# TOTAL DISSOLVED GAS SUPERSATURATION BIOLOGICAL EFFECTS,

# REVIEW OF 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 |

# ABSTRACT

This paper supplements a review of total dissolved gas (TDG) supersaturation literature I prepared in 1979 (Weitkamp and Katz 1980). This later review only addresses literature not covered in the original review with a few exceptions. The literature published since 1980 includes a considerable number of field investigations that provide information on the biological effects of TDG supersaturation under conditions that allow substantially greater hydrostatic compensation (depth) than the shallow depth laboratory studies. The recent literature indicates that TDG supersaturation results in little or no gas bubble disease (GBD) at levels up to 120% of saturation when compensating depths (2 m or more) are available. Research has shown that fish have the capacity to rapidly recover from GBD when they reach compensating depths or TDG supersaturation is decreased. Most instances of GBD have reported low incidence and severity, however there have been a few cases of substantial mortalities reported. The reported mortalities and severe cases of GBD are generally attributed to either TDG supersaturation in situations where available depths are shallow (~1 m or less) or the TDG levels are exceptionally high (>130%). Field investigations have not demonstrated population effects resulting from TDG supersaturation.

Generally the biological effects of TDG supersaturation appear to be influenced by the depth distribution of the fish or invertebrates resulting from their natural behavior, and there is limited evidence suggesting active avoidance of high TDG levels. Commonly TDG supersaturation occurs during high flows that increase water depths in rivers resulting in greater depths available to fish and over invertebrates, thereby reducing the potential effects. Generally the difference in sensitivity of various life stages is related to differences in behavior.

TDG supersaturation is a natural phenomenon that occurs at falls. Niagara Falls has been demonstrated to produce TDG levels of 120-130%. It is likely that natural falls in the Columbia River System and other locations naturally exposed fish populations to substantial TDG levels. TDG levels that were prevalent downstream from many dams in the 1960s to 1980s have been reduced by various measures, however spill required for fish passage frequently results in moderate levels of TDG supersaturation (up to 120% and higher).

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# INTRODUCTION

Regulation of total dissolved gas (TDG) supersaturation has been a water quality issue since the late 1960s when TDG or nitrogen supersaturation was identified as a potential threat (gas bubble disease, GBD) to fish and other aquatic resources downstream from dams. In 1980 we published a comprehensive review of TDG (Weitkamp and Katz 1980). Since that publication there has been a great deal of relevant literature published. This review is an attempt to summarize and provide a coherent description of information that has become available since Weitkamp and Katz (1980). In a few instances earlier literature is cited to support specific information.

## EXISTING CRITERION

The existing TDG criterion of 110% was promulgated prior to the development of most of the information currently available. The information available in the late 1960s was derived primarily from a modest number of investigations conducted in laboratory conditions where the fish were held in vessels with a maximum depth of 1 m or less. Because of the role that hydrostatic pressure plays in the biological effects of TDG supersaturation, these laboratory studies tested worst case conditions that are not representative of most real world situations.

## CURRENT CONDITIONS

We are still faced with the major question, is the water quality criterion of 110% of saturation necessary to adequately protect aquatic biota or is it sufficiently protective? This question is raised because of the extreme physical and operational measures necessary to reduce the frequency and degree of exceedances of the TDG criterion at many dams. In some cases such as the Columbia River System, measures to protect fish through increased river flow by spillway discharge result in exceedances of the existing TDG criterion. Operational changes to limit TDG supersaturation are contrary to the objective of increasing flow to simulate natural river conditions. Waivers of the TDG criteria have been routinely granted for the Columbia and Snake Rivers in recent years by Washington and Oregon to allow spill of substantial volumes of water to bypass downstream migrant juvenile salmon during the spring and summer. These waivers have allowed 120% TDG downstream from dams and 115% in dam forebays.

Generally TDG supersaturation regulation is an issue associated with dams that potentially spill water or cooling water effluents that substantially raise the temperature of saturated water. These sources are present on major rivers and estuaries where there is commonly sufficient depth to provide compensation for the TDG supersaturation. Naturally produced TDG supersaturation can also occur in smaller and shallower streams downstream from falls and some rapids where there is little depth available for compensation.

The water quality criterion for TDG (110% of saturation) is based on the presumed biological effects, gas bubble disease (GBD) in fish. However, most of the investigations that substantial biological effects are laboratory studies, where the fish are held in shallow water, generally less than 1 m deep. In real world situations where fish generally encounter TDG supersaturation water depths greater than 1 m are available. Because the hydrostatic pressure (compensation) of depth mitigates the biological effects of TDG supersaturation, it is common for fish in these real world situations to experience high levels of TDG without incurring GBD or incurring both a low severity and low incidence of GBD.

The complexity of field conditions, together with the difficulty of monitoring biological conditions in the field, has made it challenging to understand the biological effects of TDG supersaturation in the real world.

The relevant information that addresses the potential biological effects of TDG supersaturation includes information that describes both laboratory and field investigations from many different locations. The information from field investigations helps us to interpret the laboratory data. There are several types of information available that identify the effects of TDG supersaturation in the natural waters of rivers, reservoirs and lakes. These types of information include:

- affect of depth (hydrostatic pressure compensation) on TDG exposure experienced by fish,
- observations of fish exposed to TDG supersaturation in natural waters,
- information on the behavior (depth distribution) of species occurring in natural waters, and
- degree, duration and frequency of TDG supersaturation.

Information of this nature provides a basis for understanding the biological risk of TDG supersaturation to fish inhabiting natural waters.

## DISEASE v TRAUMA

In this document the term “gas bubble disease” is used rather than “gas bubble trauma”. The word disease has clear historical precedence over trauma. Thus’ trauma would only be more appropriate than disease if it were more technically appropriate. A review of definitions in a number of medical dictionaries indicates the disease and trauma have broadly overlapping definitions, and that disease is not inappropriate for the malady caused in aquatic organisms by TDG supersaturation.

## GBD SIGNS

Signs of GBD have been well document for many years (Marsh and Gorham 1904, Weitkamp and Katz 1980), however some recent investigations have added to the understanding and recognition of GBD signs. Both recognition of GBD signs and understanding their relationship to long-term effects and mortality are important.

It appears that the relationship of GBD signs to long-term effects and mortality is influenced differently by chronic and acute exposures (Mesa et al. 2000). In general the available literature indicates chronic exposures are generally to modest TDG levels, roughly 5-25% higher than ambient pressure (depth), whereas acute exposures result from TDG levels greatly exceeding ambient pressures (25-40%). With chronic TDG exposures the extent and severity of GBD signs appear to be related to the severity of the disease. Acute exposures appear to result in internal bubble formation that disrupts vascular circulation to a degree that results in organ failure and death, sometimes prior to substantial development of external signs. Chronic v acute exposures can be influenced by the natural behavior of fish with fish intermittently receiving chronic and acute exposures as they descend and ascend in highly supersaturated water. In these cases the incidence and severity of GBD appears to be substantially lower than expected for fish held in shallow depths with acute TDG levels.

Bouck (1980) described GBD as a noninfectious, physically induced process caused by uncompensated hyperbaric pressure of total dissolved gases. He identified three stages of GBD based on laboratory observations as: 1) a latent period of gas equilibration, nonlethal cavitation (bubble formation), and increasing morbidity; 2) rapid and heavy mortality: and 3) protracted survival despite lesions and dysfunction resulting in total mortality. Emboli forming in the blood can cause homeostasis that produces these three stages.

Smith (1988) reported the histologic changes that occur in rainbow trout with GBD. Along with numerous emboli in the gill filaments, he found thrombi in blood vessels, sometimes in the absence of emboli. Fish with affected eyes occasionally showed mild hemorrhaging, displacement of lenses, and sometimes degeneration of the cornea. Exophthalmia was produced by emboli in adipose tissue posterior to the choroid, and sometimes within the choroid or between the choroid and retina. Emphysema occurred in the dermis and between the dermis and epidermis, as well is in connective tissue of fins, gill arches and the mouth roof. Smith (1988) did not find bacterial or fungal infections in this 30 d exposure investigation.

Speare (1990) provides information on the progression of histopathology of exophthalmia. Small gas emboli occurred first in the choroid gland. Bubble size increased markedly in chronic cases. Chronic cases progressed to large retro bulbar bubbles that caused severe exophthalmia (Figure 1), with stretching of the optic nerve and retinal blood vessels producing severe distortion of the posterior aspects of the globe. In acute cases, ocular lesions were limited to anatomical displacement of tissue and local degeneration of compressed tissues around the perimeter of bubbles. Speare (1991) observed vascular lesions, ranging from cellular degeneration to exfoliation, associated with intravascular gas bubbles in juvenile Chinook exposed to 110-124% TDG in laboratory tanks.

Mesa et al. (1997) concluded that laboratory studies suggest that external examinations of GBD signs are adequate to determine the severity of GBD in juvenile salmonids. They report that the first GBD signs to appear are bubbles in the lateral line, which may show a progressive worsening over time and have low inter-individual variation (Maule et al. 1997b). However, bubbles in the lateral line may develop poorly during chronic exposures to TDG supersaturation. Bubbles in the fins are commonly prevalent (Figure 2), show a progressive worsening over time, and may be a relatively persistent sign of GBD. However, they concluded that there is no available quantitative method for evaluating the severity of fin bubbles, and GBD signs may not develop during acute exposures to high TDG. More recently Mesa et al. (2000) concluded that GBD signs generally do not show clear trends of worsening over time, however at 130% TDG, the severity of all GBD signs is highly correlated with mortality (laboratory investigation).

**Figure 1. Severe exophthalmia in a juvenile Chinook salmon.**

Beeman et al. (2003c) and Morris et al. (2003a) reported that differences in diameters of trunk lateral line pores of several fish species have an effect on the progression of GBD signs. They found the percent of lateral line occlusion was inversely related to pore size for longnose sucker >largescale sucker > northern pikeminnow ≥ Chinook salmon ≥ redside shiner.

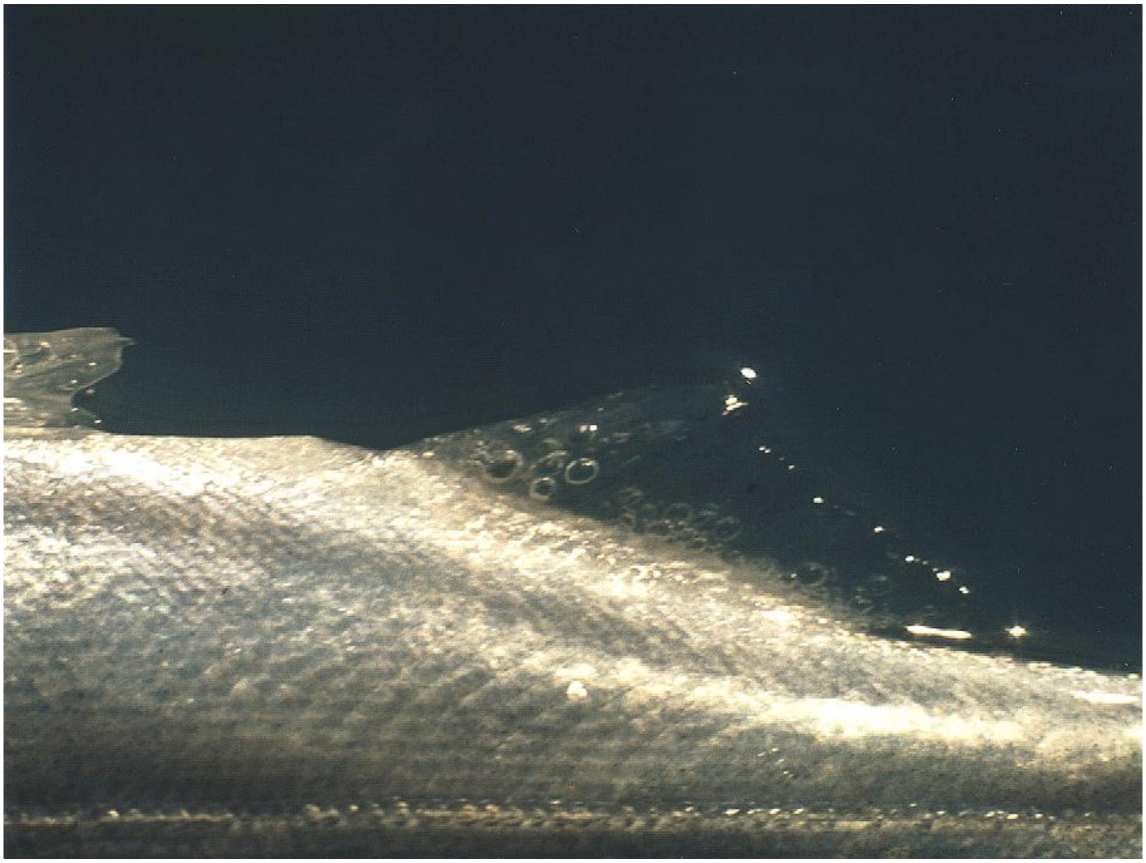

**Figure 2. Severe bubbles in lateral line and ventral fin of juvenile salmon.**

## HYDROSTATIC COMPENSATION (DEPTH)

TDG supersaturation is a unique parameter in the water quality regulation in that it is regulated as a percent of saturation rather than as a concentration. That is: it is a percent of the amount of air that water will hold when it is in equilibrium (100%) with the atmosphere at the total pressure present at the water's surface (ambient atmospheric or barometric pressure). At greater depths the total pressure increases (atmospheric + hydrostatic) causing the amount of atmospheric gases (oxygen, nitrogen, argon, $CO_2$, etc.) the water will hold in an equilibrium state to increase relative to the water's surface pressure. It is this same total pressure that provides this compensation that commonly prevents fish and invertebrates from developing internal bubbles when they are in water that is supersaturated with reference to surface pressure.

