

TOTAL DISSOLVED GAS SUPERSATURATION SUMMARY OF BIOLOGICAL EFFECTS FROM LITERATURE 1980-2007

DRAFT

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ACRONYMS

atms	atmospheres of pressure (1 atm = 14.7 lb/in ² , 760 mm Hg or torr)
ft ³ /s	cubic feet per second
cfs	cubic feet per second
ESA	Endangered Species Act
FERC	Federal Energy Regulatory Commission
ft	feet
GBD	gas bubble disease
h	hour
kcfs	one thousand cubic feet per second
LC ₅₀	lethal concentration, 50% of population
LT ₂₀	lethal time, to mortality 20% of population
m	meter
mm Hg	millimeters of mercury (pressure)
m/s	meters per second
Pa	Pascal (unit of pressure, 1 atms = 101.3 kPa)
kPa	kilopascal, 1,000 Pascals
%	percent
RM	river mile
TDG	total dissolved gas
TDGP	total dissolved gas pressure
torr	metric unit of pressure = 1 mm Hg

INTRODUCTION

Total dissolved gas (TDG) supersaturation has been regulated as a water quality parameter since the early 1970s because of its potential to harm fish and other aquatic organisms. However, the criterion was initially established on the basis of the results of laboratory studies that did not incorporate natural depth factor that is important in determining the biological effects of TDG supersaturation. This summary reviews relevant literature produced during the last 27 years and summarizes the information available to substantiate retention or revision of the current TDG criterion of 110% of saturation. The review covers that literature produced since the previous review by Weitkamp and Katz (1980). Abstracts of the literature supporting this summary are provided by Weitkamp (2008a). The information is analysed and discussed in Weitkamp (2008b).

Most of us find understanding the biological effects of TDG supersaturation challenging because of the role that water pressure (hydrostatic pressure) plays in both producing TDG supersaturation and in limiting its effects. It is water pressure that causes air bubbles to pass into solution resulting in supersaturation. These same water pressures at various depths provides hydrostatic compensation that causes the fish to be exposed to much lower actual TDG levels than those that are reported relative to surface pressure. TDG levels are almost always reported with reference to surface pressure rather than with reference to the depth at which the measurements are made or at which the fish reside.

A substantial body of literature now exists that provides information on exposure of fish and invertebrates to TDG supersaturation in field conditions (rivers, reservoirs, lakes) that commonly provide opportunity for hydrostatic compensation. The information available from field investigations documents that gas bubble disease (GBD) does occur at some level in some species when TDG levels exceed 120%. However, GBD is generally of low incidence and severity in most field situations if TDG is not much greater than 120% of saturation.

Much of the information on the biological effects of TDG supersaturation comes from research and monitoring conducted in the Columbia River Basin where numerous dams frequently produce TDG levels exceeding 120% during spring high flows. In the Columbia River Basin forced spill also occurs to enhance anadromous fish passage. Variances in water quality criteria (115-120% TDG) have commonly been passed by several states to allow substantial spill for this fish passage.

The TDG levels in rivers and reservoirs commonly exceed the existing water quality criterion of 110% of saturation only during periods when high river flow produced by spring runoff result in substantial spill at dams or natural falls. Anthropogenic TDG supersaturation exceeding the criterion also occasionally results from heated effluents, but recorded cases are rare. During exceptionally high spring runoff periods the hydraulic capacity of many dams is exceeded, resulting in spill of large portions of the total river flow and production of TDG supersaturation. Many of the existing regulations recognize the limited capacity to control TDG during these extremely high flows by suspending the criterion when river flows exceed the 7Q10 flow (stream flow exceeding the seven-day, ten-year frequency flood). Elevated levels of TDG (>110-130%) also occur naturally, such as recorded downstream from Niagara Falls (avg. TDG 126%).

BIOLOGICAL AFFECTS OF TDG SUPERSATURATION

It has become clear that GBD does commonly occur during high TDG levels (>120%), at least at a low incidence and severity. The literature also demonstrates that GBD is reversible and

commonly a non-fatal disease. The observations recorded in the literature indicate that the degree and nature of biological effects of TDG supersaturation that occur under field conditions are limited by the combination of:

1. moderate levels of TDG supersaturation (100-125%) that commonly occur,
2. the locations where TDG supersaturation occurs (rivers and reservoirs with substantial depths),
3. the behavior of fish (daily occupying a range of depths),
4. the capacity of fish to recover rapidly from GBD when they reach compensating depths, and
5. water depths that normally increase as flows and TDG levels increase.

