



## Canadian Water Quality Guidelines for the Protection of Aquatic Life

## DISSOLVED GAS SUPERSATURATION

**D**issolved gas supersaturation (DGS) is a condition that occurs when the partial pressures of atmospheric gases in solution exceed their respective partial pressures in the atmosphere. Individual atmospheric dissolved gases (oxygen, nitrogen, and trace gases such as argon and carbon dioxide) can often be supersaturated without adverse effects on aquatic and marine organisms. When the sum of the partial pressures of all dissolved gases exceeds atmospheric pressure, however, there is the potential for gas bubbles to develop in water and in the aquatic organisms that inhabit the water. This causes a condition known as gas bubble trauma (GBT).

DGS can result from various anthropogenic and natural causes. Hydroelectric and impoundment dams are known to cause high levels of DGS. Other sources include warm water discharges from cooling facilities (e.g., nuclear and fossil fuel powered generating plants), oxygen production by aquatic plants (enhanced by nutrients associated with industrial effluents, municipal discharges, and agricultural runoff), natural solar heating of water bodies, injection of air into pumping systems, supplemental oxygen in hatcheries, and air lift reaeration systems.

Various methods are used for calculating and reporting dissolved gas levels throughout the literature dealing with DGS and GBT in fish. For the purpose of developing water quality guidelines, DGS is defined by delta P ( $\Delta P$ ), which is the difference between total dissolved gas pressure and atmospheric pressure. Total gas pressure (TGP) is the sum of the partial pressures of all dissolved gases plus the vapour pressure of water at the prevailing water temperature.

$$\Delta P = \text{TGP} - p_{\text{Atm}} \quad (1)$$

$$\text{TGP} = p_{\text{N}_2} + p_{\text{O}_2} + p_{\text{H}_2\text{O}} \quad (2)$$

where

- $\Delta P$  = dissolved gas tension
- TGP = total gas pressure (mm Hg)
- $p_{\text{Atm}}$  = atmospheric pressure (mm Hg)
- $p_{\text{O}_2}$  = partial pressure of dissolved oxygen (mm Hg)

- $p_{\text{N}_2}$  = partial pressure of dissolved nitrogen, argon, and other trace atmospheric gases (mm Hg)
- $p_{\text{H}_2\text{O}}$  = vapour pressure of water (mm Hg)

Another common form of reporting gas tension is percent total gas tension (TGP%) of atmospheric pressure.

$$\text{TGP}\% = 100\% \times (\text{TGP} \div p_{\text{Atm}}) \quad (3)$$

For more information, see the comprehensive review by Fidler and Miller (1997), which is used as the supporting document for this guideline.

### Environmental Levels

Clark (1977) found dissolved gas levels in the Columbia River, British Columbia, below the Hugh Keenleyside Dam to be above 200 mm Hg ( $2.67 \times 10^4$  Pa) at many times throughout the year. Thermal effluent discharges have contributed to elevated levels of DGS in Okanagan Lake, British Columbia, and Wabamum Lake, Alberta. While the biological effects of DGS were not observed in Okanagan Lake where effluent was discharged below compensation depths, GBT was exhibited by fish attracted to the warm water plume under the ice cover of Wabamum Lake. Although dams and thermal discharges are a major source of DGS in some water bodies, there are numerous rivers and lakes where  $\Delta P$  levels range from 40 to above 76 mm Hg ( $5.33 \times 10^3$  Pa to above  $1.01 \times 10^4$  Pa; TGP%  $\approx 105$ – $109\%$  at sea level). Clark (1977) reported natural  $\Delta P$  levels well above 76 mm Hg in the Fraser River, British Columbia, at Lillooet, above and below Hells Gate, at Yale, at Hunter Creek, and at Agassiz.

High levels of DGS have been reported in other parts of Canada as well. GBT was identified as the cause of large mortalities in Atlantic salmon and eels below the hydroelectric dam on the Mactaquac River, New Brunswick (MacDonald and Hyatt 1973; Penney 1987). Dissolved oxygen levels ranged from slightly above to slightly below saturation. Dissolved nitrogen levels ranged from saturation levels to 127% saturation levels. In Manitoba, DGS levels 1.7 times the atmospheric saturation value (resulting from solute freeze-out) in

frozen shallow lakes caused significant levels of mortality in rainbow trout (*Oncorhynchus mykiss*), which had been stocked through the ice (Lark et al. 1979; Mathias and Barica 1985).