TDG supersaturation is nearly always described in reports and regulations as relative to surface (ambient atmospheric) pressure. Figure 3 graphically shows the relationship between reported levels of TDG supersaturation (relative to surface pressure) and the true level of supersaturation experienced by fish at various near-surface depths. The compensation rate is about 10% of saturation or 74 mm Hg per meter of depth.

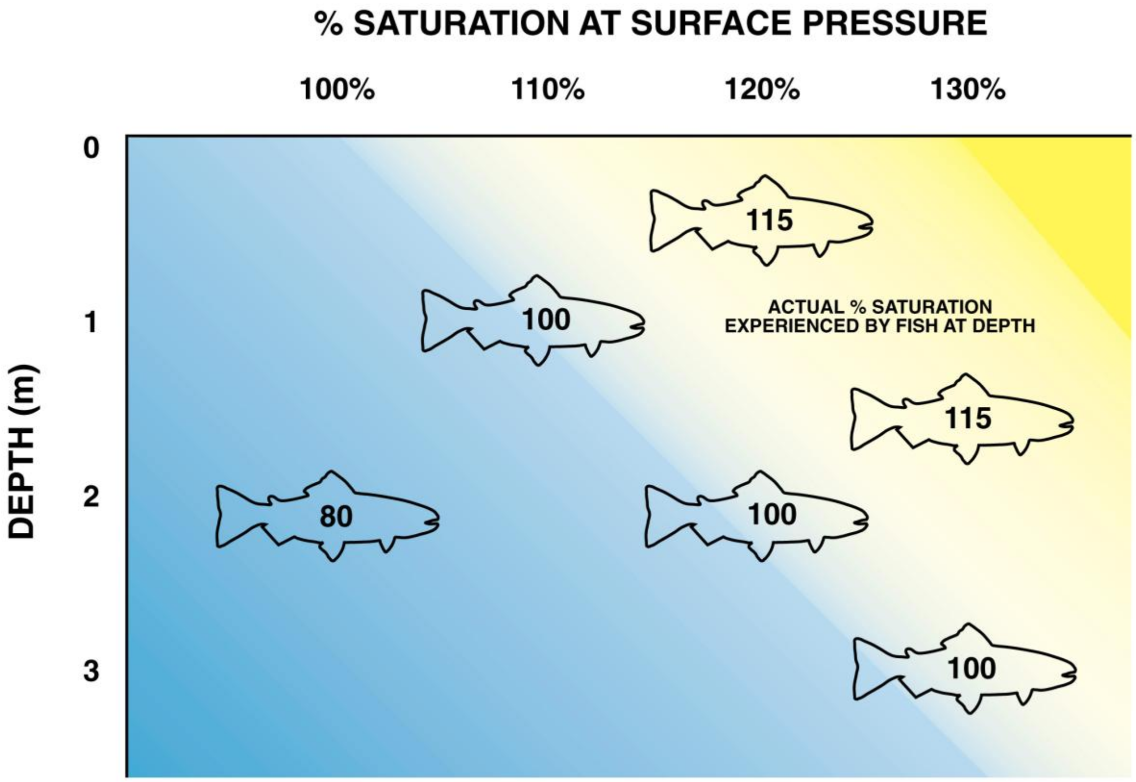

**Figure 3. Relationship of measured and actual total dissolved gas levels experienced by fish at various depths in the river (from Weitkamp et al. 2003a).**

In this review the term "true supersaturation" is used to indicate the actual TDG conditions at the specific depth or depth range of a fish's exposure. This indicates that the TDG level is actually greater than the saturation value for the combined atmospheric and hydrostatic pressure provided by the water column at the exposure depth. These are situations where the dissolved gasses will tend to come out of solution and form bubbles, potentially producing GBD in the fish or invertebrates occupying these depths.

To aid in the evaluation of the potential risk of TDG supersaturation to fish in rivers, reservoirs and lakes we initially prepared an annotated bibliography on which this literature review is based. The annotated bibliography (Weitkamp 2007) is available at:
http://www.parametrix.com/profile/tech.htm.

# TDG SUPERSATURATION EFFECTS: FIELD vs. LAB CONDITIONS

Much of our knowledge of the effects of various water quality parameters on aquatic biota has been developed through laboratory investigations where conditions can be controlled to produce results directly affected by the parameter of interest. For most water quality parameters such as dissolved oxygen or temperature the laboratory conditions are appropriate for isolating the biological effects of the specific parameter. However, this is not true for TDG supersaturation because of the controlling effect hydrostatic pressure (depth) has on the expressed biological effects as discussed above. The behavior that affects the depth distribution of aquatic biota in field conditions is not reliably replicated in the controlled condition evaluations resulting in substantial differences in the results of laboratory and field investigations.

In this section we discuss the effects of TDG supersaturation on fish under "field" conditions and contrast them to artificial laboratory conditions. We define field conditions as the exposure of fish to the actual levels of TDG supersaturation they experience at the depths that they occupy in lakes, reservoirs, and free-flowing rivers. With field conditions the fish generally have sufficient depth available to substantially reduce or eliminate their exposure to true TDG supersaturation. The following identifies how their behavior influences biological effects in field conditions.

When we consider the biological effects under the real world conditions of river and lake environments as opposed to laboratory conditions we find very different results due to available water depth. Because of the major effect hydrostatic pressure has on dissolved gas solubility, and therefore the compensation that depth provides, biological effects are almost always very much less in the field environment than in the shallow water of laboratory investigations. Available depths and fish behavior commonly provide compensation for some degree of TDG supersaturation in the natural world that is not available under laboratory conditions. Recognizing the basic difference between these two conditions is important because the established TDG criterion is based primarily on results of the laboratory investigations where fish were restricted to shallow water.

## OBSERVATION CONDITIONS

Essentially all observations of GBD place the fish under reduced pressure conditions (shallow water) prior to observation. In some cases, such as fish collected by electrofishing or seining, the fish are only exposed to the surface pressure for a few minutes prior to observation, although they are collected from shallow water. In other cases, such as fish sampled from gatewells and bypass systems at dams, the fish are generally exposed to reduced pressure conditions for hours to days prior to observation. These reduced pressure conditions tend to increase the incidence and severity of GBD signs, depending on the duration and specific pressure conditions experienced by the fish prior to observations being made. Likewise fish collected by electrofishing and beach seining are taken from the portions of the populations occupying shallower water and are therefore more likely to develop GBD signs than those members of the populations occupying deeper water.

Backman et al. (2002) found that GBD signs are more common at higher TDG values in fish bypassed at dams than in fish collected upstream or downstream from dams. This is consistent with the shallow water experience of bypassed fish that commonly occurs during diversion, collection, and holding within the bypass systems.

The signs of GBD reported are somewhat variable. Easily visible signs, subcutaneous bubbles in fins, eyes, mouth, or other body parts, along with hemorrhaging are readily visible with unaided vision used in many investigations have been conducted with unaided vision that have often not recording lateral line bubbles. Small bubbles in the lateral line are difficult to detect with unaided vision. Some reports do not distinguish if bubbles in the lateral line are or are not included in the reported observations.

## VARIABLE DEPTH

The hydrostatic pressure provided by water depth appears to provide both direct compensation and indirect benefits. Knittel et al. (1980) reported that the time a fish spent at depth prior to exposure to high TDG levels appears to provide additional protection from GBD. They observed that juvenile steelhead survival nearly doubled when the fish were held for three hours at a depth of 3 m (9.8 ft), before being exposed to TDG levels of 130% of saturation. Schnute and Jensen (1986) used a general multivariate dose-response model applied to a data set derived from available literature. They determine that water depth and fish length significantly improve the model's ability to explain variations in TDG $ET_{50}$ (effective threshold 50%) estimates. They estimated that for 50-d exposures the apparent safe levels of TDG range from 104 to 115% (in shallow water).

Intermittent exposure occurring as fish move up and down in a water column also reduces the risk of GBD for a given level of TDG supersaturation. Weitkamp (1976) observed juvenile salmonids that were moved up and down within the top several meters of the water column experienced a lower incidence of GBD and mortality with greater time at depth and less time near the surface. Similar results have been observed by Knittel et al. (1980) and Antcliffe et al. (2002).

In the real world, fish do not commonly remain at a fixed depth and most are routinely deeper than fish held in shallow laboratory tanks. Fish commonly spend much, and in many cases all of their time at depths greater than 1 m. The depths that the fish naturally occupy provide hydrostatic compensation that either avoids the effects of TDG supersaturation, or mitigates their exposure during the time they do spend in shallower water.

# CONFINED DEPTH INVESTIGATIONS

A substantial number of additional investigations have been conducted with salmonids and other fishes confined to shallow depths (< 1m) as a means to produce and investigate GBD. These studies have continued to demonstrate that fish confined to shallow water suffer substantial rates of GBD and mortality that increase as TDG levels increase. Many of the laboratory investigations restrict fish to within 30 cm of the water surface or less. However, even a small increase in available depth can make a substantial difference in the biological effects of TDG supersaturation.

## SALMONIDS

Even with the extremely shallow water of hatchery incubation trays (depth <4 cm) Jensen (1980, 1988) found exposure of steelhead embryos, alevins, and fry to constant TDG levels of 102%, 106% and 110% had limited effects. Embryo survival was not affected at any TDG level tested. Alevin survival was marginally affected at 110% with a 1.4% incidence of opercular deformities within a few days due to bubble growth in the buccal cavity. A few fry (2.6%) exposed to 111% TDG developed GBD signs in the form of burst swim bladders. Fry size was not significantly correlated with TDG after 21 days of feeding.

The tolerance of lake trout sac fry to TDG supersaturation confined in water 15 cm deep was investigated by Krise and Herman (1989). At 106% TDG, 40% of the fry had bubbles around the rim of their eyes. Many fry reared at 116% and 120% TDG had bubbles in the mouth and jaws as well. Krise and Smith (1993) describe the eye abnormalities in detail. Most sac fry survived the 40-day exposure, however many were moribund at 40 days exposure. Krise (1993) reared lake trout fry in shallow water (15 cm) for one year at various TDG levels of 101-110%, observing no GBD signs at or below 108% TDG, and in only 3% of trout reared in 110% TDG. Krise concluded 108% TDG was a safe level, but found better growth at lower TDG levels.

Fickeisen and Montgomery (1978) held mountain whitefish (*Prosopium williamsoni*) and cutthroat trout (*Salmo clarki*) in shallow (20 cm) cages within a tank. They found mountain whitefish to be slightly more susceptible than cutthroat trout to TDG with all fish dying within two days at TDG levels of 124% and 128%. Lund and Heggberget (1985) reported rainbow trout held in 117-125% TDG within 30 cm of the surface suffered higher rates of mortality than trout allowed to seek the depth of their choice in a 1.6 m deep tank. Trout restricted to a depth range of 0.3-1.6 m had lower mortality (28%) than those allowed access to the surface (43%).

When confined in water 28 cm deep at a TDG level of 130% juvenile Chinook began to die after 5 h exposure, reaching about 50% mortality at 9 h (Mesa et al. 1997). At 120% TDG in the shallow water the mortality rate followed a sigmoid curve reaching about 43% at 58 h. With both 120% and 130% TDG the Chinook developed bubbles in the lateral line, fins, and gill filaments, with the GBD developing more slowly at 120% than at 130% TDG. Exposure of Chinook to 110% for up to 28 d did not produce mortalities and resulted in few GBD signs (Mesa et al. 2000a, 2000b).

