2.2 Microbiological parameters – general

Water used as a source for a small supply of drinking water may be of unknown origin and come from a catchment prone to consistent or intermittent contamination by faecal material from domestic and farm animals, wildlife or sanitation systems such as septic tanks. As a consequence, there is a high probability of pathogenic micro-organisms being present in the source water, and adequate treatment must be applied before the water is used for domestic purposes. Because no single treatment can be expected to remove all types of pathogenic agents, a multiple barrier approach in the form of two or more sequential treatment processes is recommended.

The microbiological quality of drinking water has traditionally been assessed by monitoring for bacteria called faecal indicator organisms (coliforms, *E. coli*, and enterococci). The presence of these organisms is indicative of past faecal contamination and hence the possible presence of enteric pathogens. Although indicator organisms are generally adequate for monitoring purposes they cannot completely be relied on to indicate the absence of pathogens. This is especially true where a pathogen is environmentally more robust, or can survive treatment better than the indicators. In these circumstances the indicator may be absent even though low numbers of pathogens still remain.

An example of a pathogen with which such a discrepancy can occur is the protozoan parasite *Cryptosporidium*. This micro-organism is very much more tolerant of the action of disinfectants than faecal indicators such as *E. coli* because it forms resistant spore-like bodies called oocysts. If disinfection is the sole treatment process used, the numbers of *Cryptosporidium* oocysts will remain unchanged whereas the numbers of *E. coli* may be reduced to undetectable levels.

The use of filtration before disinfection is more effective in that an appropriate physical treatment process will remove oocysts. In addition filtration will remove much of the particulate material present which could otherwise reduce the effectiveness of disinfection by creating an oxidant demand and/or shielding microbes from the effects of the disinfectant. This is a good example of why a multiple rather than a single barrier approach is preferred. In addition the use of multiple barriers will allow some protection to remain even if one process fails.

Despite the possible shortcomings in the use of faecal indicators as a measure of the microbiological safety of a water supply, monitoring for coliforms and *E. coli* is still recommended and standards of quality are expressed in terms of these organisms. The reason for this is that monitoring for the pathogens themselves remains rather uncertain because methods of analysis are relatively insensitive and costly compared to those for faecal indicators. Additionally the absence of one pathogen will not guarantee the absence of others. Therefore *E. coli* and coliforms remain the best and most sensitive general measure of water quality.

Monitoring needs and treatment requirements should however be the subject of regular review and if a need is identified, through say an outbreak due to a specific pathogen, treatment strategies and monitoring requirements may need to be changed.

Blue-Green Algal Bloom

2.2 Microbiological parameters – general (continued)

Growth of algae can be a problem in some surface water sources and in uncovered water storage reservoirs. Algae are naturally present in all inland waters and concentrations can increase and decrease rapidly depending on climatic factors and the availability of essential nutrients. Algae can cause taste and odour in water. Some blue-green algae release toxins that may prejudice the recreational use of water. Although algal toxins have not been identified in drinking water supplies at concentrations that would affect health, it would be prudent to avoid the use of water sources that exhibit high concentrations of algae.

With the consent of the relevant environmental authorities, it may be possible in some circumstances to adopt measures to minimise the development of excessive algal blooms. The addition of iron salts to precipitate the nutrient phosphate is one measure, although it may not represent a long-term answer to the problem. In the past, dosing of copper sulphate has been used to reduce growth of algae in reservoirs but its efficacy is questionable. The forced circulation of reservoirs to keep water mixed is another measure. Blooms of algae occur mainly at the surface of reservoirs whereas water is abstracted at depth. Some forms of water treatment, including ozonation, activated carbon adsorption and addition of potassium permanganate can be effective in removing algal toxins. Algae are normally removed as particulate matter during water treatment but some algae can pass through filters or reduce the efficiency of filtration or sedimentation systems.

In 2002 the Scottish Executive Health Department published revised guidance complied relating to all aspects of Cyanobacteria. The document “Blue-Green Algae (Cyanobacteria) in Inland Waters: Assessment and Control of Risks to Public Health”[1] should be consulted for further information on this topic.

[1] www.scotland.gov.uk/library5/environment/bgac.pdf

2.3 Micro-organisms associated with outbreaks and private supplies

2.3.1 Review of outbreaks

Fewtrell *et al*[2]. tested samples from 91 private supplies in the UK, and found the microbiological quality of the water in small supplies to be generally poor, with almost 50% of the supplies failing to meet the required quality standards on at least one occasion. They concluded that, given the high level of sanitary failures, there was the possibility that harmful micro-organisms could be present, and that a significant risk to health could not be discounted.

