that the two microbiological indicators indicate potential infections by waterborne pathogens that are associated with the pollution of natural recreational waters by human and animal faeces. In this the use of both indicators appeared to be essential as Schets *et al.* (2006) showed that *E. coli* failed to indicate faecal pollution in marine waters and intestinal enterococci failed to do so in freshwaters. Still, as health risks associated with using outdoor recreational waters are not only determined by faecal pollution, additional indicators are needed to non-faecal related health risk. Skin irritations, caused by other organisms than cyanobacteria, are not monitored. Also infections by pathogens carried by waterborne invertebrate and land-dwelling vertebrate hosts are excluded from the present directive.



## CHAPTER 5. HARMFUL PHYTOPLANKTON BLOOMS

### 5.1 Harmful algal blooms

Phytoplankton organisms are natural components of aquatic systems and being the major primary producers they are essential constituents at the base of aquatic food chains. Given favourable environmental conditions phytoplankton may proliferate at a high rate (Hallegraeff, 1993). The resulting high biomass may form a visible and dense accumulation near or at the water surface and a bloom is manifest. When the proliferating organisms pose a threat to human and environmental health the blooms are referred to as harmful algal blooms.

Harmful algal blooms have been reported from aquatic systems worldwide. Reports of harmful algal blooms have become more frequent and trends over the last decades reveal that the frequency, magnitude and geographical distribution of harmful algal blooms is increasing (Van Dolah, 2000; Fogg, 2002; WHO, 2003). In aquatic environments, harmful algal blooms have been implicated in a series of human and animal poisonings (Dybas, 2003). Moreover, they pose a risk for the sustainability of ecosystems through severe overgrowth, oxygen depletion and/or release of potent toxins that may inhibit/eliminate other species. Although massive bloom formations, i.e. rapid expansion in the population size of harmful algal microorganisms, occur in marine, brackish and freshwater, they significantly differ with respect to the blooming organisms. Blooms in marine environments are often caused by eukaryotic microorganisms, such as dinoflagellates, whereas prokaryotes (cyanobacteria) often dominate the blooms in brackish and freshwater systems.

Inasmuch as they appear to be unable to colonise, invade, and grow in human or animal hosts to cause disease, these toxigenic micro-organisms are not listed among waterborne pathogens (f.e. *Aeromonas*, *Enterobacter*, *Salmonella* and *Shigella*; vide Chapter 4) in the water industry. Nonetheless, their potent toxins present waterborne hazards to health (Codd *et al.*, 2005).

### 5.2 Harmful blooms in marine environments

The global increase of harmful algal blooms in coastal waters is attributed to various factors:

- 1) rapid increase of human population and related eutrophication and pollution,
- 2) increased utilization of coastal waters for aquaculture,
- 3) transport of organisms in ships' ballast water, and
- 4) unusual climatologic events and global climate change.

The apparent increase in coastal marine harmful algal blooms globally can endanger human health through shellfish poisoning and respiratory illness. Harmful algal bloom organisms can produce a variety of toxins that may be

classified based on chemical characters as follows (Van Dolah, 2000; Hernández-Becerril *et al.*, 2007):

• **Lipophilic toxins**

- *Azspiracids*: new group of toxins produced by *Protoperdinium crassipes*.
- *Brevetoxins*: neurotoxins found in “red tide” organisms (e.g. *Karenia brevis*)
- *Okadaic acid*: diarrhetic shellfish toxin produced by *Dinophysis* and *Prorocentrum*.
- *Pectenotoxins*: scallop toxins produced by *Protoceratium* and *Lingulodinium*.

• **Hydrophilic toxins**

- *Domoic acid*: amnesic shellfish toxins produced by *Pseudo-nitzschia*.
- *Saxitoxins*: paralytic shellfish toxins produced by *Gymnodinium catenatum*, *Pyrodinium* and *Alexandrium sp.*

• **Other toxins**

- *Ciguatoxins, Gambiertoxins and Maitotoxins*: Ciguatera fish poisoning syndrome.
- *Unknown toxin(s) from Pfiesteria piscicida*, a fish-killing dinoflagellate, causing the Estuary-Associated Syndrome.

Marine algal toxins have been estimated being responsible for more than 60,000 intoxication incidents per year (worldwide), with an overall mortality rate of 1.5%; impacts are generally observed as acute intoxications (Van Dolah, 2000). The primary vector of most harmful algal bloom-related illnesses are reef fish and shellfish that ingest these algae and act as vectors to humans directly (shellfish) or indirectly through toxin bioaccumulation along the food chain in piscivorous fish (Van Dolah, 2000). Consumption of fish and shellfish, which is contaminated with harmful algal-toxins, results in five poisoning syndromes:

- 1) amnesic shellfish poisoning,
- 2) ciguatera fish poisoning,
- 3) diarrhetic shellfish poisoning,
- 4) neurotoxic shellfish poisoning, and
- 5) paralytic shellfish poisoning.

The primary target of many harmful algal toxins is the neurologic system with gastrointestinal symptoms emerging minutes to hours after exposure. In the case of amnesic-, paralytic shellfish poisoning, and ciguatera fish poisoning, acute respiratory failure may be fatal within minutes to hours, while chronic neurologic symptoms from ciguatera and amnesic shellfish poisoning may last for months to years (Van Dolah, 2000; Fleming *et al.*, 2006). Inasmuch as these neurotoxins are heat stable, cooking has no ameliorating effect on toxicity.

Primary poisonings result from eating toxin-contaminated seafood, however exposure to marine aerosols containing brevetoxins that were driven onshore by the wind, has also been linked to health complaints (Kirkpatrick *et al.*,

2004; Cheng *et al.*, 2005). It has been found that people can experience upper and lower respiratory irritation and some inflammatory response after inhaling aerosolized brevetoxins during red tide events (Backer *et al.*, 2003). In addition, adverse changes in lung function from exposure to aerosolized red-tide toxins in asthmatic persons have been found (Fleming *et al.*, 2007).