Schrank et al. (1997) held juvenile Chinook in cages (surface cages 0-0.5 m, submerged cages 2-3 m, and large net-pens with an inclined bottom 0-4 m) downstream from Ice Harbor Dam (Snake R.) and Bonneville Dam (Columbia R.) in 1995. During their investigation TDG levels were near or exceeded 130% (peak 138%) downstream from Ice Harbor Dam. They observed incidences of GBD signs of 80% 0-0.5 m cage, 52% 0-4 m cage, and 6 % 2-3 m cage. Chinook held in the 0-0.5 m cages experienced 58-100% mortality and in the 0-4 m net pens 0-84% mortality with substantial variability among tests at similar average TDG levels. When TDG decreased to about 118% the mortality of captive fish was negligible. They concluded GBD incidence and mortality rates in the confined conditions were not representative of river conditions.

Exposure of fish in shallow water at TDG levels that produce GBD and mortality does not necessarily produce long-term effects in survivors. Gale et al. (2004) exposed adult female Chinook salmon to 114-126% TDG in 0.5 m of water until one test fish died or showed signs of impending death (10-68 h). The size of the females provided no more than about 0.25 m of water depth above their backs. Following TDG supersaturation exposure the females were held for 4-6 weeks in standard hatchery conditions with no observe effects of TDG supersaturation on pre-spawning mortality, fecundity, or egg quality.

Likewise Krise and Herman (1991) found sub-yearling lake trout and Atlantic salmon were resistant to TDG supersaturation (102-115%) in tanks with depths less than 15 cm. They observed no difference in weight gain, condition factor, food conversion, or mortality through 98 days. Similarly, Schisler et al. (1999) did not find TDG levels up to 110% affect growth, morbidity, or survival of rainbow trout infected with *Myxobolus cerebralis*, the causative agent of whirling disease and reared in shallow water.

Weitkamp et al. (2000b) held young rainbow and cutthroat trout in cages with maximum depths of 2 m in the lower Clark Fork River during exceptionally high TDG levels (+140%) in 1997. All these fish died within 4 d. Most fish surviving more then 2 d exhibited severe GBD signs. In a second test with TDG levels of 123-128% less than 20% of fish died during the 4 d exposure and GBD signs were observed in a maximum of 28% of fish examined each day. Apparently these fish did not remain near the bottom of the cages where they would have experienced sufficient hydrostatic compensation to minimize the effects of the TDG levels.

Antcliffe et al. (2002) exposed juvenile rainbow to TDG levels of 110%, 114%, 116%, 122% and 144 % in water depths of 0.25 m and 1 m. They observed mortalities at 116% TDG and higher in 0.25 m, but no mortalities with 6 days exposure at 114% TDG. At 116% TDG 42% of the fish died within 9 d, and at 122% TDG 53% of the fish died within 49 h. At 122% TDG, the cumulative 96 h mortality was only 22% in fish held within 1 m of the surface as compared to 89% mortality in the fish held within 0.25 m of the surface. In 1 m deep cages the mortalities began to occur until 36 h of exposure at 122% TDG. They concluded that fish use of the range of depths available in the 1 m cage significantly delayed the initial onset of mortality and the percent cumulative mortality over a 96 h exposure as compared to the depth of 0.25 m. In cages with depths of 0-2.5 m they observed the young trout appeared to school at depths greater than 1m. Some trout were held in 0.25 m deep cages and subsequently moved to a depth of 2.5-2.75 m for 3 h or 6 h following 10% mortality at the near surface exposure. This dynamic exposures significantly delayed mortality compared to the baseline static exposure

In 1999 Weitkamp et al. (2000b) held kokanee fry in 9 m deep live-cages at the edge of Lake Pend Oreille. None of the kokanee showed GBD signs despite TDG peaks of 125% of saturation. Hydroacoustic monitoring indicated that the kokanee spent considerable time at depths greater than 1 m. During pre-dusk hours the fry were observed to slowly move closer to the surface where they remained for several hours before dispersing to depths of about 4 m. During the pre-dawn hours the fry were observed to slowly move deeper in the water column to reside at depths of 8-9 m during the day.

## NON-SALMONIDS

The effect of TDG supersaturation on white sturgeon (*Acipenser transmontanus*) larvae in shallow water (0.25 m) was investigated by Counihan et al. (1998). At 131% TDG the larval stages characterized by the formation of the mouth and gills developed bubbles in the buccal cavity and/or nares. Larvae held at 118% TDG did not experience mortality in a 10 day exposure, while those held in 131% TDG had 50% mortality in a 13 day exposure.

The tolerance of largescale sucker (*Catostomus macrocheilus*), longnose sucker (*C. catostomus*), northern pikeminnow (*Ptychocheilus oregonensis*), redside shiner (*Richardsonius balteatus*), and walleye (*Stizostedion vitreum*) to various TDG levels (115, 125, 130%) in a depth of 26 cm was reported by VanderKooi et al. (2003). At 115% TDG they observed few mortalities in up to four weeks of exposure, but dramatic GBD signs were observed with prolonged exposure in the shallow water. Exophthalmia only occurred after 216 h exposure at 115% TDG. At 125% and 130% TDG in depths of 26 cm mortalities occurred without extensive GBD signs. With exposure to 125% TDG the times to 50% mortality were northern pikeminnow 15.2 hr, largescale sucker 17 hr, longnose sucker 56 hr, redside shiner 116 hr, and walleye 169 hr. At 130% TDG $LC_{50}$ (lethal concentration 50%) times were approximately half or less than at 125% TDG. No observations were reported indicating that the differences among the species sensitives te TDG were related to behavioral differences (depth distribution) or inherent species susceptibility. They determined that the progression of GBD signs was not predictive of mortality; however the severity of GBD signs increased somewhat with higher TDG levels. VanderKooi et al. (2003) found lateral line pore sizes to be inversely related to levels of lateral line occlusion, suggesting that fish with larger pores are less likely to develop high levels of lateral line occlusion.

Cages holding fish within 20 cm of the surface within a deep tank were used by Fickeisen and Montgomery (1978) to investigate the relative susceptibility to TDG supersaturation of several resident species. They found mountain whitefish and cutthroat trout to be substantially more sensitive than largescale sucker. All whitefish and cutthroat died within 2-4 d at 120% TDG and higher levels, while 20% of the suckers remained alive through 10 days at 120% TDG. Torrent sculpins (*Cottus rhotheus*) were highly resistant with 70% surviving through 10 days at 120% TDG.

The tolerance and behavioral response of carp (*Cyprinus carpio*) and black bullhead (*Ictalurus melas*) from Italy to TDG supersaturation was reported by Gray et al. (1982) and Gray et al. (1983a). Carp exposed in tanks 30 cm-deep to 110-150% TDG had a 96 h $LC_{50}$ of about 123% TDG, while bullheads were more sensitive with an $LC_{50}$ of about 114%. The fish only avoided supersaturation at extreme levels (146% TDG). The black bullhead was slightly more susceptible to TDG supersaturation. Both species tended to be less tolerant of TDG supersaturation when forced to swim (lotic conditions) then when in quiescent conditions (Gray et al. 1983b).

Schrank et al. (1997) held resident fish (smallmouth bass *Micropterus dolomieu*, yellow perch *Perca flavescens*, and peamouth *Mylocheilus caurinus*) in cages (surface cages 0-0.5 m, submerged cages 2-3 m, and large net-pens with an inclined bottom 0-4 m) downstream from Ice Harbor Dam (Snake R.) and Bonneville Dam (Columbia R.) in 1995. The TDG levels were near or exceeded 130% (peak 138%) downstream from Ice Harbor Dam during most of their investigation. They observed GBD signs incidences of 97% in the 0-0.5 m cage, 37% in the 0-4 m cage, and 40% in the 2-3 m cage. Many resident fish had GBD signs when placed in the cages. Resident fish mortalities were 0-16% in the 0-4 m net pens. When TDG decreased to about 118% the mortality of captive fish was negligible. They concluded the cage results were not representative of fish in the river. In 1996 Shrank et al. (1998) reported the resident fish held in these cages frequently showed an increase in GBD signs from 0 to 86% with high TDG levels averaging above 125% for a prolonged period with peaks up to 142%. They found no correlation between GBD signs and mortalities in the captive fish.

Ryan and Dawley (1998) also reported holding resident fishes in cages (0-0.5, 2-3, and 0-4 m) during periods of high TDG (125 to >140%) in the Snake River during 1997. They found the highest incidence of GBD in fish held in the 0-0.5 m cage when TDG levels were higher than 120% with bubbles in the lateral line frequently increasing after a 4 d holding period. When TDG levels were less than 120% GBD signs were static or decreased in fish held in the net pens. Ryan et al. (2000) summarizes the results of the cage experiments from 1994 to 1997 during periods of high TDG. They were unable to keep holding conditions consistent for deep and shallow cages, and therefore were unable to use the data for all years in development of a model.

Counihan et al. (1998) reported the rapid development of GBD in white sturgeon (*Acipenser transmontanus*) larvae held in shallow water (25 cm) and exposed to high TDG (118-131%). They observed no mortality in sturgeon larvae held in 118% of saturation for 10 days however, exposure times of 15 minutes were sufficient to produce bubbles in the buccal cavities and nares of some larvae. GBD resulted in positive buoyancy and altered behavior. Exposure of larvae to 131% TDG for 13 days produced a 50% mortality.

## INVERTEBRATES

Nebeker et al. (1981) determine the effects of TDG supersaturation on the survival and adult emergence of mayflies *Timpanoga hecuba*, caddisflies *Dicosmoecus gilvipes*, mosquitoes *Culex peus*, and midges *Cricotopus* sp. in laboratory tests. They found all insects were more tolerant of TDG supersaturation than fish. The 96 h $LC_{50}$ for the mayfly was 129% TDG. At 125% TDG the mayfly $LT_{50}$ was 2.7 d, while at 135% TDG the caddis fly $LT_{50}$ was 45 d. Adult midges and mosquitoes emerged at TDG levels > 140%. Adult mayflies and caddis flies did not emerge at TDG levels of about 134% and higher.

White et al. (1991) conducted bioassays (<1m) of macroinvertebrates showing that most were negatively affected by 127% TDG or greater in shallow water. *Baetis tiicaudatus* was the most susceptible with adverse effects observed at 115% TDG. *Ephemerella inermis* and *Tricorthyodes minutus* had a susceptibility threshold near 118% TDG.

Bubble formation in the ciliate *Tetrahymena pyriformis* was investigated by Hemmingsen (1986). Rupture of the ciliate cells did not occur until TDG pressures exceeded 25 atm, an extremely high TDG level.

## GBD OBSERVED UNDER FIELD CONDITIONS

Since the late 1960s researchers have recognized that TDG supersaturation in the Columbia River system has occurred and been a potential problem for juvenile and adult salmonids (Weitkamp and Katz 1980). This concern has been responsible for much of the research on TDG supersaturation and GBD. Commonly, substantial spill at numerous dams has produced TDG supersaturation throughout much of the river system during spring high flow periods. During these times, resident fish as well as juvenile and adult migrant salmonids have been exposed for substantial periods to TDG supersaturation. Dams throughout western U.S. and Canadian rivers, as well as other areas, have been found to produce TDG supersaturation at sufficient levels to raise concern for potential GBD in the aquatic biota.

In recent years spring and summer spill has occurred at many Columbia River basin dams to provide fish passage at the direction of the regulating agencies. This spill has produced substantial levels of TDG supersaturation, commonly in the range of 120% of saturation and higher.

Most of the investigations of GBD in fish exposed under field conditions have occurred in the Columbia River Basin (Figure 4) where the numerous dams have frequently produced substantial levels of TDG either through involuntary (inadequate powerhouse capacity) or required spill for fish passage. Monitoring efforts have investigated by juvenile and adult salmon migrants as they become available during dam passage, and resident fishes sampled from the reservoirs.

Recent literature provides a substantial number of observations of fish collected from field or natural conditions (rivers, reservoirs, lakes) where TDG levels have been substantial (120-150%). These observations provide information on the incidence and severity of GBD under conditions where fish commonly have the opportunity to occupy depths providing partial or complete hydrostatic compensation of the recorded TDG levels. However, it should be recognized that the collection methods tend to sample fish primarily from relatively shallow water. Collecting fish from shallow water conditions provides only that portion of the total fish population that is most likely to experience GBD. Likewise, bypass systems at dams divert fish into shallow water where they are held for some time prior to being examined. Therefore, these samples are likely to over estimate the incidence of GBD in the total fish population.