Poor microbiological quality of groundwater systems has caused many disease outbreaks in the USA. Between 1971 and 1994, 58% of U.S. waterborne disease outbreaks (356 in number) were caused by contaminated groundwater systems – 70% of these outbreaks were considered to be due to contamination of the groundwater source as opposed to the distribution system[3]. Being generally smaller and less well-equipped than public supplies, and being subject to less stringent surveillance and regulatory requirements, private supplies by their very nature are more likely to suffer water quality failures. In the UK 18 outbreaks of waterborne disease were associated with private water supplies between 1970-94 in which over 1,388 people were affected in England and Wales[4]. The figure is almost certainly an underestimate as no data from 1987-93 were available and because of inherent under-reporting problems. In Scotland, private supplies caused 21 out of 57 waterborne disease outbreaks between 1945 and 1987 (37%)[5]. These 21 outbreaks gave rise to at least 9,362 cases[6].

Furtado *et al*[7]. have reviewed outbreaks of disease associated with both public and private water supplies in England and Wales between 1992 and 1995. In this period ten outbreaks were associated with public supplies, and nine with private supplies. The pathogen responsible for all ten outbreaks from public supplies was *Cryptosporidium*. In contrast, the most common pathogen associated with private supplies was *Campylobacter*, along with *Cryptosporidium* and *Giardia*. In one outbreak the causal agent was not identified.

2 Fewtrell, L., Kay, D. and Godfree, A. (1998). The microbiological quality of private water supplies. *Journal of the Chartered Institution of Water and Environmental Management* **12** (1), 45-47.

3 Craun, G.F. and Calderon, R.L. (1997). Microbial risks in groundwater systems: epidemiology of waterborne outbreaks. In: *Under the microscope. Examining microbes in groundwater.* American Water Works Association Research Foundation, Denver Colorado.

4 Fewtrell, L. and Kay, D. (1996). *Health risks from private water supplies.* CREH report EPG 1/9/79, University of Leeds, Leeds.

5 Benton, C., Forbes, G.I., Paterson, G.M., Sharp, J.C.M. and Wilson, T.S. (1989). The incidence of waterborne and water-associated disease in Scotland from 1945-1987. *Water Science and Technology* **21**, 125-129.

6 Lamb, A.J., Reid, D.C., Lilly, A., Gauld, J.H., McGaw, B.A. and Curnow, J. (1998). *Improved source protection for private water supplies: report on the development of a microbiological risk assessment approach.* School of Applied Sciences, Robert Gordon University, Aberdeen, 86pp.

7 Furtado, C., Adak, G.K., Stuart, J.M., Wall, P.G., Evans, H.S. and Casemore, D.P. (1998). Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992-5. *Epidemiology and Infection* **121**, 109-119.

This difference is striking, and is most likely to reflect the differences in treatment strategies between public and private supplies. Public supplies usually employ a multiple barrier treatment strategy, normally with chlorination or possibly some other form of disinfection as a final stage. Any disease-causing organism which may have survived or broken through the conventional water treatment processes is therefore exposed to disinfection as a final barrier to prevent its entry into the distribution system. Provided that it is susceptible to the disinfectant, it will cause no problems. However, *Cryptosporidium* oocysts are highly resistant to chlorination at the levels applied in water treatment, and consequently this organism appears to be the most troublesome in public water supplies.

With private supplies a variety of treatment strategies are used, but often there is no treatment at all, and disinfection is both much less prevalent and can suffer from problems of unreliability. This allows the possibility for outbreaks to be caused by organisms that could easily be inactivated if some form of disinfection was employed. *Campylobacter* is such an organism. It is more susceptible to chlorine, ozone and ultraviolet (UV) irradiation than indicator organisms such as coliforms and *Escherichia coli*, but if there is no disinfection it can still cause outbreaks of disease, as has been observed with private supplies.

Table 2.1 summarises the outbreaks associated with private water supplies in the UK over the 25-year period from 1970 to 1995. This is not to say that these were the only outbreaks, but they are the ones that have been documented and have been subject to epidemiological investigation.

The following sections give a more detailed description of the micro-organisms that have been responsible for the outbreaks shown in Table 2.1.