The literature indicates that in most cases where GBD signs have been observed in fish collected from rivers and reservoirs both the incidence and severity of the signs have been low. Levels of TDG in the range of 110-120% have at times resulted in rates of GBD in the general range of 1-10% in those fish species that commonly inhabit shallow water. The severity of the GBD signs observed in these fish is generally minor, that is a level or rank 1, with a few bubbles (blisters) in the fins of the fish. When TDG levels have substantially exceeded 120% for prolonged periods of weeks, some higher rates and severities of GBD have been reported in those fish inhabiting shallow water. However, even these higher TDG levels rarely have resulted in observed mortalities. TDG levels of 130% and higher substantially elevate the incidence and severity of GBD signs observed in fish collected from shallow water and juvenile salmon migrants collected at dams.

There appears to be an inherent conservative factor in observations of GBD. Records of GBD likely tend to over estimate the incidence of the disease in populations as a whole because of the collection techniques. Collection of fish by electrofishing or from bypass systems at dams results in sampling of that portion of the population most likely to have GBD as the sampled fish are taken from the shallowest of available depths or held in shallow water for some period prior to examination.

Investigations that restrain fish and invertebrates in shallow water (<1 m) greatly exaggerate the incidence and severity of GBD as compared to field conditions. Although these shallow water investigations are essential for investigating GBD, they do not realistically represent the biological effects that occur in field conditions, principally due to the much greater depths available in field conditions.

MORTALITIES – POPULATION EFFECTS

Instances of fish mortality due to GBD under field conditions have been reported in a several reports. Since the first recorded major mortality of fish in the Columbia River Basin that occurred downstream from John Day Dam in 1968, there have been only a few mortality instances reported. Beeman et al. (2003a) mention that fish kills have occurred, at least in net pens, in Rufus Woods Lake downstream from Grand Coulee Dam on the Columbia River. These mortalities appear to have been caused by TDG supersaturation, but no specific information is provided. However, growth patterns of resident rainbow trout in Rufus Woods Lake over a period that includes several years of high TDG supersaturation (1996, 1997) were reported by Maule et al. (2003) and Beeman et al. (2003b) to not show differences sufficiently large to suggest annual influences of TDG supersaturation.

Colt et al. (1991) reported that a significant mortality of salmonids occurred in the American

River, California, during a major flood in 1986 with TDG levels of about 126-132% resulting from spill at Folsom Dam. In the Missouri River downstream from Harry S. Truman Dam large numbers of fish were killed by GBD when TDG levels we up to 139% (Crunkilton et al. 1980). Lutz (1995) reported 15 fish kills (<50 to several thousand fish) occurred downstream from Red Rock Dam, Iowa between 1983 and 1994 that she attributed to GBD. During these mortalities TDG levels averaged 121% with peak levels as high as 140% where maximum river depths were as shallow as 0.6 m. Mortalities occurred only when Des Moines River flows were relatively low during supersaturation resulting in shallow water (avg. 1.1 m) that did not allow hydrostatic compensation.

Although substantial mortalities have been reported in several situations, these mortalities have not resulted in obvious population effects. Maule et al. (2003) were unable to detect sublethal population effects in resident fish of Lake Rufus Woods following multiple years of supersaturation.

GBD CHARACTERISTICS

The basic signs of GBD described in recent literature have not changed from those described in early literature, but more information is now available on the relation of GBD signs to death and recovery. The severity of externally visible signs appears to be related to severity of the disease and mortality at low and moderate TDG levels up to approximately 130%. At high TDG levels mortality appears to be unrelated to GBD signs. Mortality appears to be caused primarily by bubbles in the vascular system. Invertebrates with bubbles in their digestive tract may experience buoyancy that makes them more susceptible to predation.

Bubbles produced by TDG supersaturation disappear rather rapidly with either increased hydrostatic pressure or transfer to water that has little or no TDG supersaturation. Bubbles in the gills are lost within minutes while bubbles in the fins may take hours to days to disappear depending on pressure and TDG conditions. Under chronic GBD conditions it appears that scar tissue may be produced by bubbles in fins, but debilitating effects have not been reported.

SPECIES, LIFE STAGE VARIABILITY

A wide variety of fish and invertebrates and life stages are now covered by the available literature. Differences in fish species susceptibility to GBD have been identified. It appears that in some cases these differences are due to differences in behavior (depth distribution) that actually produce differences in exposure. This is supported by field observations that show higher incidences and greater severity of GBD in those species that tend to remain in shallow water. In other cases the differences in GBD susceptibility may be due to anatomical differences (physoclistous v physostomus).

Differences in GBD susceptibility have been identified for different life stages. Embryos are clearly more resistant than young fish (larvae and juveniles) (Jensen 1980, 1988), including embryos of females previously exposed to TDG supersaturation (Gale et al. 2004). This resistance is explained by the hydrostatic pressure within the egg capsule that is higher than atmospheric pressure. Young fish are commonly less resistant than adult fish, apparently resulting from different depth distributions.