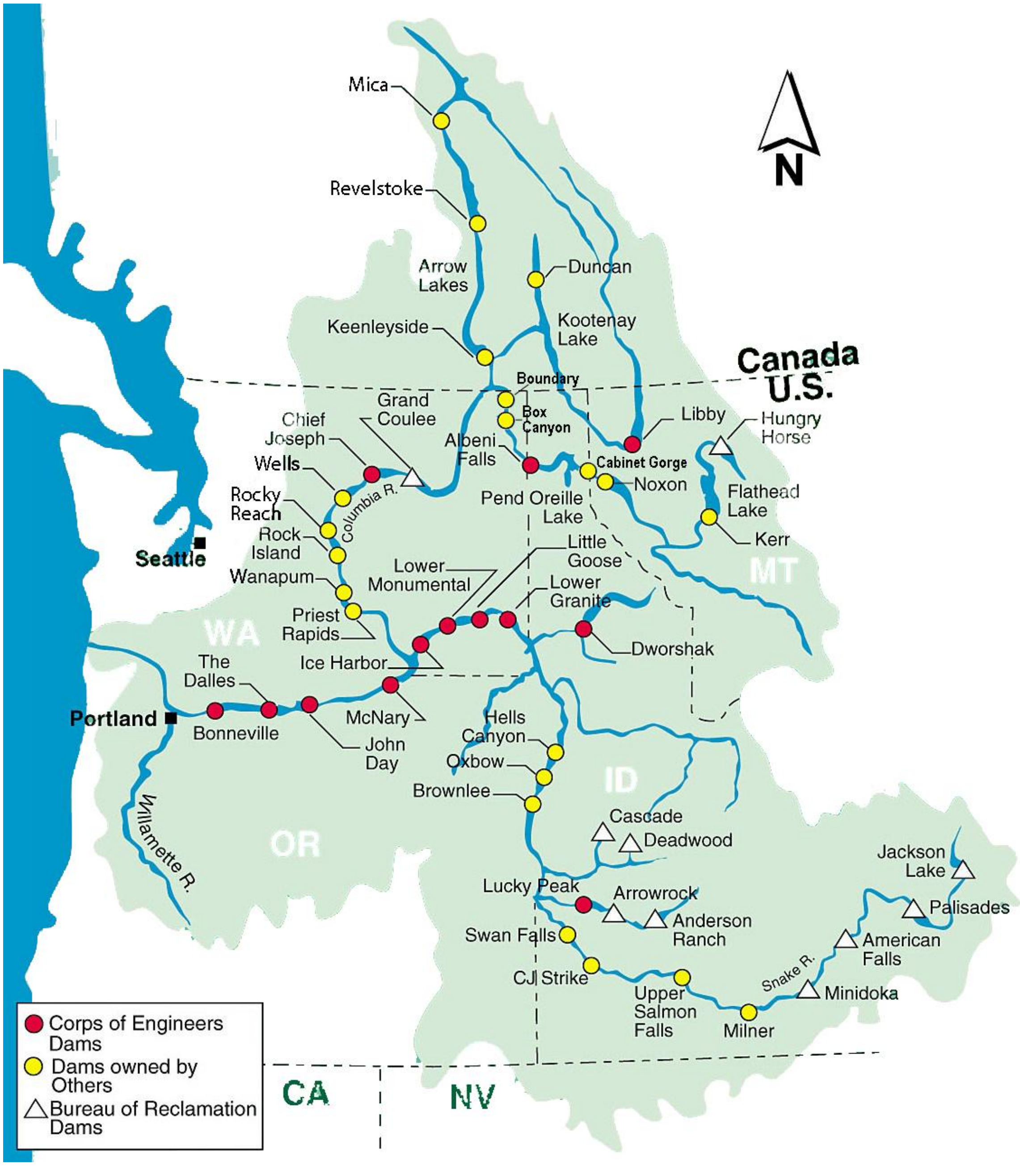

Figure 4. Major hydroelectric projects in the Columbia River Basin.

## INCIDENCE/SEVERITY GBD

Determination of the biological effects of TDG supersaturation in natural waters requires an understanding of the incidence and severity of GBD in populations exposed to TDG supersaturation. Although severity of GBD signs is not a direct indicator of mortality, it does provide useful complimentary information to the incidence of GBD, particularly with chronic (moderate TDG) but not acute exposures (very high TDG). Information is available on various life stages of salmonids from developing embryos to maturing adults.

## Juvenile Salmonids

Some information is available on the earliest life stages of salmonids. Gale et al. (2004) found the high survival of Chinook embryos produced by mature females exposed to TDG levels of 114 to 126% in shallow water (0.5 m). Survival to eyed-stage was high (> 94%) for all groups. They conclude that acute exposures of females to moderate TDG levels prior to spawning did not affect reproductive success of female Chinook salmon. Previously Jensen (1980) observed steelhead embryos exposed to TDG supersaturated water (110 %) in very shallow water (apparently < 4 cm) did not suffer any detectable mortality. Following hatch, alevins were only marginally affected with a slight increase in mortality.

Much of the data identifying the presence or absence of GBD in juvenile salmonids during periods of TDG supersaturation has come from routine examination of downstream migrating juveniles collected from bypass systems of major Columbia and Snake River dams. Generally these data have shown low incidence and severity of GBD in the juvenile salmonids. Most of the records of GBD in young salmonids are not accompanied by any evidence of mortalities. The available information also indicates that in field conditions when mortalities have been documented the TDG levels are commonly near or grater than 130%.

In 1985 with TDG levels seldom exceeding 120% Dawley (1986) monitored juvenile salmonids for GBD signs in the lower Columbia River at The Dalles and Bonneville Dams. No GBD signs were detect any during the period of peak TDG. In 1986 with TDG exceeding 120% throughout much of the river in May and early June GBD signs were observed in one or two fins of 0-7% of fish monitored (Dawley 1986).

Toner and Dawley (1995) reported GBD signs were infrequently observed downstream from Bonneville Dam, Columbia River, during 1993 when TDG levels reached a peak of 128%, and were above 120% for only 4 d. GBD signs were seen in young Chinook (1%), coho (3%), and steelhead (2%). In 1994 Toner et al. (1995) examined juvenile salmonids downstream from Ice Harbor, and Bonneville Dams. Downstream from Ice Harbor Dam TDG levels were above 130% for 7-11 h each day in early May peaking at 136%. The TDG levels reached or exceeded 120% on most days in May, however average TDG levels remained below 120% during this period. They observed GBD signs in 5-10% of fish captured by electrofishing downstream from Ice Harbor Dam, but rarely in fish collected downstream from Priest Rapids and Bonneville Dams.

In 1996, TDG levels were high in the lower Snake and Columbia Rivers during the spring migration period, exceeding 120% for the first eight weeks of monitoring (Hans and Maule 1997). They reported that GBD signs in fish collected at the dams remained relatively low with averages of about 2-4% of spring Chinook and 6-7% of steelhead. At Ice Harbor Dam on the Snake River the incidence of GBD signs was 3.7% in spring Chinook, and 5.9% in steelhead. At lower Columbia River dams the incidence was 2.3% in spring Chinook and 7.3% in steelhead. At John Day Dam the incidence of GBD signs was 5.5% in spring Chinook and 9.8% in steelhead. At Bonneville Dam the incidence of GBD signs in spring Chinook was 0.8% and 9.9% in steelhead. Mesa and Maule (1997) concluded it appeared GBD was not a threat to migrating juvenile salmonids when TDG supersaturation was < 120%.

Monk et al. (1997) collected, tagged and then exposed juvenile steelhead at lower Snake River Dams to TDG supersaturation oy 113-127% in shallow water (46 cm) to develop relatively sever signs of GBD prior to releasing the fish back to the river. Only 47% of the steelhead released that initially had GBD signs retained GBD signs when recaptured at the downstream dam, while fish showing no signs at release had a 5.9% incidence of signs at recapture.

Backman et al. (2002) collected smolts upstream and downstream from Columbia Basin dams and compared the incidence of GBD signs with that of smolts collected by the Smolt Monitoring Program, which collected smolts from dam bypass systems during 1996 through 1999. They found that fewer than 2% of salmonids displayed external signs of GBD, and most of those had less than 5% fin occlusion. Backman et al. (2002) found that GBD disease is more common in fish bypassed at dams than in fish collected upstream or downstream from dams during periods of high TDG levels. Bypass systems have shallow depths that exacerbate exposure to true TDG supersaturation. However, the data for each of these groups followed the same trend of increasing GBD with increasing TDG. Backman et al. (2002) concluded that the incidence and severity of GBD signs in fish from the river and dam bypass systems were less than expected based on laboratory studies.

Backman et al. (2002) provide a regression analysis of juveniles examined from dam bypass systems as well as fish collected from the river. This analysis indicates that the incidence of GBD remains low with TDG levels of 120% or less, and then rises steeply as TDG increase above 120% (Figure 5).

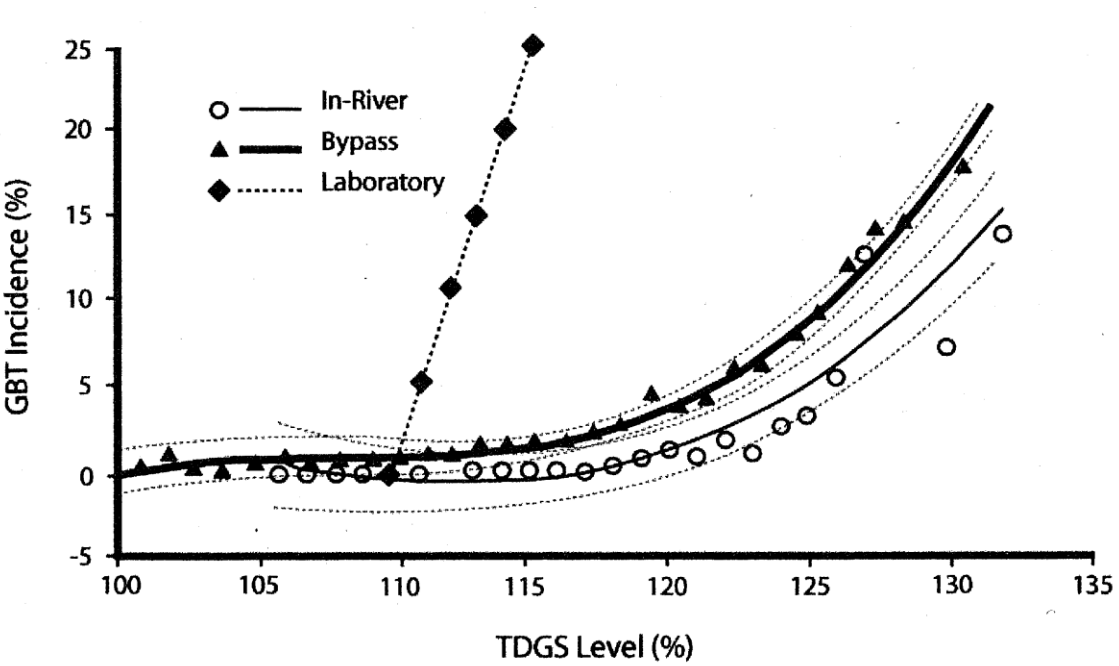

**Figure 5. Incidence of gas bubble disease in juvenile salmon, Columbia River 1996-1999, from Blackman et al. (2002).**

In the mid-Columbia region (Wells Dam RKm 829 to Priest Rapids Dam RKm 639) juvenile salmon have been routinely collected and monitored for GBD at a number of dams. Fish have been collected by several means from gatewells and bypass systems as they migrate past the dams.

Monitoring of juvenile salmon migrating past Rocky Reach Dam (RKm 762) and Rock Island Dam (RKm 730) has been conducted for a number of years (Mesa and Maule 1997, Grassell and Hampton 2001, Grassell et al. 2000a, Hampton 2002, 2003; Hampton and MacDonald 1998, 2000, Murdoch and McDonald 1997, Maitland et al. 2003). TDG levels in the range of about 110% to 120% have been common during the spring migration period. The incidence of GBD has been in the range of 1-5% for Chinook (yearling and sub-yearling) and steelhead. In 1997 when TDG levels exceeded 120% for two months the incidence of GBD was 20-80% in the juvenile salmon collected at these two dams. In 1997, a small portion of the fish (0.5-1.6%) had bubbles in their eyes, indicating more sever GBD with the prolonged TDG levels well above 120%. In 2000 Grassell et al. (2000a) found GBD signs in 2.7% of juveniles at Rocky Reach Dam and 3.5% at Rock Island Dam. The TDG levels upstream from Rocky Reach ranged from 100% to 121% of saturation, while TDG levels downstream ranged from 102% no 132% (Grassell et al. 2000b).