2.3.2 Campylobacter species

The genus *Campylobacter* includes fourteen species with the most important human pathogens being the thermophilic species: *C. jejuni, C. coli* and *C. upsaliensis. Campylobacter jejuni* is a major cause of gastrointestinal illness and is common in the faeces of a wide variety of wild and domesticated animals. In England, Campylobacters are the main causes of food-related gastroenteritis. *C. jejuni* may adopt a dormant state with modified metabolism known as 'viable but non-culturable'. The stimulus for the adoption of this state is stress, which includes the starvation conditions found in water.

The natural habitat of *Campylobacter* species is the intestinal tract of warm-blooded animals, particularly pigs and poultry. Faeces from infected animals will contain extremely high numbers of *Campylobacter* (several million per gram). Contamination of water sources should therefore be regarded with concern since the infective dose is relatively low, and 500 organisms have been shown to initiate infection. Most infections are relatively mild and resolve within a few days without medical intervention. However, serious complications can occur, particularly in the old or very young.

Although *Campylobacter* does not multiply readily outside its natural habitat, survival in water can be protracted. They are more sensitive than *E. coli* to chlorine, ozone and UV at the levels normally applied as part of water treatment, so any viable organisms passing through the earlier stages of water treatment should not enter the supply. However, the action of disinfectants can be hindered if the water has not been conditioned adequately by the removal of dissolved organic material and particulates. The latter can have a shielding effect particularly where UV disinfection is being used.

Compliance with the normal standard of 0 *E. coli* in 100 ml should provide adequate protection. However between 1981 and 1994 there were nine recorded outbreaks involving nearly 700 cases associated with private water supplies.

Table 2.1 Summary of outbreaks of disease associated with consumption of water from private supplies over the period 1970 to 1995

Pathogen	Number of outbreaks	Total cases
Campylobacter species	8	>647
Cryptosporidium	2	15
Cryptosporidium and *Campylobacter*	1	43
Escherichia coli serotype O157	1	4
Giardia	1	31
Paratyphoid fever	1	6
Streptobacillary fever	1	304
Viral gastroenteritis	3	>998
Unknown	1	51
Total:	**19**	**>2,099**

Compiled from:

Fewtrell, L. and Kay, D. (1996). *Health risks from private water supplies*. Report No. EPG 1/9/79. Centre for Research into Environment and Health, University of Leeds.

Furtado, C., Adak, G.K., Stuart, J.M., Wall, P.G., Evans, H.S. and Casemore, D.P. (1998). Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992-5. *Epidemiology and Infection* **121**, 109-119.

Galbraith, N.S., Barrett, N.J. and Stanwell-Smith, R. (1987). Water and disease after Croydon: A review of water-borne and water-associated disease in the UK 1937-86. *Journal Institution of Water and Environmental Management* **1**, 7-21.

2.3.3 *Cryptosporidium* species

Cryptosporidia are unicellular protozoan parasites. Although many species are currently recognised, infecting a wide range of animals, *C. parvum* is thought to be the only species to cause the diarrhoeal disease cryptosporidiosis in humans. The incubation period (from infection to onset of symptoms) is 7 to 10 days. Person to person spread does occur. Infection is initiated by ingestion of the transmission stage which is a small (5 μm), round, resistant structure called an oocyst. Infected humans can excrete 10^8 oocysts per day and calves and lambs can excrete 10^9 oocysts per day. The broad range of hosts, high outputs from infected individuals and environmental resistance ensure a high level of contamination in the environment. Cattle, sheep and human sewage are the main sources of contamination of private water supplies.

Cryptosporidium oocysts have been detected in river, canal, lake and reservoir waters, filter back wash waters, sewage effluents and in some groundwaters. The concentrations fluctuate considerably. 'Spikes' may occur in river waters after periods of heavy rainfall. Oocyst concentrations between 7 and 48,400 per 100 litres have been reported in raw surface water sources in the USA. Higher loadings have been reported in water receiving agricultural drainage.

In sporadic cases (as distinct from outbreaks) the number of *C. parvum* infections is highest in children under five years of age. Most infections are reported during the spring and early autumn, the former probably coincidental with the lambing and calving season, and the latter with increased rainfall.

Physical treatment processes (coagulation, sedimentation and filtration) can provide an effective barrier to the parasite. Removal efficiencies of 99.8% for *C. parvum* oocysts by coagulation, clarification and sand filtration have been reported for efficiently operated systems. Membrane filters with sufficiently small pore sizes will effectively remove oocysts. Chlorination at the concentrations used in water supply does not inactivate oocysts. Disinfectants used in combination or sequentially may enhance disinfection activity against *C. parvum* and certain forms of UV treatment may be successful. However, very few published disinfection studies have been performed under conditions that are representative of actual high-risk conditions. A combination of catchment control, physical barriers and disinfection is required to provide protection from the organism.