### Biological Effects

Gas-bubble disease in fish has been attributed to excess carbon dioxide, oxygen, and nitrogen gases in water. Fish characteristically develop bubbles under the skin, in the fins, tails, and mouth, behind the eyeballs (pop-eye disease), and in the vascular system, causing gas embolism and death. Bubbles may develop in yolk sac fry, causing them to float upside down; surviving fry frequently show white spots (coagulated yolks) due to gas bubble (Rucker 1972).

The major signs of GBT that cause death or high levels of stress in fish are

- bubble formation in the cardiovascular system, causing blockage of blood flow and death (Jensen 1980; Weitkamp and Katz 1980; Fidler 1988);
- overinflation and possible rupture of the swim bladder in young (or small) fish, leading to death or problems of overbuoyancy (Shirahata 1966; Jensen 1980; Fidler 1988; Shrimpton et al. 1990a, 1990b);
- extracorporeal bubble formation in gill lamella of large fish or in the buccal cavity of small fish, leading to blockage of respiratory water flow and death by asphyxiation (Fidler 1988; Jensen 1988); and
- subdermal emphysema on body surfaces, including the lining of the mouth; blistering of the skin of the mouth may also contribute to the blockage of respiratory water flow and death by asphyxiation (Fidler 1988; White et al. 1991).

### Toxicological Studies with Freshwater Species

A comprehensive review by Fidler and Miller (1997) reveals that lethal signs of GBT vary with species as well as water  $\Delta P$ , which in turn varies with water  $pO_2$  and depth. Also, a number of distinct thresholds for GBT mortality may exist for a given species. Mortality seems to

occur suddenly, sometimes with few nonacute symptoms preceding death at high levels of DGS.

Sockeye salmon (*O. nerka*) were found to have a threshold  $\Delta P$  of 125 mm Hg ( $1.67 \times 10^4$  Pa). Cutthroat trout (*Salmo clarki*) and steelhead trout (*O. mykiss*) appear to have a definitive threshold at a  $\Delta P$  of  $\approx 115$  mm Hg ( $1.53 \times 10^4$  Pa) and a probable lower threshold at a  $\Delta P$  of 76 mm Hg ( $1.01 \times 10^4$  Pa). Chinook salmon (*O. tshawytscha*) are found to have thresholds in the range of  $\Delta P \approx 130$ –140 mm Hg ( $1.73$ – $1.87 \times 10^4$  Pa) and 76–78 mm Hg ( $1.01$ – $1.04 \times 10^4$  Pa). The indication that there may be more than one threshold for mortality implies that different mechanisms are responsible for mortality.

Bentley et al. (1976) examined the effects of DGS on squawfish (*Ptychocheilus oregonensis* Richardson). Fish weighing 534 g (364 mm in length) were held in water at a  $\Delta P$  of 76 mm Hg ( $1.01 \times 10^4$  Pa) without mortalities over a 12-d period. When water  $\Delta P$  was increased to 129 mm Hg ( $1.72 \times 10^4$  Pa), a 10% mortality occurred in 4.8 d. After 12-d exposure, mortalities had not reached the 50% level. As water  $\Delta P$  was increased to 152 mm Hg ( $2.03 \times 10^4$  Pa), the 10% mortality level decreased to 41 h and the 50% mortality level was achieved in 9.7 d. At a water  $\Delta P$  of 198 mm Hg ( $2.64 \times 10^4$  Pa), the 10% mortality level decreased to 19 h while the 50% level decreased to 20 h.

Colt et al. (1985) studied the response of channel catfish (*Ictalurus punctatus*) to DGS and found that juvenile fish (4.80–5.03 g) were fairly resistant to  $\Delta P$  levels up to 76 mm Hg ( $1.01 \times 10^4$  Pa). At this level, a 1% mortality occurred in a 35-d exposure period. As  $\Delta P$  was increased to 117 mm Hg ( $1.56 \times 10^4$  Pa), the level of mortality rose to 56% in 35 d.