The incidence of GBD in young salmonids examined at these dams may have been exacerbated by the water depth in the holding facilities (Rocky Reach 0.8 m, Rock Island 0.25 m) where juveniles are held for 0.5 to 24 h prior to examination. The severity of the GBD signs has been generally low as seen at other Columbia River dams.

Sampling of juvenile migrants at Priest Rapids Dam has been conducted in recent years. In 1997, the incidence of GBD signs during the spring migration at Priest Rapids Dam was: Chinook 8.7%, steelhead 9.4%, and sockeye 21.5% with TDG levels of 120-135% (Hagen et al 1998). However, during the summer the incidence of GBD signs dropped to 2.3% in subyearling Chinook with TDG levels of 110-125%. Duvall et al. (2002) reported juvenile salmon had an incidence of GBD signs of 1.7 to 8.3% during spring spill periods of 1996 to 2002 when TDG averaged 113 to 130% (Table 1). During 1998, TDG levels briefly exceeded 125% (Hagen and Weitkamp 1999b). They observed similar incidences of GBD signs (1.7-5.8%) in summer migrants with TDG levels averaging 113-120.1%. The majority (91%) of fish with GBD signs had a rank 1 (5% or less area affected), the lowest level within the GBD ranking and none showed the most severe rank 4.

**Table 1. Incidence of GBD signs in juvenile salmonids captured at Priest Rapids Dam, 1996-2002 (from Duvall et al. 2002).**

| Year | Season | Incidence GBD (%) | Average TDG (%) |
|---|---|---|---|
| 1996 | sp | 8.5 | 124 |
| | sm | 1.8 | 117 |
| 1997 | sp | 11.1 | 130 |
| | sm | 2.3 | 116 |
| 1998 | sp | 3.8 | 116 |
| | sm | 4.7 | 113 |
| 1999 | sp | 3.6 | 114 |
| | sm | 1.7 | 113 |
| 2000 | sp | 8.3 | 114 |
| | sm | 5.8 | 114 |
| 2001 | sp | 3.9 | 112 |
| | sm | 2.7 | 110 |
| 2002 | sp | 4.3 | 116 |
| | sm | 5.9 | 120 |

The incidence of GBD in young Chinook, coho, steelhead, and lamprey in the Clearwater River (Snake River tributary) downstream from Dworshak Dam from 1995 through 1999 was reported by Cochnauer (2000). The TDG levels exceeded 110% for more than a month during each year and in 1995-1997 exceeded 120% for 10, 17 and 20 days respectively. The incidence of signs of GBD was less than 1%. Rainbow trout showed the highest incidence of GBD (4.5% and 9.4%) in 1996 and 1997 when TDG exceeded 120% for the greatest number of days. Most fish showing signs of GBD had the lowest rank (mild signs). Small numbers of mountain whitefish and largescale sucker were also observed to exhibit signs of GBD.

In the Hells Canyon reach of the Snake River Richter et al. (2007) used electrofishing to collect resident and anadromous salmonids downstream during periods of TDG supersaturation (up to 143%). When TDG did not exceed 120% they did not observe GBD signs. They found resident rainbow trout had a high incidence (17%) of GBD during periods when TDG was near or exceeded 130%. They did not find GBD signs in any Chinook, kokanee, or steelhead.

Parametrix (1998) released approximately 5,000 hatchery cutthroat trout into the lower Clark Fork River during a period of TDG levels of up to 120%. Only eleven of these fish were recaptured, apparently due to rapid migration to Pend Oreille Lake or deep water in the river. No GBD signs were observed in the captured cutthroat. Ten radio tagged hatchery cutthroat indicate that these hatchery fish remained in the river for at least 3 days.

Weitkamp et al. (2000a) collected juvenile kokanee from the lower Clark Fork River, Idaho, When TDG levels were 105-137%. None of the kokanee fry that were released from the Cabinet Gorge Hatchery and collected by fyke net near the river mouth 18.5 km (10 mi) downstream showed GBD signs.

In 2006 TDG supersaturation occurred downstream from Libby Dam, Kootenai River (upper Columbia River Basin) during spill tests. Supersaturation exceeded 110% for 20 days, reaching a peak of 131% TDG. GBD was observed in rainbow trout (*Oncorhynchus mykiss*), westslope cutthroat trout (*O. clarki lewisii*), kokanee (*O. nerka*), bull trout (*Salvelinus confluentus*) and mountain whitefish (*Prosopium williamsoni*). Signs in trout were observed on the fourth day of spill and increased in frequency as spill continued.

Mathias and Barica (1985) found GBD to be a cause of mortality in juvenile rainbow trout held under the ice in a shallow prairie lake. In their investigation of mortalities to stocked fingerlings they determined at least some of the mortalities were the result of TDG supersaturation. TDG levels exceeded 130% when the mortalities occurred.

## Adult Salmonids

Signs of GBD were first observed in adult salmon at John Day Dam during a substantial mortality (Beiningen and Ebel 1970). During the spring of 1968 all water passed over the spillway at John Day Dam because the turbines were not yet installed. During this period TDG levels were measured in the range of 123-142%. The migration of adult salmon was also delayed at John Day Dam tailrace due to problems in the fish-passage facilities, causing the adults to spend a prolonged period of time in the supersaturated water. These extreme conditions produced the only recorded substantial mortality of adult salmon resulting from TDG supersaturation.

Dauble and Muller (1993) determined that there was some potential risk of TDG supersaturation to adult salmon migrating upstream in the Snake River during the spring spill periods. However, they did not have sufficient information to evaluate the degree of the risk.

In 1986, during a prolonged period with TDG exceeding 120% throughout the lower river in May and early June, Dawley (1986) reported no GBD signs were observed in 28 adult Chinook and steelhead examined at McNary Dam (RKm 472). More recently Fryer (1995) reported monitoring of 3-4% of adult Chinook, sockeye and steelhead migrating past Bonneville Dam in the spring of 1995. Visible GBD signs were not detected in any of the monitored fish.

Backman and Evans (2002) examined adult salmon for GBD signs in the lower Columbia River from 1995 to 1999. They examined 4,667 adult Chinook salmon, 1,878 sockeye salmon, and 1,431 steelhead at Bonneville Dam to determine the incidence of GBD relative to TDG supersaturation. The GBD signs they observed were generally minor (<5% fin occlusion), with severe bubbles (>26% fin occlusion) observed in sockeye salmon (15 fish) and steelhead (2 fish) only when TDG exceeded 126%. Chinook salmon were rarely observed with GBD, despite the sampling of large numbers when TDG levels exceeded 130%. Fryer (1995) reported observations of adult Chinook, sockeye and steelhead migrating upstream through the bypass facilities at Bonneville Dam for GBD signs during 1995. No visible GBD signs were observed during a period when TDG levels were 114-120%. Adult salmon were also examined upstream in the Hanford Reach when TDG levels were generally 110-119% without observing any fish with GBD signs.

Backman and Evans (2002) developed polynomial regression models evaluating the effects of TDG supersaturation on adults of three salmonid species (Chinook, sockeye, steelhead) (Figure 6). These regressions are derived from observations of large numbers of adults examined as they passed over Bonneville Dam during periods of high TDG. The regressions show an absence of GBD signs until TDG levels are well above 120% of saturation. They did not find a relationship between TDG supersaturation and GBD in Chinook, but a significant positive association was identified for sockeye and steelhead.

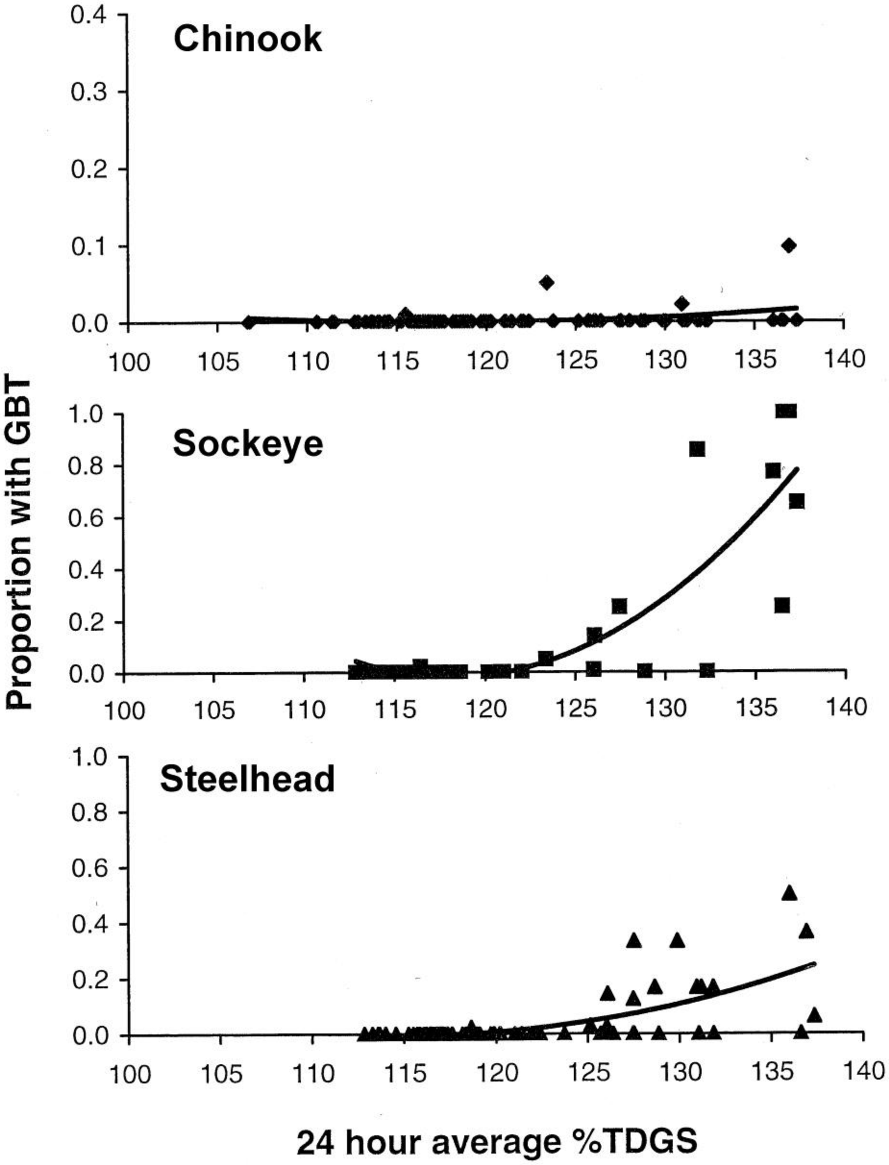

**Figure 6. GBD incidence (polynomial regression models) for Columbia River juvenile salmonids (from Backman and Evans 2002).**

Resident adult salmonids were collected from the lower Clark Fork River, Idaho, by Weitkamp et al. (2000a, 2003) during four years with moderate (115-125%) to very high TDG levels (>140%). They found no GBD in Lower Clark Fork River resident salmonids in 1998 when TDG levels were variable with frequent TDG peaks of 115 to 125%. In 1999, during high TDG levels (125-130%) they detected GBD signs in one brown trout of the 102 salmonids examined (Parametrix 1999). In 2000, they observed a relatively low incidence of GBD in rainbow trout (4%) and brown trout (4%), and none in cutthroat trout and bull trout during a period when TDG levels frequently reached daily peaks of 120-130% interspersed with lower levels (Weitkamp et al. 2003).

Johnson et al. (2005) found that radio-tagged adult Chinook salmon migrated at depths exceeding

2 m the majority of the time. These fish spent only minutes near the surface thereby encountering only brief exposure to true supersaturation with TDG levels at and above 120%. They concluded that their data suggest little potential for negative effects of TDG supersaturation on adult Chinook salmon under average river conditions, despite the fact that fish tissues were probably supersaturated with dissolved gases relative to surface pressure.

Cochnauer (2000) conducted sampling of resident fish for GBD signs in the Clearwater River downstream from Dworshak Dam over five years. He observed GBD signs predominately in rainbow trout, mountain whitefish, and largescale sucker. Most fish had GBD signs of the least severe rank and were associated with TDG levels approaching 120% for extended periods of time. The incidence of GBD was generally less than 1%, with the exception of rainbow trout that had an incidence of 7% in 1997, an extremely high flow year with TDG exceeding 120% for most of late April and early May. No mortalities were reported.