Private supplies derived from surface waters and from some springs will be vulnerable to contamination with oocysts particularly in agricultural catchments. Here the use of a filtration stage in treatment is advisable to physically remove oocysts of *Cryptosporidium*, with disinfection aimed at inactivating any remaining viruses and bacteria.

[8] CREH (2001). Report on the incidence of *Cryptosporidium* in water, Research Contract DWI 70/2/129.

[9] Hancock, C., Rose, J.B. and Callahan, M. (1997). The prevalence of *Cryptosporidium* and Giardia in US groundwaters. *Proceedings International Symposium* on Waterborne *Cryptosporidium*, Ed Fricker, C.R., Clancy J.L. and Rochelle P.A. American Water Works Association, Denver, CO, 147-152.

2.3.3 *Cryptosporidium* species (continued)

Clapham reported that *Cryptosporidium* oocysts were found at one time or another in 60% of 15 private water supplies in the Bradford area, which had been monitored over roughly a 3-month period[10]. Oocysts were detected in 21 of a total of 150 samples taken (14%). These supplies were monitored after rainfall events (when the likelihood of oocysts entering supplies is thought to be increased) during the winter season (December to February not usually a peak time for *Cryptosporidium* infections). This study clearly demonstrated that *Cryptosporidium* oocysts (and also *Giardia* cysts) can enter high-risk private supplies when conditions permit.

A more recent study[8] monitored seven private water supplies in the UK for a range of micro-organisms including *Cryptosporidium* and *Giardia*. A monitoring cabinet was installed at each site and daily samples of 1,000 litres were taken. Each site was monitored daily for six weeks in two phases, May to June 2000 and October and November 2000. The results are summarised in Table 2.2. Microbiological water quality deteriorated following heavy rainfall. At Site 7, filtration and electrochlorination were installed between Phases 1 and 2 but cysts were still found during extreme weather conditions.

Contamination of groundwater by *Cryptosporidium* and *Giardia* was reported in 12% of 199 groundwater sites in the USA, with the majority of positive detections in springs, infiltration galleries and horizontal (collector) wells[9]. Reports of *Cryptosporidium* and *Giardia* in groundwater in the UK are sparse, in part because high cost and difficult analytical procedures have made sampling very sporadic in public and private supplies alike.

In the Third Report of the Group of Experts' report on *Cryptosporidium* in Water Supplies[11] further advice on private water supplies is given in Chapter 10 of that Report. The advice contained in the Report is implicit in the guidance given throughout this manual and the reference to the Report is provided for completeness.

[10] Clapham, D. (1997). *Cryptosporidium incidence in private water supplies and correlatory indicators*. Directorate of Housing and Environmental Protection, Bradford Metropolitan District Council.

[11] *Cryptosporidium in Water Supplies*. Third Report of the Group of Experts to Department of the Environment, Transport and the Regions & Department of Health. (1998) HMSO ISBN 1 85112 131 5

Table 2.2 Cryptosporidium and Giardia in seven private supplies in the UK

Site		Samples found to contain cysts (%)			
		Cryptosporidium		*Giardia*	
		Phase 1	Phase 2	Phase 1	Phase 2
1	SW England	2.4	0.0	2.4	0.0
2	Scotland	75.0	60.0	50.0	42.5
3	S Wales	2.4	2.4	56.6	50.0
4	N Ireland	0.0	0.0	2.4	12.1
5	Scotland	33.3	15.4	56.0	65.9
6	S Wales	0.0	0.0	10.0	29.0
7	Yorkshire	52.2	57.7	91.0	84.4

2.3.4 *Giardia* species

Giardia are a group of flagellate protozoans which grow in the intestinal tracts of both vertebrates and invertebrates. *G.duodenalis* infects more than 40 species of vertebrates, including humans. It causes a diarrhoeal disease in humans and between 4,000 and 7,000 cases are reported in England and Wales each year. The disease can be transmitted by direct contact with infected animals and humans, or by consumption of water, food or beverages contaminated by the faeces of infected humans or animals. *Giardia* forms cysts that are infectious and these survive well in aqueous environments. The cysts are oval in shape and 9 to 12 μm in length. A large number of animals (including humans) are potential carriers of *Giardia* infectious to humans. In the UK, the organism is endemic in sheep, cattle, rodents, cats and dogs. Birds have also been suggested to be a potential reservoir of *Giardia* infectious to humans.