Gray et al. (1982) also found that black bullhead (*Ictalurus melas*) (average 27.7 g) survived exposures to a  $\Delta P$  of 55 mm Hg ( $7.33 \times 10^3$  Pa) for 96 h, but experienced a 50% mortality after exposure at a  $\Delta P$  of 435 mm Hg ( $5.80 \times 10^4$  Pa) for 5.6 h. The 96-h  $LC_{50}$  for the black bullhead was estimated to be 109 mm Hg ( $1.45 \times 10^4$  Pa). Based on the probit data provided, it appears that the upper limit for  $\Delta P$ , which would ensure 100% survival of this species, is  $\approx 76$ –90 mm Hg ( $1.01$ – $1.20 \times 10^4$  Pa).

Fidler (1984, 1988) and Shrimpton et al. (1990a, 1990b) examined the effects of DGS on physiological parameters such as swim bladder pressures, intracorporeal and

extracorporeal bubble formation, blood pH, blood pO<sub>2</sub>, and blood catecholamines in relation to water ΔP and pO<sub>2</sub>, water depth, and fish size. From the results of these studies, threshold prediction models were derived for specific signs of GBT in fish.

$$\Delta P_{SB} = 73.89 \times wd + 0.15 \times pO_2 \quad (4)$$

$$\Delta P_{EW} = 73.89 \times wd + 83.0 \quad (5)$$

$$\Delta P_{CV} = 73.89 \times wd + 0.21 \times pO_2 + 83.0 \quad (6)$$

where

ΔP<sub>SB</sub> = water ΔP required to initiate overinflation of the swim bladder in rainbow trout (mm Hg)

ΔP<sub>EW</sub> = water ΔP required to initiate subdermal emphysema and extracorporeal bubble growth between gill lamella in rainbow trout (mm Hg)

ΔP<sub>CV</sub> = water ΔP required to initiate bubble growth in the cardiovascular systems of rainbow trout (mm Hg)

wd = water depth at which the fish is located (m)

pO<sub>2</sub> = partial pressure of dissolved oxygen (mm Hg)

Nebeker et al. (1976) conducted experiments on several invertebrate species including a cladoceran (*Daphnia magna*), western crayfish (*Pacifastacus leniusculus*), and three stoneflies (*Acronuria californica*, *A. pacifica*, and *Pteronarcys californica*). Examination of the test animals after exposure to the various levels of DGS showed clear evidence of bubbles in the gut and extrapolated lethal thresholds of TGP% = 111% (ΔP ≈ 84 mm Hg; 1.12 × 10<sup>4</sup> Pa). Results indicate that freshwater invertebrates are either less sensitive or as sensitive to some of the signs of GBT as freshwater fish.

Colt et al. (1984a) exposed bullfrog tadpoles (*Rana catesbeiana*) to ΔP levels of 160–170 mm Hg (2.13–2.27 × 10<sup>4</sup> Pa) for 4 d with no apparent effect. When exposure was increased to 10 d, mortalities increased along with a systemic bacteria infection. The intestinal tract and gallbladder were also filled with gas bubbles. Colt et al. (1987) exposed adult bullfrogs (*R. catesbeiana*) to several levels of DGS. At the highest level (ΔP = 240 mm Hg; 2.67 × 10<sup>4</sup> Pa), a 40% mortality occurred in a 24-h period. At all levels of ΔP above 128 mm Hg (1.71 × 10<sup>4</sup> Pa), animals had extensive blistering of external skin surfaces and bubbles in the vascular system. Colt et al. (1984b) also exposed adult

African clawed toads (*Xenopus laevis*) to DGS. The authors reported that extensive bubble formation occurred in interdigital webbing and subcutaneously on body surfaces. Death resulted from bubble formation in the vascular system and secondary bacterial infections.

No data were found in the literature describing the effects of DGS on aquatic plankton, algae, or vascular plants, however, based on the signs of GBT in fish and other aquatic organisms and an understanding of the processes involved in bubble formation and growth, some effects of DGS on aquatic plants can be anticipated. For example, if bubbles form in the surrounding water and become attached to plankton and algae, these plants may float to the water surface. Since bubble formation in “clean” water appears to occur at a ΔP of ≈76 mm Hg (1.01 × 10<sup>4</sup> Pa), this is a threshold that may apply to aquatic plants.