A volatile toxin associated with the dinoflagellate *Pfiesteria piscicida* is suspected to impact human health through the respiratory route (Hudnell, 2005). A human intoxication syndrome, with symptoms such as headaches, respiratory irritations, skin lesions, skin “burning” upon contact with water, forgetfulness, disorientation, and acute confusion, was associated with direct exposure to estuaries and exposures to estuarine aerosols around the time of *Pfiesteria*-related fish kills (Glasgow *et al.*, 1995; Grattan *et al.*, 1998; Silbergeld *et al.*, 2000). The syndrome is nowadays referred to as Possible Estuary Associated Syndrome (Hudnell, 2005).

### 5.3 Effects of climate change on marine harmful algal blooms

The direct components of predicted climate change affecting marine organisms over the next century are 1) temperature increase, 2) sea level increase and subsequent changes in ocean circulation, and 3) decrease in salinity (Harvell *et al.*, 2002).

Temperature can influence the amount and type of harmful algal toxins produced (Siu *et al.*, 1997; Hwang & Lu, 2000). Temperature also directly affects the growth of algae. Future changes in summer surface temperatures may result in an 11% areal increase in the potential geographic distribution of the marine bloom-forming cyanobacterium *Trichodesmium* due to the poleward shift of the 20 °C isotherm (Breitbarth *et al.*, 2007). Although blooms are not expected along the Dutch coast, cells can survive at 17 °C for several weeks and *Trichodesmium* can be transported to higher latitudes by oceanic currents and ballast water.

Warmer sea temperatures can encourage a shift in species composition of algae toward the more toxic dinoflagellates. Experiments revealed that due to climate change the risk of harmful dinoflagellate and raphidophyte blooms in the Dutch coastal zone will increase rather than decrease (Peperzak, 2003). In a follow-up, Peperzak (2005) showed that expected temperature increase, in combination with water column stratification, led to a doubling of growth rates of potentially harmful dinoflagellates and raphidophytes, meaning that the frequency and intensity of harmful algal blooms may increase in the future. Analyzing long-term datasets, Edwards *et al.* (2006) showed that since the late 1980s an environment has established that favours the growth and earlier succession of flagellates and dinoflagellates in the North Sea. Furthermore, they conclude that the recent large harmful algal blooms that have manifested most conspicuously off the coast of Norway appear to be associated with warm temperatures coupled with the general decrease in salinity in the Norwegian Coastal Current, but that also the German and Dutch Bight are among the vulnerable areas (Edwards *et al.*, 2006). Looking in the past at pre-industrial age cyst records of ‘red tide’

histories, Mudie *et al.* (2002) found that cyst abundances were an order of magnitude larger when summer surface temperatures were up to 5 ° warmer. Hence, they found another line of evidence indicating that climate change (including surface temperature and storminess) is the main driving force stimulating blooms (Mudie *et al.*, 2002).

Upsurges of toxic phytoplankton blooms in Asia are strongly correlated with the El Niño Southern Oscillation cycle. The concomitant changes in sea surface temperature correlate with changes in the frequency of ciguatera disease in the Asian Pacific (Hales *et al.*, 1999). A well-documented example of how human health threats from the ocean are affected by climate is provided by cholera. The causative agent of cholera (*Vibrio cholerae*) is a facultative human pathogen found in coastal waters that causes the acute gastrointestinal disease cholera, a major health threat in poor nations that frequently results in mortality. Sea surface temperature in the Bay of Bengal is correlated with algal blooms and outbreaks of cholera in Bangladesh (Harvell *et al.*, 1999). It has been revealed that the dissolved organic matter during intense phytoplankton blooms has the potential to support explosive growth of *V. cholerae* in seawater, which might be an important factor in disease propagation (Mouriño-Pérez *et al.*, 2003). Thus, an increase in sea surface temperature, along with eutrophication, might stimulate both algal blooms and vibrios (Hayes *et al.*, 2001).

Despite several uncertainties, such as correctness of the projected temperature and precipitation scenarios, expected future Rhine discharge and North Sea stratification, wax or wane of nutrient loadings, and the overall response of the key players, climate change will increase rather than decrease the risk of harmful dinoflagellate and raphidophyte blooms in the Dutch coastal zone (Peperzak, 2005). The recurrently blooming raphidophytes *Fibrocapsa japonica*, *Chattonella marina*, *C. antiqua* in Dutch coastal waters are toxic to fish. However, the dinoflagellates *Dinophysis acuminata* and *Prorocentrum minimum* are well-known potent causative agents of diarrhetic shellfish poisoning. Symptoms are expressed rapidly (30 min to a few hours) and include diarrhoea, nausea, vomiting, and abdominal pain. Chronic exposure may promote tumour formation in the digestive system. The diatom *Pseudo-nitzschia multiseries* causes amnesic shellfish poisoning, of which symptoms are nausea, vomiting, abdominal cramps, decreased reaction to deep pain, dizziness, hallucinations, confusion, short-term memory loss and seizures (Masó and Garcés, 2006). Overall, the effects of the predicted increase in blooms of these phytoplankters will range from impacting the natural food chain, cultured marine food products, human health, to touristic and recreational activities.

## 5.4 Harmful algal blooms in brackish and freshwater environments

In estuaries and inland freshwater systems, eutrophication has stimulated the appearance of cyanobacterial proliferations (blooms). Such blooms can hamper recreation as especially mass developments of surface-dwelling and scum-forming cyanobacteria are an aesthetical nuisance, both visually (unaesthetic look, low water transparency) and odiferous (bad smell). Moreover, blooms indicate water quality deterioration and may pose a serious threat to animal and human health as several cyanobacteria can produce a variety of very potent toxins (e.g. Codd *et al.*, 2005; Dittmann & Wiegand, 2006). The potential health concern of poisonings by cyanobacterial waterblooms, in combination with the strong indications that the frequency of blooms and their geographic spread is likely to grow with increasing eutrophication and global climate change, has led to an increased attention worldwide. In Europe, hazardous cyanobacterial blooms, scums, or mats have been reported to occur in at least 24 countries: Belgium, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, The Netherlands, Norway, Poland, Portugal, Russia, Slovakia, Slovenia, Spain, Sweden, Switzerland, Ukraine, United Kingdom (Codd *et al.*, 2005).