In 2002, a series of spill events at Libby Dam on the Kootenai River produced TDG levels of 120% to >125% of saturation (Dunnigan 2002). Less than 1% of resident fish within 1.7 miles of the dam exhibited GBD signs. The majority of the bull trout, rainbow trout and mountain whitefish collected via electrofishing one week after the final spill event exhibited fin damage, presumably caused by necrosis of the fin tissue caused by gas emboli. An exceptionally high spill at Libby Dam in 2006 produced GBD signs in substantial numbers of resident salmonids (Marotz et al. 2007). In late spring TDG levels exceeded 120% (123-131%) for nearly 20 days. Rainbow trout, westslope cutthroat trout, kokanee, bull trout, and mountain whitefish were collected by electrofishing in shallow water during the 2006 spill to determine the incidence of GBD. Following 4 days of TDG exceeding 120%, GBD signs were observed in about 20% of the fish collected by electrofishing. By the 11th day, 80-100% of each species showed signs of GBD.

Resident salmonids were observed with GBD signs in the Kootenai River downstream from Libby Dam during spill tests that elevated TDG levels to above 110% for 20 days with a peak of 131% (Marotz et al. 2006). Rainbow trout, westslope cutthroat trout, kokanee, bull trout, and mountain whitefish were collected by electrofishing, with trout showing GBD signs on the fourth day of spill that increased in frequency as spill continued. Signs including multiple hemorrhages on the ventral body surface, bubbles in fins, eyes, and operculum; and split fins were observed in all bull trout and cutthroat captured after 11 days of spill, and in most rainbow and mountain whitefish. Hemorrhages increased when TDG levels approached 131% and were less prevalent when TDG levels declined toward 124%.

One of the concerns for TDG supersaturation is the potential affect it may have on reproduction. Recently, Gale et al. (2004) exposed mature female Chinook salmon to TDG levels of 114 to 126% in shallow water (0.5 m) to determine the affects of acute exposure on reproductive performance. They stopped test exposures at the first mortality (10 to 68 h). They did not observe an affect of TDG supersaturation on pre-spawning mortality or fecundity when comparing treated fish to experimental controls and to the general hatchery population. Egg quality did not differ between treatment and control fish. Fertilization rate and survival to eyed-stage was high (> 94%) for all groups. They conclude that these acute exposures to moderate TDG levels did not affect reproductive success of female Chinook salmon.

In the Bighorn River downstream from the Yellowtail Afterbay Dam White et al. (1991) monitored resident rainbow and brown trout for evidence of adverse affects of TDG supersaturation. This very shallow river has mid-channel depths of 1.8 m in the area of substantial TDG supersaturation. They sampled sites close to the dam (2.4 km downstream) and distant from the dam (14.5 km). Fish near the dam had higher rates of GBD than downstream fish and larger fish had higher rates than smaller fish. The incidence of GBD in brown trout was 28-64% immediately downstream from the dam. Fish with GBD signs were only observed within 8 km of the dam, with rainbow trout always having a lower incidence than brown trout. The mean TDG level near the dam was 116% with a range of about 105-123%. Downstream the TDG level range was 102-128%. The trout tended to concentrate near the river banks during the spring when high TDG levels occurred, exposing them to the higher TDG levels in the shallower portion of the available water depths.

## Non-Salmonids

Investigations of the affects of TDG supersaturation on non-salmonid fishes in Pacific Northwest and other rivers have included a substantial number of species over the last 30 years. GBD and some mortalities have been recorded at various locations.

Smallmouth bass and northern pikeminnow were collected from fishermen (apparently held in shallow water for a short period) and examined for GBD signs by Montgomery and Becker (1980) when TDG levels in the Columbia River were greater than 115%. They observed a high incidence of GBD with bubbles in the opercula, body, and fins of 72% of the smallmouth bass and 84% of the northern pikeminnow.

A series of reports provides information on the incidence and severity of GBD in resident non-salmonid fishes of the Columbia and Snake Rivers from 1994 through 1998. Schrank et al. (1997) reported high rates of GBD occurred in resident fish collected downstream from Ice Harbor Dam on the Snake River in 1995 when TDG levels remained near or above 130% (peak 138%) resulting from a prolonged turbine outage. They found a relatively high incidence of GBD in smallmouth bass (16.5%), crappie (13.6%), and brown bullhead (11.8%). In Priest Rapids Reservoir on the Columbia River GBD signs relatively low in resident fish when TDG levels were 118-125% (sandroller 5.6%, sculpin 4.8%, and smallmouth bass 1.6%). Downstream from Bonneville Dam general GBD signs were nearly absent in 1,936 resident fish examined with TDG levels of 114-120% from late April to mid-August, 1995, however, bubbles were observed in the lateral lines of 3.8% of fish examined.

In 1996, TDG supersaturation was high (125-145%) throughout the lower Snake and Columbia Rivers for 5-8 weeks (Schrank et al. 1998). They sampled resident fishes in Priest Rapids Reservoir as well as downstream from Priest Rapids, Ice Harbor, and Bonneville Dams. In Priest Rapids Reservoir resident relatively high GBD incidences were observed in suckers 26.6%, chiselmouth 6.9%, stickleback 5.9%, pumpkinseed, 5.1%, bluegill 16.7%, and sculpins 15.6%. Downstream from Priest Rapids Dam in the free-flowing Columbia River the GBD incidences were substantially lower (avg. 6.5%) with the highest incidences in suckers (13.2%), northern pikeminnow (2.1%), chiselmouth (4.5%), and smallmouth bass (7.7%). High TDG downstream from Ice Harbor Dam resulted in 6 species having 11-22% incidences of GBD, while downstream from Bonneville Dam the GBD incidences were low (suckers 3.3%, northern pikeminnow 3.4%, redside shiner 2.5%, and sculpin 3.4%). They observed few fish with GBD when TDG levels did not exceed 120%, but found up to 15% of the resident non-salmonids had GBD when TDG levels were between 120% and 130%.

Ryan et al. (2000) summarized the survey of non-salmonids for signs of GBD downstream from several Columbia and Snake River dams during the spring freshets of 1994-1997. In 1994, when TDG exceeded 120% for six weeks, they observed GBD in only 2.9% of 3,367 non-salmonids. In this survey of 39,924 nonsalmonids they reported GBD signs were rare in non-salmonids when TDG did not exceed 120% of saturation.

Parametrix (2000) monitored resident fishes in the Columbia River downstream from Rocky Reach Dam when TDG levels reached 130% for a brief period, but remained below 120% most of the spill period. They examined 2,134 resident fish during the spring with none showing GBD signs when TDG levels were 103-127%. During the summer they examined 866 resident fish for GBD signs, finding 18% showing some GBD signs when TDG reached a high of 134%. Fish with GBD signs included three-spined stickleback (50.5%), northern pikeminnow (22.0%), redside shiner (15.5%), peamouth (7.6%), and chiselmouth (3.7%). Most GBD signs were slight hemorrhaging between fin rays or at the base of fins and in the lateral line. Bubbles were only observed in a single fish.

In 1993, Toner and Dawley (1995) sampled resident fish downstream from Bonneville Dam in shallow water by beach seine when TDG levels were 103-120% finding GBD signs infrequently. Salmonids had higher incidences of GBD than resident fishes. GBD signs were infrequently observed when TDG levels reached a peak of 128%, but were above 120% for only 4 d. They observed GBD signs in less than 1% of peamouth and moderate to severe GBD signs (large bubbles on body and exophthalmia) in less than 1% of sticklebacks and prickly sculpins examined. They did not observe any GBD signs in invertebrates collected (dragonfly larvae, crayfish, Asian clams).

Maule et al. (2003) examined the scale patterns in longnose suckers, northern pikeminnow, and walleye, as well as rainbow trout from Rufus Woods Lake, downstream from Grand Coulee Dam. None of the species showed growth differences related to TDG levels recorded in the reservoir during their life period of 1996 to 1998. These observations indicate the levels of TDG supersaturation occurring during the years of 1996 through 1998 had not caused a population effect in the prevalent species. During this period the extremely high flow year of 1997 resulted in TDG levels in the range of 120-143% for nearly two months in Rufus Woods Lake.

In the Hanford Reach of the Columbia River, Montgomery and Becker (1980) found GBD signs present in 72% of smallmouth bass and 84% of northern pikeminnow when TDG levels exceeded 115%. The fish were collected from shallow spawning areas where the fish would be most susceptible to GBD. The observed GBD signs were bubbles beneath the skin, but no mortalities were reported.

Cochnauer (2000) found largescale suckers in the Clearwater River downstream from Dworshak Dam showed an increased incidence of GBD signs associated with TDG levels approaching 120%. However, most suckers exhibited only minor GBD signs (rank 1).

In Hells Canyon on the Snake River, Idaho, there is a three dam complex operated by Idaho Power that produces TDG supersaturation during periods of substantial spill (Richter et al. 2007). Richter et al. (2007) used electrofishing to collect fish downstream from these dams during periods of TDG supersaturation up to 143%. When TDG did not exceed 120% they did not observe GBD signs. Following 30 consecutive days of TDG levels exceeding 120%, 63% of all fish collected showed some GBD signs. They found the highest incidence of GBD in white crappie, smallmouth bass, and rainbow trout during periods when TDG exceeded 120%. They did not find GBD signs in any mottled sculpin, pumpkinseed, Chinook, kokanee, or steelhead.

Maule et al. (2003) evaluated the growth patterns of resident longnose suckers, northern pikeminnow, and walleye downstream from Grand Coulee Dam for years that included high TDG supersaturation in 1996 and 1997, followed by low TDG in 1998 and 1999. The TDG levels exceeded 120% for several months in 1996 and 1997, reaching 130-151% in 1997. Maule et al (2003) concluded that differences in age-at-length were not sufficiently large to suggest that TDG supersaturation had affected fish growth.

Resident non-salmonids in the lower Clark Fork River, Idaho were monitored (electrofishing, traps) from 1997 through 2000 during spring high flows that produced varying TDG supersaturation levels (Weitkamp et al. 2000a, Weitkamp et al. 2003b). In 1997 TDG levels were commonly above 130% for nearly two months, reaching peak levels near 150%. Although the sample size was not large in 1997 (103 fish) even with the exceptionally high TDG levels only 6 resident fish showed GBD signs, apparently due to the substantial river depths available. Another 7 fish showed hemorrhages or exophthalmia without visible bubbles that may have been signs of GBD. In 1998, GBD signs were detected in only one of the 1,671 non-salmonids examined, with TDG levels commonly reaching daily peaks of 115-125%. In 1999, GBD signs were detected in 10 of 15 species collected with a total incidence of 4.7%. Largescale sucker (11.4%) and yellow bullhead (14.3%) had the highest GBD rates. When TDG levels were 120-137% from late May to early July 1999 they observed GBD signs in 4.7% of the resident fish collected (n = 3,661), primarily in two sucker species, peamouth, and yellow bullhead. Weitkamp et al. (2003) reported GBD signs in only one largescale sucker from the lower Clark Fork River during 2000 when daily peak TDG levels (115-130%) occurred on many days with substantially lower levels occurring during most of each day. No dead or moribund fish were observed in the river during the four years of monitoring.

Fish were again collected from the lower Clark Fork River in 2006 by electrofishing the shallow shoreline areas to determine the incidence of GBD (Parametrix 2006). Only 29 (2.1%) of the 1,354 fish examined showed GBD signs. Fish were observed with GBD signs about one week after the peak TDG levels of the season (137%). Only one fish was observed with GBD signs after June 9, despite nearly continuous TDG levels of 118-126% through June 23. Bullheads had the highest incidence (38%) of GBD although only a small number were captured (32). None of the 328 trout had GBD signs except 5 of the brown trout (3.8%). One of 164 whitefish showed GBD signs. As in previous years with high TDG levels, those fish exhibiting signs of GBD generally had bubbles only in the dorsal or caudal fins.

Monitoring at Red Rock Dam, Iowa indicated TDG supersaturation (110-134%) occurred in three-fourths of the observations from 1983 to 1994 (Lutz 1985). Sublethal and lethal GBD was detected in live fish and from fish kills downstream from the dam where maximum water depths ranged from 0.6 m to 5.2 m over time. Fifteen fish kills were recorded with TDG levels averaging 121% during the kills, however the highest TDG levels did not result in fish mortality. The occurrence of periodic gas supersaturation-induced fish kills was linked to continued high dissolved gas pressures during periods when the discharge from the reservoir was substantially decreased at the dam resulting in decreased water depths downstream.