### **Toxicological Studies with Marine Species**

Cornacchia and Colt (1984) reported that 10- to 31-d-old striped bass larvae (*Morone saxatilis*) developed overinflated swim bladders and bubbles in the intestinal lumen at TGP% levels of 102.9 and 105.6%. At a TGP% of 105.6%, there was a 33% mortality in a 78-h period. In other experiments, these authors found that 19-d-old fish succumbed to a TGP% of 106.3% with a 35% mortality in a 72-h period. However, in 29-d-old fish, TGP% levels of 106.3% did not produce any mortalities over a 72-h period.

Gray et al. (1985) examined the tolerance of sea bass (*Dicentrarchus labrax*) and striped mullet (*Mugil cephalus*) to the effects of DGS at temperatures of 20°C and 26°C. They reported that at 20°C, postlarval sea bass (30 mm in length) survived 96-h exposures to a ΔP of 152 mm Hg (2.03 × 10<sup>4</sup> Pa), but experienced 50% mortalities after 96 h (LC<sub>50</sub>) at a ΔP of 207 mm Hg (2.76 × 10<sup>4</sup> Pa). At 26°C, the 96-h LC<sub>50</sub> was a ΔP of 166 mm Hg (2.21 × 10<sup>4</sup> Pa). Fingerling sea bass (100 mm in length) survived a 96-h exposure at a temperature of 20°C and a ΔP of 114 mm Hg (1.52 × 10<sup>4</sup> Pa), but suffered a 50% loss after 96 h at a ΔP of 122 mm Hg (1.63 × 10<sup>4</sup> Pa). Based on these observations, they concluded that the upper ΔP limit for 100% survival at 20°C is 152 mm Hg (2.03 × 10<sup>4</sup> Pa) for postlarval sea bass and 114 mm Hg (1.52 × 10<sup>4</sup> Pa) for fingerling sea bass. They also reported that the fish were overbuoyant due to a large bubble inside the body cavity.

In experiments with striped mullet (*Mugil cephalus*), Gray et al. (1985) found that at 20°C, postlarval fish (31 mm in length) survived 96-h exposures at a  $\Delta P$  of 144.5 mm Hg ( $1.86 \times 10^4$  Pa), but experienced a 50% mortality after 96 h at a  $\Delta P$  of 223 mm Hg ( $2.97 \times 10^4$  Pa). Fingerling striped mullet (130 mm in length) survived 96-h exposures at a  $\Delta P$  of 114 mm Hg ( $1.52 \times 10^4$  Pa), but suffered a 50% mortality after 96 h at a  $\Delta P$  of 188.5 mm Hg ( $2.51 \times 10^4$  Pa). Based on these observations, they concluded that the upper  $\Delta P$  limit for 100% survival is 144.5 mm Hg ( $1.93 \times 10^4$  Pa) for postlarval mullet and 114 mm Hg ( $1.52 \times 10^4$  Pa) for fingerling mullet. It should be noted that for fingerling sea bass the upper  $\Delta P$  limit for 100% survival is the same as for the striped mullet. For postlarval stages of these species, the upper  $\Delta P$  limit for 100% survival of the sea bass is slightly higher than that for the striped mullet.

Malouf et al. (1972) reported the occurrence of GBT in three species of bivalve molluscs (*Crassostrea virginica*, *C. gigas*, and *Mercenaria mercenaria*). Massive blisters were found on the valves of oysters, and bubbles were observed in gill filaments. DGS was produced by heating water in a closed container, however, dissolved gas levels were not reported. Lightner et al. (1974) reported on GBT in the juvenile brown shrimp (*Penaeus aztecus*). Stage II protozoal, larval shrimp developed GBT after being exposed to DGS. Most animals had bubbles under the carapace and in the gut, and there was 100% mortality. No actual dissolved gas levels were reported, however, the water was held at a  $\Delta P$  of 258.5 mm Hg ( $3.45 \times 10^4$  Pa) with subsequent decompression.

Bisker and Castagna (1985) report that at TGP% levels above 108%, *Mulinia lateralis* (Say) floated to the surface and gas bubbles were visible in tissues. *Mya arenaria* (Linne) showed similar, though less severe, behaviour, while *Mercenaria mercenaria* (Linne) did not appear to be adversely affected. Some mortalities were observed in the study at a TGP of 120%, but these were not significantly different from those of controls.