The toxins that are being produced by cyanobacteria (so-called cyanotoxins) can be characterized by their chemical or toxicological properties (Haider *et al.*, 2003; Codd *et al.*, 2005; Wiegand & Pflugmacher, 2005; Dittmann & Wiegand, 2006). Based on their chemical structures, cyanotoxins are divided into three classes:

- 1) oligopeptides,
- 2) alkaloids, and
- 3) lipopolysaccharides.

Based on their toxic mechanisms to vertebrates cyanobacterial toxins can be classified in five functional groups:

- 1) hepatotoxins (microcystins and nodularins),
- 2) neurotoxins (anatoxins, saxitoxins, neosaxitoxins, and BMAA),
- 3) cytotoxins (cylindrospermopsin),
- 4) dermatotoxins (aplysiatoxin, debromoaplysiatoxin and lyngbyatoxin), and
- 5) irritant toxins (lipopolysaccharides).

### • **Hepatotoxins**

*Microcystins* and *nodularins* both are cyclic peptides consisting of seven and five amino acids, respectively. Over 70 structural variants of microcystins and six variants of nodularins are known. All structural congeners accumulate in the liver, where they inhibit protein phosphatases and distort the cytoskeleton causing major liver damage. They are tumour promoters and nodularin is also a carcinogen.

### • **Cytotoxins**

*Cylindrospermopsin* is an irreversible inhibitor of protein biosynthesis. This alkaloid causes necrosis of liver, kidney, thymus, spleen, intestine and lungs, and displays mutagenic and possibly even carcinogenic activity.

### • **Neurotoxins**

*Saxitoxins*, alkaloids of which more than 10 structural variants are known, block neuronal transmission by binding to the voltage-gated Na<sup>+</sup> channels in nerve cells. The entering sodium flow is stopped by saxitoxins, which leads to muscle paralysis and death by respiratory arrest in mammals.

*Anatoxins* are common alkaloid neurotoxins that may be lethal by causing respiratory failure. The variants *anatoxine-a* and *homoanatoxine* mimic the neurotransmitter acetylcholine. They bind irreversibly to the nicotinic acetylcholine receptor causing continuous production of action potentials leading to overstimulation of the muscle cells. The variant *anatoxin-a(s)* is a potent acetylcholinesterase inhibitor.

*β-N-methylamino-L-alanine* is a nonprotein amino acid that has been linked recently to a neurodegenerative disease and Alzheimer's disease in humans (Cox *et al.*, 2005).

### • **Dermatotoxins**

*Lyngbyatoxin A*, *aplysiatoxin*, and *debromoaplysiatoxin* are all three being produced by *Lyngbya majuscula*, which is considered the most damaging skin irritant organism. Filaments of this cyanobacterium can become trapped under the swimming costume where they cause intense irritation and blistering. Besides being the major cause of dermatitis, all three toxins are tumor promoters (Falconer, 1999).

### • **Irritant toxins**

*Lipopolysaccharides* are recognized to cause fever in mammals and to be involved in septic shock syndrome.

Public health concern regarding cyanobacteria centres on the ability of many species to produce cyanotoxins. Cyanotoxins may fall into two of the four groups of water-related diseases. They may cause waterborne disease when ingested, and water contact disease primarily through recreational exposure (WHO, 1998). After bathing and showering in water containing cyanobacterial blooms, skin or oral mucosal contact might result in allergic reactions resembling hay fever and asthma, and skin, eye and ear irritations. Accidental ingestion of the toxins in recreational waters may result in gastro- or hepato-enteritis disorders (Pitois *et al.*, 2000). Recreational exposure to toxic cyanobacteria was assessed to be very likely at a large proportion of Germany's fresh water bathing sites (Chorus, 2002). However, epidemiologic studies are still rare.

Bathing at sites with *L. majuscula* revealed both increased skin and eye symptoms that were associated with an increased level of water exposure, and younger age groups and women reporting more symptoms than men (Osborne *et al.*, 2007). Less severe skin irritation is common after bathing in water contaminated by freshwater cyanobacteria, as is mild gastroenteritis (Pilotto *et al.*, 1997; Stewart *et al.*, 2006a; b).

Despite still growing research efforts there is hardly any evidence on possible long term adverse effects of chronic low dose cyanotoxin exposure on human health. Testing chronic effects of cyanotoxins on human lymphocytes *in vitro* revealed that the cyanotoxins were more harmful than benzene and sodium arsenite, suggesting that cyanotoxins may be a more serious environmental hazard than generally recognised (Repavich *et al.* 1990). There is mounting evidence supporting the association of the naturally occurring neurotoxin  $\beta$ -N-methylamino-L-alanine (BMAA) to neurodegenerative processes (Papapetropoulos, 2007). BMAA has been linked recently to a neurodegenerative disease and Alzheimer's disease (Cox *et al.*, 2003; Murch *et al.*, 2004; Cox *et al.*, 2005). Increased occurrence of cyanobacterial blooms and dense surface scums suggest a potential for widespread human exposure to this 'slow toxin', which has been found in 29 of 30 strains tested (Cox *et al.*, 2005).