The cysts survive well in aqueous environments and show greater resistance to UV, chlorine, and ozone than bacteria and viruses, but they are less resistant than *Cryptosporidium* oocysts. *Giardia* cysts are inactivated by boiling or pasteurisation. Normal coagulation, sedimentation and filtration processes, if operated correctly, should achieve at least a 3-log (99.9%) removal of cysts.

Outbreaks associated with drinking water can occur where human or animal faeces contaminate the supply and there is inadequate treatment, filtration or chlorination. In the UK the threat to public supplies is regarded as being minimal due to the use of multiple treatment barriers. Post treatment contamination, such as could occur after repairs to mains that are not adequately cleaned and disinfected before recommissioning, could still be a particular risk.

Private supplies should be regarded as being at greater risk if the catchment is prone to contamination by the faeces of animals, if there is a rapid route for recharge to reach the raw water intake and if there are inadequate treatment barriers. Table 2.2 in Section 2.3.3 shows the results of sampling seven private water supplies in the UK for *Giardia*.

2.3.5 *Escherichia coli* serotype O157

Escherichia coli (*E. coli*) is a bacterium, most strains of which live harmlessly in the gastro-intestinal tracts of people and animals. However, a few types have acquired virulence factors. These organisms are often harmful to people and can cause severe disease. Particularly important factors are toxins. One group of toxins was originally recognised by their ability to kill cultured Vero cells, (African green monkey kidney cells) hence the name Verocytotoxins (VTs). The toxin-carrying *E. coli* therefore became known as Verocytotoxin-producing *E. coli* (VTEC).

In the laboratory *E. coli* of all kinds are classified by identifying the antigenic (antibody producing) structure of two different molecules on their surface, O and H. There are more than 170 O serogroups. These can be subdivided into H serotypes. In the UK the overwhelming majority of VTECs causing human disease fall into serotype O157:H7, although other serotypes are not looked for in most laboratories. In addition to VTEC belonging to serogroup *E. coli* O157, other groups such as O111, O26, O103 and O145 have been identified as emerging pathogens throughout Europe.

Although verocytotoxin genes are necessary to cause serious disease, they alone are not sufficient. Indeed, many other *E. coli* strains have these genes but are not particularly pathogenic. Additional virulence factors are needed. In *E. coli* O157, the best studied example, some are coded by genes on a length of DNA in the bacterial chromosome called a pathogenicity island; they include proteins that help the organisms to adhere to the large bowel wall. Others are coded by genes on a plasmid (which facilitates the exchange of genetic material between bacteria). The most common kind of severe disease caused by these organisms is haemorrhagic colitis and so as a group they have been called enterohaemorrhagic *E. coli* or EHECs.

On an evolutionary time scale *E. coli* O157 (and other VTEC) are brand new, the first outbreaks caused by them occurring less than twenty years ago. It was first identified as a cause of human illness in 1982 in the USA, and there have since been numerous reports world-wide of infection with the organism.

The infectious dose (the number of bacteria necessary to produce an infection) of *E. coli* O157 appears to be very low, probably less than 100 organisms and possibly as low as 10. People can become infected through contact with the faeces of infected animals, by passing the organism from person to person, or by the consumption of contaminated foods or water.

2.3.6 *Streptobacillus* species

One outbreak of Streptobacillary fever has been associated with a private water supply. Over 300 people were affected and the illness was thought to be due to contamination of a spring source by rats.

Streptobacillary fever is a rare infection in the UK, and is caused by a bacterium called *Streptobacillus moniliformis*. This organism is normally associated with rat bites causing an illness termed rat-bite fever. However the bites of infected mice, squirrels, weasels, dogs and cats have also been recorded as causes of the illness. *Streptobacillus moniliformis* occurs in animals as a commensal inhabitant of the nasopharynx, but can occasionally be excreted in urine.

Haverhill fever is a form of Streptobacillary fever that is normally connected with direct contact with animals. It was named after the first recorded epidemic of the disease in Haverhill (Massachusetts, USA) caused by consumption of raw milk and milk products. An outbreak in the UK occurred at a boarding school in Essex in 1983. Although this was at first thought to be caused by the school's supply of unpasteurised milk, further case-controlled studies gave a strong association with the school's private water supply. This was a spring source where there was evidence of rat infestation. This source was used for the hot water supply in the school, but engineering investigations showed the possibility of cross-connection with the potable supply.