Some work has shown that GBD may make young fish more susceptible to predation (Mesa and Warren 1997). However, other work indicates predators tend to have decreased activity and settle to or swim near the bottom as they develop GBD, making them less able to prey on smaller fish

(Bentley and Dawley 1981, VanderKooi et al. 2003).

The capacity of fish to detect and avoid TDG supersaturation has not been resolved. Most investigations indicate fish have little or no capacity to detect TDG supersaturation.

TDG CONTROL

Considerable progress has been made in developing operational and structural modifications to hydroelectric facilities to reduce the TDG levels they produce. Spillway deflectors (flip lips) have been installed on many spillways to prevent water with entrained air from plunging to the bottom of stilling basins. Operational and structural modifications have reduced TDG in spillway discharges, but commonly have not been adequate to eliminate exceedances of the 110% TDG criterion.

Monitoring is now routinely conducted at many hydroelectric facilities using commercially available monitoring equipment based on the semi-permeable membrane technology. Most of these records are readily available, but not generally published.

A number of computational models have been developed to help understand the entrainment of air and subsequent supersaturation of water downstream from spillways. Most of these models are empirically based and require adaptation to specific dams.

NATURAL TDG SUPERSATURATION

In addition to human-caused TDG supersaturation there are situations where TDG supersaturation naturally occurs at levels exceeding the TDG criterion of 110%. Recently monitoring demonstrated that Niagara Falls produces average TDG values of about 126%. It is likely that major falls such as Kettle Falls and Celilo Falls on the Columbia River historically produced high TDG levels, particularly during high spring flows.

EXISTING TDG CRITERION

The existing water quality criterion for TDG (110% of saturation) was promulgated in the early 1970s based on the presumed biological effects of GBD in fish and invertebrates resulting from laboratory investigations. Motivation for regulation of TDG came with passage of the Clean Water Act of 1965 stimulating promulgation of state water quality criteria. At that time there was very little information available on the biological effects of TDG supersaturation under field conditions. Thus, the scientific information available to support a criterion was derived primarily from laboratory investigations that restricted fish and invertebrates to shallow water (30 cm to 1 m total depth). These shallow water data were used to justify the initial criterion of 110% (105% in Oregon). The promulgation approach was one of caution to ensure that there would be little potential for injury to fish in the natural environment if the criterion were met. This approach did not necessarily assume that meeting the criterion was essential to protect fish, but that available information was not adequate to set a higher level with reasonable assurance that adequate protection would be provided.

In the early 1970's the states of Washington and Oregon promulgated dissolved nitrogen (N_2) criteria before we commonly recognized that TDG rather than just N_2 was a more appropriate parameter. Washington set their TDG criterion at 110% of saturation, while Oregon initially set its criterion at 105%, and subsequently raised it to 110%. Based on these initial criteria, the U.S. EPA initially proposed a criterion of 110% of TDG saturation in the 1972 Blue Book. The EPA

1976 Red Book revised the justification, but retained the same 110% of saturation criterion.

In 1977, the American Fisheries Society reviewed the EPA Redbook criterion. Wes Ebel, Gerry Bouck, Kirk Beiningen, W. R. Penrose, and Don Weitkamp reviewed the criterion using information available at that time. Although there was not unanimous agreement that 110% of saturation was necessary, the group agreed that this criterion would be protective. Their review was still constrained by the dearth of field information with nearly all available information derived from laboratory investigations, and some provided by live cage studies.

CONCLUSIONS

It appears that in most field situations that the TDG levels of 110-120% produce little if any GBD, and that the severity of GBD under these conditions is likely to be minor if it does occur. The hydrostatic compensation available to fish and invertebrates in field conditions where TDG supersaturation generally occurs, together with the depths used through the fish's natural behavior, generally avoids the affects seen in laboratory investigations. The literature reviewed in this document, and a previous literature review (Weitkamp and Katz 1980), do not support population effects resulting from TDG levels of 120% and lower. Aquatic population effects have not been observed in most cases where they have been investigated with TDG levels much greater than 120%.

The field data indicate that the 110% TDG criterion is very conservative. A criterion of 120% would adequately protect aquatic biota in most field situations where TDG supersaturation occurs. We suggest fish and invertebrates in field conditions with available depths of 2 m or greater would be adequately protected by a TDG criterion of 120%.

Hatchery water supplies and very shallow streams that do not offer an opportunity for hydrostatic compensation may require TDG levels of 110% to adequately protect those fish and invertebrates that live in these shallow conditions.