Organisms responsible for cyanobacteria toxin poisonings include at least several dozens of species of which the most notorious ones are members belonging to the genera *Anabaena*, *Aphanizomenon*, *Cylindrospermopsis*, *Lyngbya*, *Microcystis*, *Nostoc* and *Planktothrix* (Carmichael, 2001). However, with ongoing developments in sensitive analytical methods, more and more previously unsuspected species appear toxigenic. Many species of planktonic cyanobacteria possess specialized intracellular gas vesicles, which are minute proteinaceous hollow cylinders filled with air. Together with a light driven accumulation or consumption of carbohydrates (ballast molecules) they enable the organism to regulate its buoyancy and thus to actively seek water depths with optimal growth conditions. The organisms may exhibit a diel migratory pattern. During the early morning surface scums are formed, while the scum dissipates during the day; by early evening the cyanobacteria are distributed through the water (Van Rijn and Shilo, 1985). In darkness carbohydrates are being used in the biochemical pathways giving the organisms a positive buoyancy, while in upper water layers in the light, through accumulation of photosynthates (ballast), the organisms become negatively buoyant (*Figure 3*). When biomass is high the formed surface layer may shade the cells below it still in the water column allowing the onset of a persistent layer. This process is enhanced significantly when mixing conditions in the water change from well mixed to more stagnant due to weather changes from windy to stable. Inasmuch as the regulation of buoyancy by changing the amount of gas vesicles is slow, organisms that are adapted to more turbulent mixing by enlarged gas vesicles will take a few days to reduce their buoyancy in order to adapt to more quiescent conditions (Chorus & Bartram, 1999).

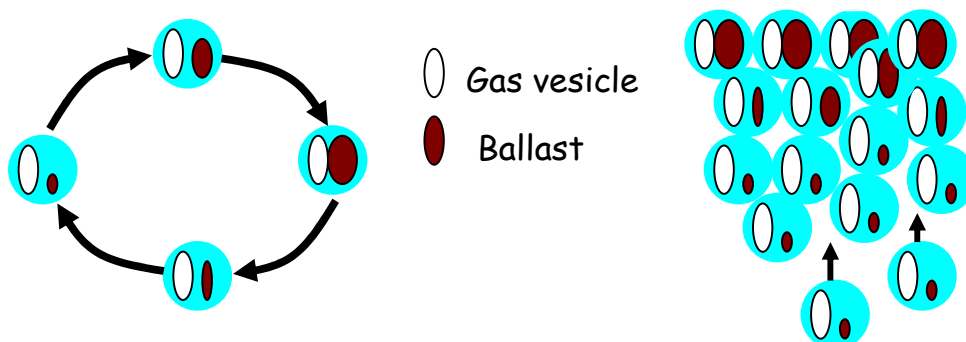


Figure 3. Vertical displacement of a cyanobacterium in the watercolumn as a consequence of production and consumption of ballast carbohydrates (left) and the formation of a surface-scum (right). (After Verspagen, 2006).

This ability of many frequently occurring toxigenic cyanobacterial species (e.g. *Anabaena* spp., *Aphanizomenon* spp., *Microcystis* spp.) to form scums implies a toxin concentration by a factor of 1,000 or more in a few hours. These scums may signify a high health risk for bathers and others involved in body-contact water sports (Chorus & Bartram, 1999). Acute hazardous situations occur when on the water surface accumulated cyanobacteria are blown together by wind, forming stable scums along downwind (leeward) shores (Figure 4).

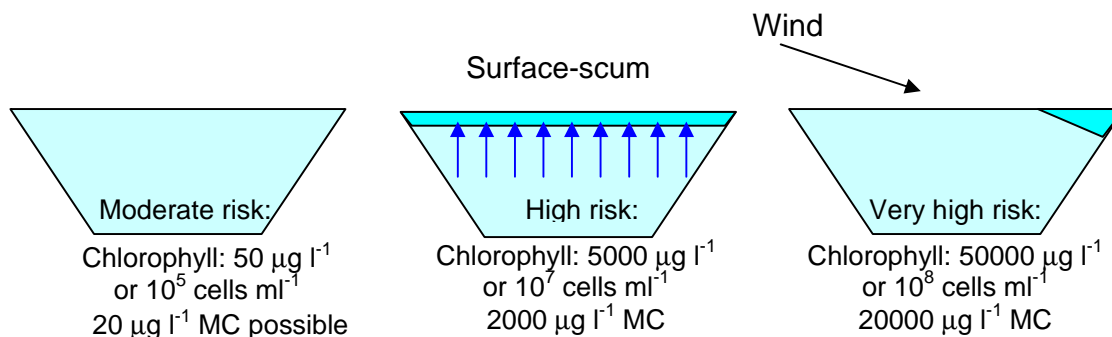


Figure 4. Schematic illustration of scum formation changing the cyanotoxin risk for adverse health effects from moderate (left), via high (middle) to very high (right). A concentration of cells from a 4 m watercolumn into a 4 cm surface layer implies a 100-fold accumulation to a high risk level scum (left to middle). A 1000-fold accumulation occurs when wind sweeps scums from 100 m into 10 m near the shores (after Chorus & Bartram, 1999).

Recreational water use is likely to be a major route of exposure to cyanotoxins in some parts of the world (Chorus & Bartram, 1999). Anecdotal evidence indicates that children, and even adults, may be attracted to play in scums (Figure 5).





Figure 5. Children playing on the shore of Lake Reeuwijk (2007).

The presence of scums caused by cyanobacteria is a readily detected indicator of a high risk of adverse health effects for those bathers who come into contact with the scum. Immediate action to control scum contact is recommended for such situations (Chorus & Bartram, 1999).

## 5.5 Effects of climate change on harmful cyanobacterial blooms

Long-term climate change may increase the frequency of heat waves and heavy rainfall events. Increased temperature is expected to result in increased mineralization and thereby nutrient availability (Weyhenmeyer, 2001). Heavy rainfall leads to deterioration in the quality of surface waters by potential sewage overflow and enhanced nutrient run-off. Because of a combination of increased nutrient concentrations and water temperature, it is expected that there may be an increase in the number of cyanobacterial blooms that could adversely affect the health of those engaged in recreational water contact (Hunter, 2003). Even slightly increased temperatures already could lead to higher biomass and dominance of cyanobacteria in some aquatic systems (Fernald *et al.*, 2007).