Streptobacillus moniliformis is a Gram negative bacterium, which generally occurs as short coccobacillary forms or rods (0.5 μm in length). Its small size may allow passage through water treatment filters particularly if there has not been a coagulation stage. The organism is pleomorphic and so should be susceptible to disinfectants. It can however occur in chains of filaments, which may increase its tolerance of disinfection.

2.3.7 Paratyphoid fever

Notifications of paratyphoid fever in the UK decreased about four-fold from the late 1930s to the 1980s[12]. Information collected since the early 1970s has shown that roughly half the cases of paratyphoid fever in the UK were caused by *Salmonella paratyphi A*, 95% of these being contracted abroad, and half were caused by *S. paratyphi B*, with over 70% of these being infected abroad. There have been no reported cases or outbreaks of water-borne disease due to *S. paratyphi A.*

Paratyphoid B fever is usually food-borne, but two water-borne outbreaks have been recorded since 1970, consisting of 96 cases, with no deaths. One of these outbreaks involved a private water supply. This was an unpublished PHLS report of six cases in Herefordshire in 1975. These resulted from consumption of well water that was contaminated by leakage of sewage from a domestic drain close to the well.

Salmonellae would be expected to respond to water treatment processes in a similar way to coliform organisms and *E. coli*, and would have a similar susceptibility to chlorination or other disinfection processes. A well-maintained multiple barrier system would therefore give good protection. As salmonellae, if present in raw water at all, would be less numerous than coliforms and *E. coli*, the absence of these indicator organisms should give a good assurance of the absence of this particular pathogen.

2.3.8 Enteric viruses

There are several types of viruses, which can be present in faeces that can cause viral enteritis, notably, the rotaviruses, the Noroviruses, and members of the enterovirus group. There have been several relatively large outbreaks (each of more than 100 cases) of viral enteritis associated with private water supplies derived from surface water. In each of these there was evidence of faecal contamination of the supply which had either been inadequately chlorinated, or not disinfected at all. Birds also carry viruses and can contaminate reservoirs and open cisterns.

Although filtration will remove viruses to some degree the primary treatment barrier is disinfection, either by chlorine or UV irradiation. As stated earlier, for these to be most effective waters must be conditioned by filtration (to remove particulates and aggregates of viruses) before disinfection. Where disinfection is the only treatment being employed, it must be maintained and its performance monitored regularly to optimise disinfectant activity for the prevailing water conditions.

[12] Galbraith, N.S., Barrett, N.J. and Stanwell-Smith, R. (1987). Water and disease after Croydon: A review of water-borne and water-associated disease in the UK 1937-86. *Journal of Institution of Water and Environmental Management* **1**, 7-21.

2.3.9 Blue-green algae (Cyanobacteria)

Blue-green algae can occur in quantity in lochs, lakes, ponds, canals and reservoirs. While usually green or blue-green in colour, they may be blue, black, dark brown or red.

When present in high concentrations, colonies of blue-green algae can often be seen with the naked eye; they may resemble fine grass cuttings or take the form of small irregular clumps or pinhead-sized spheres. Blue-green algae in high concentrations in the water column form "blooms" and, when blown on to a downwind shore, form scums which may be centimetres thick. Scums may also be seen in slow-flowing rivers and streams downstream from lochs or lakes.

Blue-green algae may also grow on the bottom of shallow water bodies and on shoreline rocks. They occasionally form thick gelatinous mats which may be exposed as the water level falls or may detach from the bottom and reach the shoreline. These mats are usually very dark in colour (black, dark brown or green) and cohesive and are sometimes mistaken for sewage.

Some types of algae, for example filamentous algae, occasionally form surface scums and growths of some water plants, particularly duckweed, might be mistaken for blue-green algae.

Surveys in different parts of the world have found that between 45% and 90% of blooms of blue-green algae produce toxins. These toxins are largely retained within the blue-green algal cells during their development and growth phases and are released, in the main, on cell death.

Blue-green algae of several genera can produce a range of toxins including neuro- and hepatatoxins and lipopolysaccharides. An algal bloom may contain more than one species, each producing different toxins. In addition, the toxicity of one species might change over time to a pattern that night vary for different places on a particular water body. Evidence of toxicity comes from reports of the effects of exposure of people and of animals to algal blooms and from laboratory investigations of algal blooms.

Algal blooms are inherently complex and assessment of the associated risks to public health is not straightforward. Such assessments should therefore take account of specialist advice such as that identified in "Blue-Green Algae (Cyanobacteria) in Inland Waters: Assessment and Control of Risks to Public Health"[1].