The cause of mortalities of fish in the River Nidelva, South Norway in 1978 was suspected to be TDG levels of 120-180% for a prolonged period of time, but no direct observations were reported for GBD incidence or severity (Heggberget 1984). Subsequently (1980) perch (*Perca fluviatilis*), eel (*Anguilla anguilla*), and brown trout (*Salmo trutta*) were exposed to 120-180% TDG in cages in the river and wild fish were observed. Only fish held near the surface in cages were killed while fish kept at 3 m depth were mildly affected, and few wild fish were killed.

### Invertebrates

In the Bighorn River downstream from the Yellowtail Afterbay Dam, Brammer (1991) monitored invertebrates for evidence of adverse affects of TDG supersaturation (up to 124%). This very shallow river has mid-channel depths of 1.8 m in the area of substantial TDG supersaturation. Brammer determined invertebrate sensitivity to high gas saturation levels was expressed through increased buoyancy which could cause involuntary drift. However, he found invertebrate community composition was similar between sites close to the dam (2.4 km downstream) and distant from the dam (14.5 km). Community structure was very similar between sites during April, but differed strongly during late summer and fall sampling periods. Effects of TDG supersaturation immediately downstream from the dam appeared to be displacement of some invertebrates due to increased buoyancy. However, invertebrate densities (32,658 invertebrates/m$^3$) were not low. Brammer concluded that if downstream displacement occurred,, upstream migration before oviposition was compensatory. White et al. (1991) concluded temperature differed between the two sites during the summer may have influenced the observed differences in invertebrate densities that were not present during the cooler spring.

In 1993, Toner and Dawley (1995) did not observe GBD signs in invertebrates (dragonfly larvae *Gomphus* sp., crayfish, and Asian clams *Corbicula* sp.) collected downstream from Bonneville Dam during 1993 when TDG levels reached a peak of 128%, but were above 120% for only 4 d. Toner et al. (1995) observed GBD signs only in a small portion of cladocerans collected downstream from Ice Harbor Dam in 1994 when TDG reached a peak of 136% and reached or exceeded 120% on most days in May, but average TDG levels remained below 120% during this period.

Schrank et al. (1997) found only one invertebrate species that showed GBD signs during 1995 in monitoring invertebrates potentially affected by high levels of TDG supersaturation in the Snake and Columbia Rivers. They observed 3.5% of cladocerans sampled downstream from Ice Harbor Dam, Snake River, had bubbles when TDG levels reached a peak of 138% and were commonly in the range of 115-125% for a prolonged period. Downstream from Bonneville Dam, GBD was observed only in 0.5% of cladocerans examined with TDG frequently 115-120% for three months. They reported that they found no substantive GBD signs or related mortality among invertebrates even in environments where fish suffered severely.

Ryan et al. (2002) collected invertebrates at several Columbia-Snake River sites weekly from depths of up to 0.6 m using a hydraulic epibenthic pump and Ponar dredge, or plankton net at the water surface. They detected GBD signs in only 7 of 5,424 invertebrates examined in 1994-1997 with TDG levels in the range of 120-135% for prolonged periods. Schrank et al. 1997 detected GBD signs in only 7 Cladocerans (0.5%) of the 5,434 invertebrates examined although the invertebrates were collected when TDG levels were within a range previously shown to produce GBD signs in a laboratory investigation.

Parametrix (2002) reported monitoring of invertebrates in shallow water downstream from Rocky Reach Dam, Columbia River, during periods of TDG supersaturation. With TDG levels up to 115% they observed GBD signs in only 2 of 7,405 invertebrates examined. When TDG levels reached 130% for a brief period, but remained below 120% most of the spill period, they found GBD signs in one bristle worm and one mayfly (*Hexagenia* sp.), comprising 0.02% of the total of 9,885 invertebrates examined.

## DEPTH DISTRIBUTIONS

The hydrostatic compensation of several meters water depth provides protection from bubble formation and GBD to migratory and resident fishes as well as invertebrates with the TDG levels generally encountered in field conditions. A number of investigations since 1980 have focused on the depth distributions of fishes during periods of TDG supersaturation to evaluate the actual exposure the fish receive.

## Juvenile Salmonids

There have been a number of investigations of the depth distributions of young anadromous salmonids in the Columbia River Basin. Generally these studies have found the young salmon occupy a range of depths and vary their depth over time, commonly occupying shallower depths during daylight hours and greater depths at night. Young salmonids occupy a range of depths that are sufficiently deep most of the time to avoid exposure to actual TDG supersaturation. The greater depths in these ranges commonly provide sufficient hydrostatic compensation to produce recovery from exposures during they encounter during the time they spend at near surface depths. This assumes that there is not a segment of the populations that remains in shallow water, which is supported by available information.

Maule et al. (1997) monitored the depth of migrating juvenile steelhead between Ice Harbor and McNary Dams (Snake-Columbia Rivers) using depth sensitive radio tags. Median depths of the young steelhead were between 1.1 m to 4.3 m with median TDG levels in the river of 120% to 126%.

The depth distribution of young salmon near both sides and the center of McNary Dam reservoir was investigated by Feil and Rondorf (2000). Depth distributions varied across the reservoir as well as between day and night, and with time during the migration season with anywhere from 3% to 40% above the compensation depth. Table 2 provides the percentages recorded above the TDG level compensation depth that varied from 2m to 2.5 m over time and location.

**Table 2. Percentages of juvenile salmonids above TDG compensation depths (2-2.5 m) in McNary Dam reservoir, May, 1997 (Feil and Rondorf 2000).**

| Season / Time | WA side | Center | OR side |
|---|---|---|---|
| May, early | | | |
| day | 23% | 33% | 18% |
| night | 14% | 20% | 9% |
| May, middle | | | |
| day | 18% | 39% | 30% |
| night | 18% | 23% | 25% |
| May, late | | | |
| day | 3% | 40% | 24% |
| night | 19% | 23% | 28% |

The depth distribution of juvenile Chinook salmon and steelhead was evaluated by Beeman et al. (2003) and Beeman and Maule (2006) by tagging the fish with pressure-sensing radio transmitters and monitoring their depths between Ice Harbor Dam tailrace (lower Snake River) and McNary Dam forebay. The mean depths of yearling Chinook generally increased with distance from Ice Harbor Dam, ranging from 1.5 m in the Snake River to 3.2 m near the forebay with maximum depths of about 10 m. The mean depths of juvenile steelhead ranged from 2.0 m in the Snake River to 2.3 m near the McNary Dam forebay with maximum depths of about 12 m. Chinook were deeper during the day than at night, while steelhead were deeper at night than during the day. Beeman and Maule (2006) concluded that hydrostatic compensation, along with short exposure times in the area of greatest TDG, reduced the effects of TDG exposure below those generally shown to elicit GBD signs or mortality. Steelhead downstream from Grand Coulee Dam in Lake Rufus Woods had median depths of 1.6 m with abrupt daily changes near sunrise and sunset (Beeman et al. 2003b).

## Adult Salmonids

During the spring and summer of 2000, 228 adult Chinook salmon were tagged at Bonneville Dam in the lower Columbia River with archival radio data storage transmitters that recorded depth and water temperature as the fish migrated upstream through the lower Columbia and Snake Rivers (Johnson et al. 2005). Swimming depths of 131 adult Chinook indicated they spent the majority of their time at depths greater than 2 m, which provide hydrostatic compensation for in-river TDG levels (Figure 7). The adult Chinook commonly spent only minutes at depths shallower than 2 m. The Chinook tended to migrate deeper in tailraces, where TDG levels are generally highest, than in reservoirs.

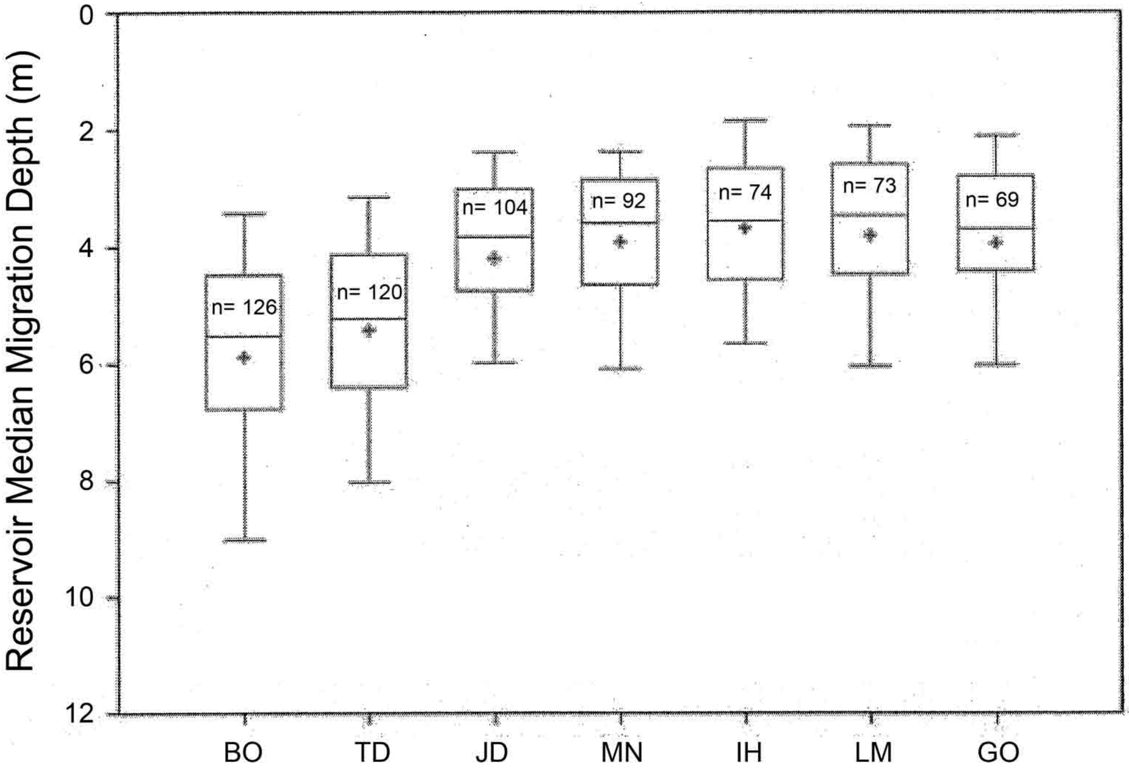

**Figure 7. Depth distribution of adult Chinook salmon in Columbia and Snake River reservoirs (mean = +, median = horizontal line in box, quartiles = boxes, 10-90 percentile = lines) (Dams: BO = Bonneville, TD = The Dalles, JD = John Day, MN = McNary, IH = Ice Harbor, LM = Lower Monumental, GO = Little Goose) (from Johnson et al. 2005.**

Weitkamp et al. (2000a, 2003a) collected resident salmonids and non-salmonids in the lower Clark Fork River by electrofishing and placed depth-sensing radio tags in healthy adults to evaluate their depth distributions. Individual fish were tracked during several years when high TDG occurred throughout the lower Clark Fork River (commonly 120-130%). The resident salmonids commonly spend most of their time near or below depths of about 2 m (Table 3). Evaluation of GBD in resident fishes during this period found few fish exhibited GBD signs unless TDG levels were near or exceeded 130% for prolonged periods, and then few fish show more than relatively minor GBD signs (Weitkamp et al. 2003b). Tagged rainbow trout tended to remain in the river for relatively brief periods before returning to Lake Pend Oreille where extreme depths are available.

**Table 3. Depth distributions (m) for tagged fish in the lower Clark Fork River (from Weitkamp et al. 2003a).**

| SPECIES | Average median depth [1] (m) | Maximum depth (m) | Average range (km) |
|---|---|---|---|
| rainbow trout | 1.8 | 6.1 | 6.1 |
| Brown trout | 2.6 | 8.7 | 6.6 |
| cutthroat trout | 1.7 | 10.4 | 7.1 |
| bull trout | 2.2 | 11.8 | 7.7 |
| mountain whitefish | 1.7 | 4.3 | 8.3 |
| northern pikeminnow | 3.3 | 15.4 | 3.5 |
| largescale sucker | 2.3 | 11.4 | 6.3 |

[1] Average of median depths for individual fish.

## Non-Salmonids

Gill net sampling of northern pikeminnow (*Ptychocheilus oregonensis*) in the Little Goose Dam tailrace showed that most occupied depths below 3 m and were therefore not exposed to true supersaturation at the levels present in the river (Bentley and Dawley 1981).