In general, a far greater number of calamities caused by cyanobacteria is registered in the tropics and regions with higher temperatures during the summer, which could be due to higher toxin production and the temperature dependence

of the toxic effects, including far higher exposure rates during summer. Increasing temperature from 22 to 28 °C resulted in 2-fold higher toxin quota in *Aphanizomenon* sp., which was observed even while the organisms displayed a higher growth rate and thereby possibly a higher toxin dilution through cell division (Dias *et al.*, 2002). However, in general, there is no clear effect of temperature on toxin production, which seems to vary substantially between species. Where some studies revealed similar temperatures for both maximum toxin production and growth rates (Wicks and Thiel, 1990; Lehtimäki *et al.*, 1994; Yin *et al.*, 1997; Park *et al.*, 1998), others have found that the optimum temperature for toxin production was lower than that for growth (Van der Westhuizen and Eloff, 1985; Watanabe and Oishi, 1985; Sivonen, 1990; Lehtimäki *et al.*, 1997; Hobson and Fallowfield, 2003).

In the Baltic Sea, high water temperatures and stable weather conditions favour the development of nitrogen-fixing cyanobacteria, and mass-occurrences formed by *Aphanizomenon flos-aquae*, *Nodularia spumigena* and *Anabaena* spp. Over the years, a significant increase in cyanobacterial biomass has been observed (Suikkanen *et al.*, 2007). It appeared that temperature (>16 °C) is the main factor determining the onset and intensity of blooms dominated by *N. spumigena* in the Baltic Sea (Wasmund, 1997; Kanoshina *et al.*, 2003). Also for the notorious *Microcystis* species the onset of blooms correlate with water temperatures that exceed 15 °C (Reynolds, 1973). Moreover, the increase in growth rate of cyanobacteria with temperature might be faster than that of other algae (Coles and Jones, 2000). Hence, global warming may favour cyanobacteria directly through the effect of elevated temperatures on physiological processes, reflected in considerably higher growth rates (Chu *et al.*, 2007).

Another aspect associated with the expected future climate conditions is a reduced turbulent mixing and concomitant enhanced stratification in deep lakes. Inasmuch as the heat wave of summer 2003 in central Europe had air temperatures similar to those predicted for an average summer during the late 21<sup>st</sup> century, this event allowed a “natural experiment” to assess how temperate lakes will react when exposed to the increased ambient summer air temperatures that will be encountered in a generally warmer world. It was found that surface temperature and thermal stability in summer 2003 were the highest ever recorded. The extremely high degree of thermal stability resulted in extraordinarily strong hypolimnetic oxygen depletion (Jankowski *et al.*, 2006). Such conditions will favour the proliferation of buoyant cyanobacteria in eutrophic lakes and elevate the chance for the development of surface blooms (Huisman *et al.*, 2004).

The predicted climate changes in The Netherlands include milder and wetter winters, and warmer springs (Van Vliet *et al.*, 2002; Van Oldenborgh & Van Ulden, 2003). Interestingly, in 14 out of 17 lakes studied in Europe after extremely mild winters the cyanobacterial biomass was found to increase considerably by a factor up to 100 (Weyhenmeyer *et al.*, 2002). In the largest natural French lake, Lac du Bourget, warmer than average winter/spring periods allowed an earlier water stratification and a competitive advantage to *Planktothrix*

*rubescens* leading to blooms (Jacquet *et al.*, 2005). Using regional climate change scenarios from South Sweden, Arheimer *et al.* (2005) predict that already in February/March cyanobacteria will reach a total biomass up to  $1.3 \text{ mg C l}^{-1}$ , which is several times exceeding the current situation. They also predict radical changes in lake biochemistry with up to 50 % increased concentrations of total phosphorus, and 20 % more total nitrogen (Arheimer *et al.*, 2005). Future scenarios for the Swedish Lake Erken indicate enhanced nutrient turnover rates, increased bacterial activity (mineralization) in sediment, and increased summer planktonic algal biomass that is dominated by cyanobacteria (Blenckner *et al.*, 2002). Based on modelling Mooij *et al.* (2007) conclude that global warming will increase the probability of a shift from a clear to a turbid state with predicted higher summer chlorophyll-a concentrations and a stronger dominance of cyanobacteria during summer. These indications are in concordance with the prediction that effects of climate change will mimic the effects of eutrophication due to an expected increase in the external nutrient loading (Kilham *et al.*, 1996).