LITERATURE CITED

- Beeman, J. W., D. A. Venditti, R. G. Morris, B. J. Adams, and A. G. Maule. 2003a. Chapter I: Depths and hydrostatic compensation of farmed fish and wild fish in Rufus Woods Lake. Pages 2-47 in, Beeman, J. W., D. A. Venditti, R. G. Morris, D. M. Gadomski, B. J. Adams, S. P. VanderKooi, T. C. Robinson, and A. G. Maule. 2003. Gas bubble disease in resident fish below Grand Coulee Dam final report of research. U.S. Geological Survey, Western Fisheries Research Laboratory, Cook, Washington.
- Beeman, J. W., D. A. Venditti, R. G. Morris, D. M. Gadomski, B. J. Adams, S. P. VanderKooi, T. C. Robinson, and A. G. Maule. 2003b. Gas bubble disease in resident fish below Grand Coulee Dam final report of research. U.S. Geological Survey, Western Fisheries Research Laboratory, Cook, Washington. 157 p.
- Bentley, W. W., and E. M. Dawley. 1981. Effects of supersaturated dissolved atmospheric gases on northern squawfish, *Ptychocheilus oregonensis*. Northwest Science 55:50-61.
- Colt, J. E., K. Orwicz, and D. Brooks. 1991. Gas supersaturation in the American River California USA. California Fish and Game 77(1):41-50.

- Crunkilton, R. L., J. M. Czarnecki, and L. Trial. 1980. Severe gas bubble disease in a warmwater fishery in the Midwestern United States. *Transactions of the American Fisheries Society* 109:725-733.
- Gale, W. L., A. G. Maule, A. Postera, and M. H. Peters. 2004. Acute exposure to gas-supersaturated water does not affect reproductive success of female adult Chinook salmon late in maturation. *River Research and Applications* 20(5):565-576.
- Jensen, J. O. T. 1980. Effects of total gas pressure, temperature and total water hardness on steelhead eggs, and alevins. A progress report. Pages 15-22 in *Proceedings 31st Northwest Fish Culture Conference*, Courtenay, British Columbia.
- Jensen, J. O. T. 1988. Combined effects of gas supersaturation and dissolved oxygen levels on steelhead (*Salmo gairdneri*) eggs, larvae, and fry. *Aquaculture* 68(2): 131-139.
- Lutz, D. S. 1995. Gas supersaturation and gas bubble trauma in fish downstream from a Midwestern reservoir. *Transactions of the American Fisheries Society* 124:423-436.
- Maule, A. G., B. J. Adams, R. G. Morris, J. W. Beeman, and D. A. Venditti. 2003. Chapter IV: Growth of resident fishes does not correlate with years of high gas supersaturated water. Pages 109-133 in, Beeman, J. W., D. A. Venditti, R. G. Morris, D. M. Gadomski, B. J. Adams, S. P. VanderKooi, T. C. Robinson, and A. G. Maule. Gas bubble disease in resident fish below Grand Coulee Dam final report of research. U.S. Geological Survey, Western Fisheries Research Laboratory, Cook, Washington.
<http://wfrc.usgs.gov/pubs/reportpdf/usgsfrgbdgrandcouleedam.pdf#page=54>
- Mesa, M. G., J. J. Warren, K. M. Hans and A. G. Maule. 1997. Progression and severity of gas bubble trauma in juvenile Chinook salmon and development of non-lethal methods for trauma assessment. Pages 55-90 in Maule, A. G., J. Beeman, K. M. Hans, M. G. Mesa, P. Haner, and J. J. Warren. 1997. Gas bubble disease monitoring and research of juvenile salmonids. Annual Report 1996 (Project 96-021), Bonneville Power Administration, Portland, Oregon.
- VanderKooi, S. P., R. G. Morris, J. W., Beeman, and A. G. Maule. 2003. Chapter II: The progression and lethality of gas bubble disease in resident fish in Rufus Woods Lake. Pages 48-86 in, Beeman, J. W., D. A. Venditti, R. G. Morris, D. M. Gadomski, B. J. Adams, S. P. VanderKooi, T. C. Robinson, and A. G. Maule. Gas bubble disease in resident fish below Grand Coulee Dam final report of research. U.S. Geological Survey, Western Fisheries Research Laboratory, Cook, Washington.
<http://wfrc.usgs.gov/pubs/reportpdf/usgsfrgbdgrandcouleedam.pdf#page=54>
- Weitkamp, D. E. 2008a. Total dissolved gas literature 1980-2007, an annotated bibliography. Unpublished Report, Parametrix, Bellevue, Washington.
- Weitkamp, D. E. 2008b. Total dissolved gas supersaturation biological effects, review of literature 1980-2007. Unpublished Report, Parametrix, Bellevue, Washington.
- Weitkamp, D. E., and M. Katz. 1980. A review of dissolved gas supersaturation literature. *Transactions of the American Fisheries Society* 109:659-702.