### Other Jurisdictions

British Columbia recently approved provincial  $\Delta P$  criteria of no more than 24 mm Hg ( $3.20 \times 10^3$  Pa at pAtm of  $1.01 \times 10^5$  Pa) for fish in shallow water <1 m (see Table 1; TGP% of  $\approx 103\%$  of sea level atmospheric pressure), and 76 mm Hg ( $1.01 \times 10^4$  Pa or TGP% of  $\approx 110\%$ ) at local water depths are >1 m (Fidler and Miller

1997). Furthermore, the province has developed a site-specific TGP water quality objective of 110% of sea level atmospheric pressure for the Columbia River system from the Hugh Keenleyside Dam to Birchbank (Butcher 1992). In the United States, the USEPA has published DGS guidelines that recommend a maximum TGP of 110% of local atmospheric pressure (USEPA 1986).

### Freshwater, Estuarine, and Marine Guidelines

The following guidelines are protective of aquatic organisms (fish, invertebrates, and plants).

- A. Where local water depth at a given location in a water body exceeds 1 m, the maximum  $\Delta P$  should not exceed 76 mm Hg ( $1.01 \times 10^4$  Pa at pAtm of  $1.01 \times 10^5$  Pa) regardless of water  $pO_2$  levels. For sea level conditions, this corresponds to a TGP% of  $\approx 110\%$ .
- B. Where local water depth at a given location in a water body is <1 m, the guideline should be based on equation (4), which describes the threshold for swim bladder overinflation as a function of water depth and  $pO_2$  levels. For surface water sea level conditions, this corresponds to a TGP%  $\approx 103\%$ .

In cases where it is necessary to determine whether to use guideline A or B, guideline B should be used in absence of information to the contrary. The most conservative application of the guideline will be to use equation (4) with  $h=0$ . At a water depth of 0 m and a  $pO_2$  of 157 mm Hg ( $2.09 \times 10^4$  Pa), the  $\Delta P$  must not exceed 24 mm Hg ( $3.20 \times 10^3$  Pa at pAtm of  $1.01 \times 10^5$  Pa), which corresponds to a TGP% of  $\approx 103\%$  at sea level. This would apply to shallow water bodies and for stream margins, where the area <1 m deep is used by juvenile fish.

Information on the lack of residence of juvenile fish at river margins could be a basis to apply the less conservative guideline A, provided the presence of other stressors do not suggest a more conservative application.

Table 1 shows the results of combining the two guideline criteria. For water depths >1 m,  $\Delta P$  levels >76 mm Hg ( $1.01 \times 10^4$  Pa at  $1.01 \times 10^5$  pAtm) are unsafe for fish regardless of water  $pO_2$  or depth. For water depths <1 m, the maximum allowable  $\Delta P$  depends on water  $pO_2$ .

**Table 1. Maximum allowable  $\Delta P$  as a function of water depth and  $pO_2$ .**

Water depth (wd) -(m)	Maximum allowable $\Delta P$ at water $pO_2$ -mm Hg					
	50	100	157	200	250	300
0	8	15	24	30	38	45
0.1	15	22	31	37	45	52
0.2	22	30	38	45	52	60
0.3	30	37	46	52	60	67
0.4	37	45	53	60	67	76
0.5	44	52	60	67	76	76
0.6	52	59	68	76	76	76
0.7	59	67	76	76	76	76
0.8	67	76	76	76	76	76
0.9	76	76	76	76	76	76
$\geq 1.0$	76	76	76	76	76	76

- C. For natural background levels higher than the recommended guideline, there should be no anthropogenic increase in the  $\Delta P$  or TGP% over the background levels.
- D. For hatchery environments, the DGS is defined by equation (4) with  $wd = 0$ . For surface water sea level conditions, this corresponds to a TGP% of  $\approx 103\%$ . This guideline allows for consideration of DGS in systems using oxygen supplementation. For example, using equation (4) (see Table 1,  $wd = 0$  m), if  $pO_2$  is 250 mm Hg ( $3.33 \times 10^4$  Pa or 164% of saturation), then the maximum allowable DGS is 38 mm Hg ( $5.07 \times 10^3$  Pa; TGP% is 105%).