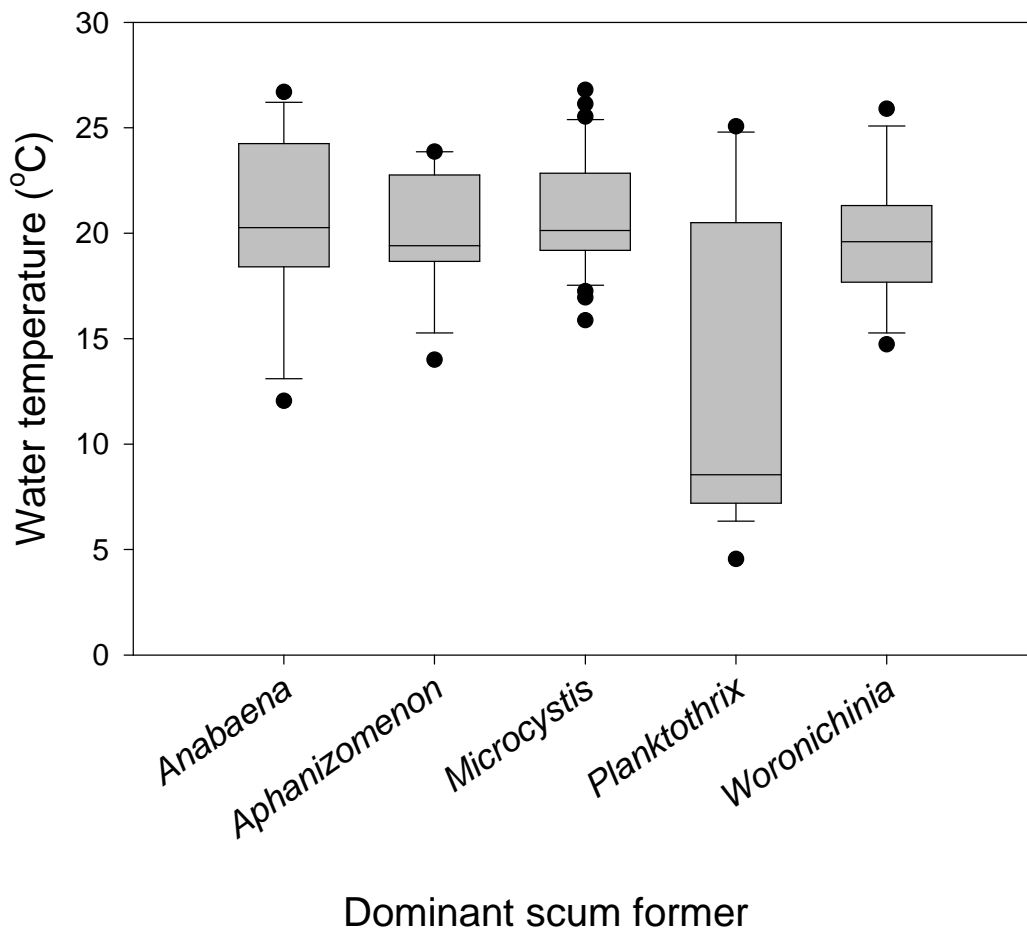


Figure 6. Box-plots of water temperatures at which surface scums of five most prominent cyanobacteria genera were found. Boxes show medians and 25th and 75th quartiles, bars indicate 10th and 90th percentiles, and dot symbols represent outliers.

In shallow lakes, a stronger dominance of phytoplankton is expected in future, as phytoplankton growth may be stimulated by higher internal P loading, which is highly sensitive to temperature (Søndergaard *et al.*, 2003; Jensen *et al.*, 2006). The P-loading of lakes is further expected to increase due to the expected increase of net precipitation in winter and an increase in extreme rainfall events (Mooij *et al.*, 2005). Inasmuch as the growing season is extended, the risk of prolonged periods with blooming of noxious cyanobacteria most likely increases (Jeppesen *et al.*, 2007).

Lower water temperatures favour *Planktothrix agardhii* development in comparison with *Aphanizomenon flos-aquae* and *Microcystis* spp. (Dokulil and Teubner, 2000). Analyzing data on scums, dominant cyanobacteria genus and water temperature gathered by the cyanoteam of the Aquatic Ecology & Water Management Group of WUR, revealed that *Planktothrix* forms surface scums at lower temperatures (*Figure 6*). Although *P. agardhii* is frequently described as a non-scum forming organism (e.g. Chorus & Bartram, 1999), this classification might be unjustified as in The Netherlands several scums of *P. agardhii* have been found (*Figure 7*). Thin paint-like accumulations along the shores have been observed regularly in autumn – spring, whereas thick flakes have been observed in summer (*Figure 7*). The closely related *P. rubescens* is a known scum former and despite it has been considered less hazardous to swimmers, because it presumably does not usually form scums during the recreational water use season (Chorus & Bartram, 1999), recent years situation in the Dutch bathing site Lake Rauwbraken shows otherwise (*Figure 7*).

The record warm year 2006 (*Figure 8*) allowed cyanobacterial blooms to persist even in winter thereby hampering recreational activities, as exemplified in *Figure 9*. Despite considerable drops in water temperatures on many locations relatively high cyanobacterial biomass remained in the watercolumns throughout winter. For Lake Rauwbraken this caused a delay in opening of the bathing site in 2007 by two months (*Figure 9*).



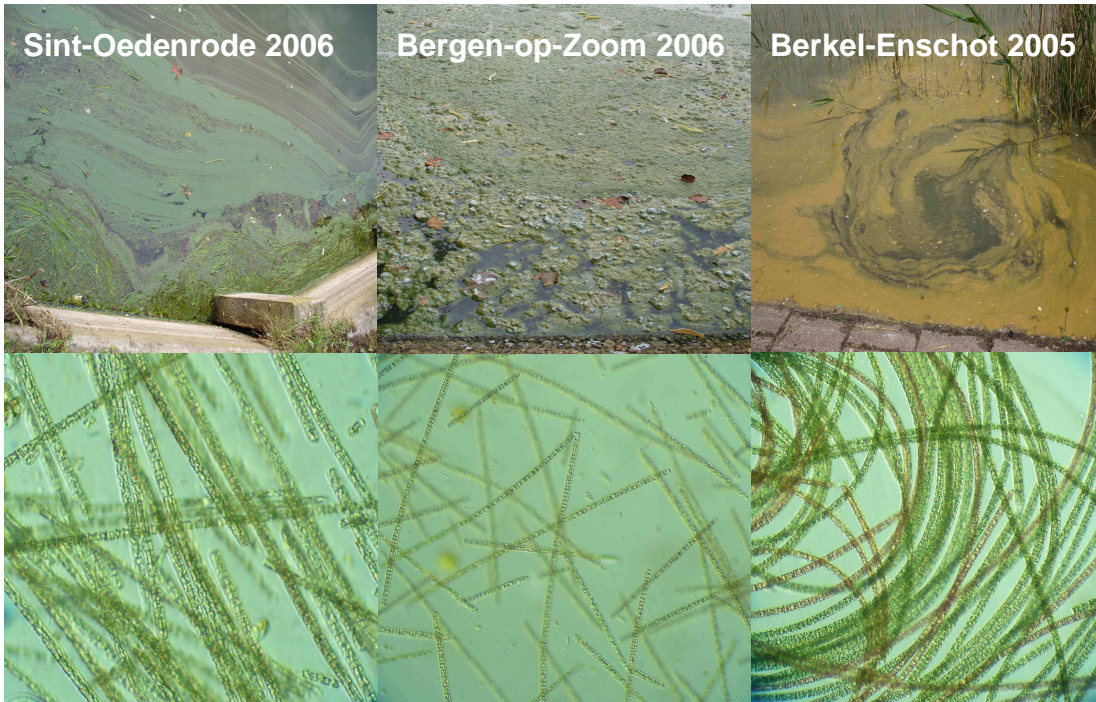


Figure 7. Pictures of cyanobacterial accumulations along the shore of two urban waters in 2006 (Sint-Oedenrode 20<sup>th</sup> October, Bergen-op-Zoom 27<sup>th</sup> July) and one bathing site in 2005 (9<sup>th</sup> June). The lower panel shows micrographs of the cyanobacteria at times of the bloom in 2006; for Berkel-Enschot close-up material is from a surface bloom of 21-12-2006.

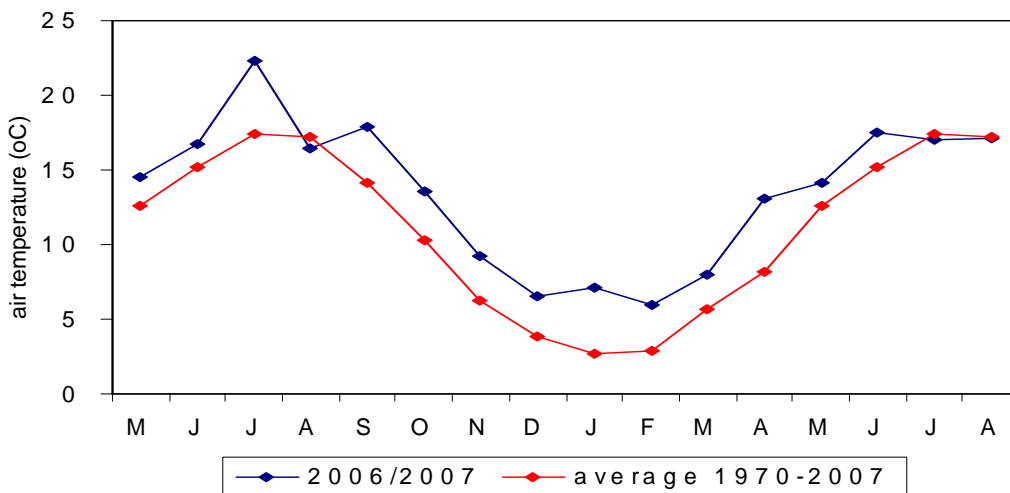


Figure 8. The air temperature in 2006/2007 compared to the average air temperature in the period 1970-2007 (source: KNMI).

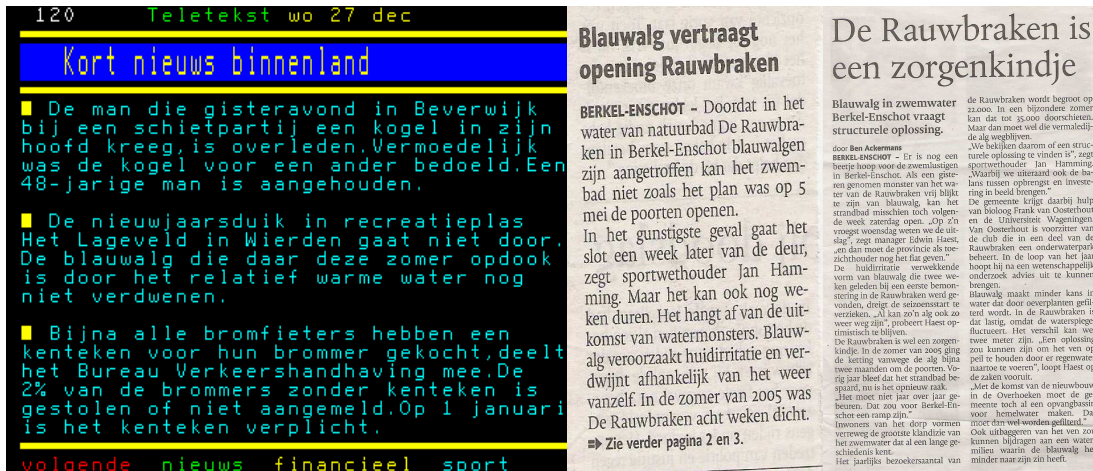


Figure 9. Teletext print of 27<sup>th</sup> December 2006 mentioning the prohibition of a New-Year's day swimming event (left panel), and newspaper messages on Lake Rauwbraken being closed due to cyanobacterial blooms in 2007.

Complaints of citizens living along the shores of water bodies with frequent occurrence of massive cyanobacterial blooms are mostly on the unpleasant odours. In the drinking water industry some relation with temperature and unwanted odiferous compounds has been found. The earthy/musty compounds, geosmin and 2-methylisoborneol, in particular are considered problematic as they can be detected in extremely low concentrations by the human nose (Young *et al.*, 1996). A significant correlation between water temperature and geosmin concentration was observed (Uwins *et al.*, 2007). Another study in two Taiwanese drinking water reservoirs revealed that the concentrations of the most prevalent odourant (2-methylisoborneol) and the most abundant cyanotoxins (microcystins) correlated well with air and water temperature and that they were higher in warmer seasons (Yen *et al.*, 2007). In 2006, the warm summer caused severe odour nuisance at several sites in The Netherlands (e.g. Almere-Haven, Tholen, Bergen-op-Zoom, Grave etc.).