Bubble growth under incubation screens can occur even at low levels of DGS, and the resulting water blockage can have severe effects on eggs and alevins. Entrained gas bubbles coalesce to form large bubbles that grow quickly even if the water supply is slightly supersaturated. Therefore, a 24 mm Hg ( $3.20 \times 10^3$  Pa) guideline should be viewed as an upper limit for incubation.

### Water Quality Guideline Derivation

In considering water quality guidelines for DGS, it was recognized that the effects of DGS on freshwater and marine organisms are quite different from the action of most toxic chemicals. Water depth plays an important role in protecting fish from the effects of DGS (Fidler and Miller 1997). In addition, many rivers and lakes have

naturally occurring levels of DGS that are potentially lethal to fish. Yet, wild fish appear to have developed strategies for surviving in these environments (White et al. 1991). Finally, the signs of GBT in fish are strongly dependent on the size of the animal and water temperature (Fidler and Miller 1997), and no standard dose-response curve exists for exposure to DGS. As a result of the many mitigating and compounding effects of environmental and biological variables, the procedures for developing water quality guidelines for DGS were more complex than those traditionally used in the development of guidelines for toxic chemicals.

The data describing the effects of DGS on marine fish suggest that they are affected by DGS in the same way as freshwater fish and that the  $\Delta P$  thresholds for the signs of GBT are very similar to those for freshwater fish. Thus, the rationale used for the derivation of DGS guidelines for freshwater fish also applies to estuarine and marine fish.

Fish as a result of swim bladder overinflation, exhibit a higher degree of sensitivity to the effects of DGS than do aquatic invertebrates, aquatic plants, and algae. That is, GBT signs in fish appear at lower  $\Delta P$  levels than have been reported for aquatic invertebrates, and the occurrence of GBT in plants and algae has not been reported. Thus, water quality guidelines derived for fish should also be protective of these other aquatic biota.

Given the wide range of environmental and biological variables that can influence the impact of DGS on fish populations, a single value numerical guideline is impractical. Such a guideline may be too restrictive in some situations and not restrictive enough in others. The first requirement of a guideline is to protect young fish from the chronic effects of swim bladder overinflation at low levels of DGS. As such, water depth and  $pO_2$  levels, as defined by equation (4), and the experimental results of Shrimpton et al. (1990a, 1990b) are central criteria for the derivation of DGS water quality guidelines.

In addition to protecting small fish, it is necessary to protect fish of all sizes from the acute signs of GBT involving subdermal emphysema, the blockage of the gill respiratory water flow by extracorporeal bubbles, and the development of bubbles in the cardiovascular system. The lowest  $\Delta P$  for acute mortality is 76 mm Hg ( $1.01 \times 10^4$  Pa at  $pAtm$  of  $1.01 \times 10^5$  Pa). Thus, 76 mm Hg ( $1.01 \times 10^4$  Pa at  $pAtm$  of  $1.01 \times 10^5$  Pa) should be the second criterion for the derivation of a DGS water quality guideline.

The combination of equation (4), the experimental results of Shrimpton et al. (1990a, 1990b), and the  $\Delta P$  threshold of 76 mm Hg ( $1.01 \times 10^4$  Pa at pAtm of  $1.01 \times 10^5$  Pa) form the basis for the guideline. Because 76 mm Hg ( $1.01 \times 10^4$  Pa at pAtm of  $1.01 \times 10^5$  Pa) is very close to the hydrostatic compensation pressure corresponding to 1 m of water depth (i.e., 73.89 mm Hg;  $9.85 \times 10^3$  Pa at pAtm of  $1.01 \times 10^5$  Pa), 1 m of water depth serves as a convenient division (or crossover point) between equation (4) and the  $\Delta P$  threshold of 76 mm Hg ( $1.01 \times 10^4$  Pa at pAtm of  $1.01 \times 10^5$  Pa).

In recognition that natural background levels that are higher than the recommended guidelines may be harmful to fish, any anthropogenic increase over background levels should not be tolerated for the protection of aquatic life.

For hatchery environments, it is recognized that shallow water depth, crowding, and added exposure to diseases increase stress beyond that encountered in natural environments. Therefore, the guideline for hatchery environments is based on equation (4). This guideline also allows for higher DGS in systems using oxygen supplementation.

Additional site-specific concerns, such as environmental variables and organism behavioural patterns, can be considered in the implementation of the guideline or in the development of site-specific objectives for a specific water body.

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