Figure 10: *Cylindrospermopsis trichome* found in 2006 (Kienehoefvijver). H = heterocyte, A = akinete.

### *Invasion of new cyanobacteria species*

Ecological responses to recent climate changes, especially rising air and water temperatures, have been observed worldwide. In general, spring activities have begun to occur progressively earlier since the 1960s (Walther *et al.*, 2002). These changes may favour rapid and successful spread of species from tropical to temperate regions all over the world. In fact, the toxigenic cyanobacterium *Cylindrospermopsis raciborskii* has spread from the tropics to northern temperate zones during the last few decades (Padisák, 1997). This species is notorious as it can produce toxins, such as saxitoxins and cylindrospermopsin, and it has been associated with some poisoning events (Hawkins *et al.*, 1985). The presence of *C. raciborskii* in surface waters is regarded a potential risk for human health due to the potent hepatotoxicity and the assumed carcinogenicity of its toxins.

*C. raciborskii* has already formed mass developments in some northern habitats (Hungary, Austria, Canada) and was first detected in The Netherlands in 1999 in the Amstelveense Poel (Van den Hove, 2001). It has been concluded that the earlier increase in water temperature due to climate change has promoted the spread of the toxigenic *Cylindrospermopsis raciborskii* to the temperate zone. This change results in earlier germination and shifts the pelagic populations into a phase with higher light intensity, thereby promoting *C. raciborskii* growth and recurrence (Wiedner *et al.*, 2007). The same mechanism could also explain the northward shift of the biogeographic boundaries of two other cyanobacteria, *Anabaena bergii* and *Aphanizomenon aphanizomenoides*, which were first detected in several German lakes in 2004 (Stüken *et al.*, 2006).

In the survey of the cyanoteam of the Aquatic Ecology & Water Management Group of WUR, *C. raciborskii* has been found in a few urban waters in N-Brabant and Gelderland (*Figure 10*). Although the biomass of *C. raciborskii* was still low, its competitive superiority over other cyanobacteria (Soares, unpubl.), might enable it elbowing out other species. Infact, Dobberfuhl (2003) observed a dramatic decrease in phytoplankton diversity in Florida lakes after the introduction of *C. raciborskii*.

If the year 2006 is taken as an example of yet to come normal situations, some inferences can be made. Several aspects of the predicted climate warming will act in concert favouring cyanobacterial blooms:

- 1) The P-loading of lakes is expected to increase due to the expected increase of net precipitation in winter, an increase in extreme rainfall events, and higher internal loading.
- 2) In deep lakes reduced turbulent mixing, concomitant enhanced stratification and severe hypolimnion oxygen depletions are predicted.
- 3) Warmer winters and spring cause a prolonged growing season.
- 4) Climate change opens a window for invasive species.

Overall, the summer average water transparency can be reduced significantly (Fig. 4 *in* Hosper *et al.*, 2007); probably more measures will be needed to fulfill Water Framework Directive goals.

## CHAPTER 6. CONCLUSIONS AND RECOMMENDATIONS

Climate change in The Netherlands will result in favorable environments for a myriad of new pathogens and vectors. Changes in temperature and precipitation will be beneficial for the development of both groups of organisms, resulting in an increased risk for recreants using surface waters and their immediate surroundings.

Changes in temperature will have various effects on both pathogen and vector populations, thereby increasing their presence and the chance of infection. Apart from the effect of increased temperatures on the growth of algae and cyanobacteria, it can also influence the amount and type of harmful toxins. Eutrophication will enhance the formation of algal and cyanobacterial blooms as well as dense populations of pathogens. Increased rainfall will result in floodings, with elevated risk of pollution, more breeding places for vectors, and forcing vertebrate hosts into closer contact with men. A prolonged growing season gives more opportunities for pathogens, vectors, and harmful algae and cyanobacteria to develop large persisting populations and to be established permanently. There will be continually invasions by organisms new to The Netherlands; and the chance of permanency is high as the environmental conditions will improve for organisms originating from the (sub)tropics.

It is, therefore, important to anticipate in time the foreseen changes in health risks for bathers. And although health care in The Netherlands is of a high standard, extra measures have to be taken into account.

- The public must be aware of the increased risks associated with recreational activities in and around surface waters. This involves not only directed information beforehand on precautionary measures, but also uniformity in, for instance, warning signs near suspicious surface waters.
- The measures already taken to reduce eutrophication (reducing nutrient loading; improved water treatment) proved to be extremely successful. There is a risk that climate change will completely undo this success. So, measures to reduce the chance of pollution through sewery systems have to be taken, as well as measures to further reduce the risk of enhanced eutrophication of water bodies indicated as recreational bathing waters. This could have consequences for the Water Framework Directive.
- In the new European bathing water directive, cyanobacteria are included, but also harmful algae have to be taken into account. Furthermore, the two included microbiological indicators do not cover pathogens that cause skin irritation. Finally the directive should include the monitoring of vectors, such as malaria or dengue carrying mosquitos.



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## Appendix: Scientific terms explained

*cyanobacteria*: also known as Cyanophyta is a phylum (or "division") of Bacteria that obtain their energy through photosynthesis. They are often referred to as blue-green algae, although they are in fact prokaryotes, not algae.

*pathogen*: or infectious agent is a biological agent that causes disease or illness to its host.

*sequela* (plural: *sequelae*): pathological condition resulting from a disease, injury, or other trauma.

*sylvatic*: is a term referring to diseases or pathogens affecting only wild (sylvan = forest-dwelling) animals. In the context of animal research, its opposite is domestic, which refers to pets, farm animals or other animals which do not dwell in the wild.

*vector*: traditionally, an organism that transmits diseases or infections; by extension, any organism that transports foreign living material.

*zoonotic*: zoonosis is any infectious disease that is able to be transmitted (vectored) from other animals, both wild and domestic, to humans or from humans to animals (the latter is sometimes called reverse zoonosis).