Beeman et al. (2003b) tracked the depth distributions of several resident fish species in Rufus Woods Lake downstream from Grand Coulee Dam using archival depth sensing tags. Median depths were northern pikeminnow-2.0 m, bridgelip sucker 2.8 m, walleye 3.7 m, longnose sucker 5.2 m, and largescale sucker 6.8 m. All fish showed abrupt depth changes near sunrise and sunset, with most fish deeper at night than during the day. The growth patterns of resident longnose suckers, northern pikeminnow, and walleye were examined by Maule et al. (2003) for years that included high TDG supersaturation in 1996 and 1997, followed by low TDG in 1998 and 1999. TDG levels had exceeded 120% for several months in 1996 and 1997, reaching 130-151% in 1997. They concluded that differences in age-at-length were not sufficiently large to suggest annual influences of TDG supersaturation on fish growth. Walleye showed a growth differential that was the opposite of what would be predicted based on the much higher TDG levels in 1996 than in 1998. These observations imply that the resident fish remained sufficiently deep to avoid the high TDG levels in 1996 and 1997.

Weitkamp et al. (2003a) evaluated the depth distribution of resident non-salmonids in the lower Clark Fork River by placing depth-sensing radio tags in healthy adults collected by electrofishing. Tracking during periods with high TDG levels commonly in the range of 120-130% showed the fish generally spend most of their time near or below depths of about 2 m (see Table 4). Most of the non-salmonid species did not show GBD signs during these periods unless TDG levels were near or exceeded 130%. Relatively minor GBD signs tended to be present in largescale sucker (11.4%) and yellow bullhead (14.3%) during periods of the higher TDG levels.

## OBSERVED GBD MORTALITIES IN FIELD CONDITIONS

Dead fish resulting from TDG supersaturation have only been reported by a few investigations. Although only a portion of dead fish are likely to be detected in field conditions there have been several mortalities of substantial number of fish related to TDG supersaturation that have been observed. The first and only substantial mortalities of adult salmonids due to GBD were recorded in the Columbia River system in the 1960s. No recent mortalities of adult salmonids due to GBD are recorded in the available literature.

The first substantial mortality of adult salmon and steelhead occurred in the Columbia River downstream from John Day Dam in 1968 (Beiningen and Ebel 1970). These conditions apparently resulted in substantial mortality due to GBD. Dead Chinook and sockeye salmon were observed downstream from the dam and adults with severe GBD signs were collected from the fishway. All water passed over the spillway at John Day Dam in 1968 because the turbines were not yet installed. The TDG levels were measured in the range of 123-143% during the mortality period. Mortalities of adult salmon were again reported in the Snake River in 1970 (Ebel 1971). Although TDG levels were 120-146% for a prolonged period, dead fish were not observed until July and most were too decomposed to identify GBD signs. The observed dead fish were not adequate to determine the effect on the population.

Beeman et al. (2003) report an unpublished report by Elston (1998) describes a mortality of 130,079 fish reared in net pens in the reservoir downstream from Grand Coulee Dam during 1997, and indicated that previous kills occurred in 1993 and 1996 due to GBD.

Dead and dying kokanee were observed during an exceptionally high spill event at Libby Dam on the Kootenai River in 2006 that produced GBD signs in substantial numbers of resident fishes (Marotz et al. 2007). In the late spring of 2006 substantial spill caused TDG levels to exceed 120% (123-131%) for nearly 20 days. During the 2006 spill, rainbow trout, westslope cutthroat trout, kokanee, bull trout, and mountain whitefish were collected by electrofishing in shallow water to determine the incidence of GBD. Although 20% of the collected fish had GBD signs after 4 days of TDG exceeding 120%, and 80-100% of each species showed GBD signs by the 11th day. It is not clear that GBD caused death of any fish under these field conditions. Dead and dying kokanee were observed, but these fish had apparently passed over the spillway because all had physical trauma (scrapes, lacerations, lost body parts, etc.). Population estimates before and after the 2006 spill event did not detect impacts to trout populations in the Kootenai River (Marotz et al. 2007).

The minor severity of GBD signs observed in most monitoring efforts provides an indication that TDG supersaturation is not resulting in substantial mortalities that affect fish populations. Mesa et al. (2000) determined that the severity of GBD signs in Chinook salmon and steelhead is highly correlated with mortality at TDG levels of about 130%. However, extremely high levels of TDG together with confinement in shallow water can result in mortalities without the development of externally visible GBD signs.

Colt (1984b) reported TDG levels in the lower Sacramento River were sufficiently higher than saturation (40-50 mm Hg) to be potentially lethal to striped bass larvae based on a high susceptibility of the larvae in laboratory tests using shallow water. However, no observations of larval mortality in field conditions were reported. In the American River Colt et al. (1991) reported a significant mortality of salmonids occurred during the major flood in 1986 with TDG levels of about 126-132% resulting from spill at Folsom Dam.

A high mortality of stocked rainbow trout was observed in the early spring by Mathias and Barica (1985) in ice-covered Canadian prairie lakes. Their investigation showed algal photosynthesis produced high oxygen levels, accompanied by rather high nitrogen levels that resulted in GBD. The elevated nitrogen levels attributed to the physical freeze-out of nitrogen from the ice in the shallow lakes resulted in substantial reduction of lake volume without loss of the dissolved nitrogen.

Crunkilton et al. (1980) reported major mortalities of resident fish downstream from Harry S. Truman Dam on the upper Osage River which they attributed to GBD. Several hundred thousand dead fish, primarily gizzard shad, green sunfish-bluegill, and freshwater drum, were observed downstream from the dam when TDG levels up to 139% occurred during April-June, 1978 and 1979. TDG supersaturation occurred throughout the 150 km of the downstream reservoir. Pelagic and near-shore species suffered the earliest and heaviest mortalities, but fish characteristic of deeper waters were increasingly killed as supersaturation persisted. Lutz (1995) reported a 15 fish kills (<50 to several thousand fish) attributed to GBD occurred downstream from Red Rock Dam, Iowa with TDG levels averaging 121%, but the highest TDG levels (134%) did not result in observed fish mortality. Fish kills occurred only at moderate discharges when the maximum river depths were about 1 m, less than when the highest TDG levels occurred at high discharge rates. The mortalities appeared to be produced due to the absence of compensating river depths.

## GENERAL POPULATION EFFECTS

There is essentially no literature that ties TDG supersaturation and GBD to population effects. In the Columbia River System there are numerous populations of anadromous salmonids that have been routinely exposed to TDG supersaturation of 120% and greater during both their juvenile downstream and adult upstream migrations. Although many of these populations have declined over the last 150 years, the available literature does not provide information linking their decline to TDG supersaturation exposure.

The growth patterns of resident rainbow trout in Rufus Woods Lake downstream from Grand Coulee Dam over a period that includes several years of high TDG supersaturation (1996, 1997) were examined by Maule et al. (2003). TDG levels had exceeded 120% for several months in 1996 and 1997, exceeding 130% in 1997, followed by low TDG in 1998 and 1999. They concluded that differences in age-at-length for rainbow trout were not sufficiently large to suggest annual influences of TDG supersaturation on fish growth. Beeman et al. (2003c) also described the growth of resident fishes in Rufus Woods Lake downstream from Grand Coulee Dam to determine if the years of high TDG corresponded to years of poor growth. They did not find a correlation of growth with TDG supersaturation despite the occurrence of very high TDG in 1996.

## GBD CHARACTERISTICS

A substantial amount of information has been developed that describes the basic characteristics of GBD. The following summarizes these observations.

## APPEARANCE / DISAPPEARANCE OF GBD

The initial pathology of GBD in rainbow trout begins with bubbles in retinal capillaries and progresses to unilateral exophthalmia when fish are exposed to 114-118% TDG in 12-cm deep rearing trays (Machado et al. 1987). They reported all moribund fish had gas displacing blood form the afferent arterioles within the gill filaments.

Fidler (1988) predict the TDG differential pressure ($\Delta P$) necessary to initiate bubble growth in the cardiovascular system or gill filaments of rainbow trout was 1.15 to 1.18 atms, which is about 115% to 118% TDG. Fidler (1988) developed a theoretical model for the thresholds for bubble formation and growth. His analysis indicates that a lower threshold occurs at TDG level of 1.1

atms and a higher threshold at 1.15 to 1.18 atms.

Antcliffe et al. (2002) found in laboratory experiments that rainbow trout did not show swim bladder over inflation or rupture as compared to control fish in all treatments (114% TDG, 10°C, 0.25 m; 118% TDG, 15°C, 0.25 m; 125% TDG, 18°C, 0.1 m). Only the most severe of these treatments resulted in mortality. All fish survived exposure to 114% TDG for six days, and 110% TGP for nine days. At 116% TGP, mortality was 42% after nine days. The $LT_{50}$ was 55 h at 122% and 5.1 h at 140% TDG exposure. In volitional cages 2.5 m deep they did not observe mortalities at 122% TDG, and interpreted this as evidence that the fish used depth to compensate for the effects of GBD.

In general, the available information indicates that bubbles (emboli) produced by TDG supersaturation disappear rather rapidly with either increased hydrostatic pressure or transfer to water that has little or no TDG supersaturation. Bubbles can disappear within minutes from the gills, some what slower from the later line (half hour), and much slower from extra-vascular tissues between fin rays (hours-days). Elston et al. (1979b) found gas bubbles in gills can be difficult to differentiate form similar appearing structures (amoeboid lipid globules). Gas emboli completely dissipated from excised gills in 2-15 minutes, and within about 30 minutes from intact gills removed from the water.

Hans et al. (1999) also examined the disappearance of bubbles from in the gill filaments, lateral lines, and external surfaces following exposure to TDG levels of 120-139% in shallow water. They found bubbles in the gill filaments reduced rapidly from 70% to 10% within 1 hr following transfer from the highly supersaturated water to water with a low level of supersaturation (104%). However, bubbles in the lateral line remained in 100% of exposed juveniles 4.5 hr after removal from the high level of supersaturation. The incidence of externally visible bubbles decreased slowly from about 95% of the fish at the end of exposure to high TDG to 40% of the fish at four days following removal from exposure. These test fish remained in shallow water at the end of their exposure, thus, they did not experience increased hydrostatic pressure that may have caused bubbles to disappear at a more rapid rate as fish in a natural environment might.

Knittel et al. (1980) described the recovery of young steelhead with GBD signs resulting from exposure to TDG supersaturation in shallow water (10, 50, and 100 cm) for near-lethal periods. These juveniles recovered completely in about 2 hr when lowered to a depth (3 m) providing hydrostatic compensation. Longer holding times at depth increased survival times during reexposure to high TDG at surface conditions.

Juvenile salmon with GBD signs were collected from Snake and Columbia River bypass systems and pressurized for 5 minutes to a 100 ft head (Montgomery Watson 1995). They found that the pressure exposure resulted in a significant reduction in GBD signs in the fins, lateral line, and gills. Their calculations indicate the reabsorption potential would be the same with 62 minutes at a head of 20 ft or 23 min. at 40 ft. They expressed concern that smolt monitoring might underestimate GBD prevalence due to the reabsorption potential, but did not consider the opposite effect of confining smolts in shallow water within bypass systems. Absolon et al. (1999) investigated the changes in GBD signs in migrating juvenile salmonids. Fish exposed to TDG supersaturation (115%) in shallow laboratory conditions were subsequently released upstream from a turbine intake and recovered 29 min. to 3 hr later from the turbine gatewell. Recaptured fish did not exhibit a statistically significant change in the severity or the prevalence of GBD. The results of these investigations indicate that GBD signs are likely to remain visible in external surfaces for days following removal from TDG supersaturation. Thus, it is likely that fish suffering from GBD would be detected by investigations that routinely sample fish from waters with substantial TDG supersaturation.

Elston et al. (1997a) investigated the rate of disappearance of GBD signs in young Chinook when they were exposed to increased pressure equivalent to a head of 30.5 m. Bubbles in the gills and the lateral line quickly disappeared, but bubbles were lost substantially slower from the fins. Bubbles in gills were found to dissipate in less than 10 minutes, and bubbles in the lateral line were nearly absent at 30 minutes. Bubbles in fins were reduced by 50% by 30 minutes and were nearly absent at 120 minutes. This investigation indicates GBD bubbles are rapidly reabsorbed from the vascular system, but some what slower from peripheral tissues when fish descend in a water column to a substantially increased hydrostatic pressure. This differential between internal and peripheral bubble reabsorption may explain in part why chronic exposures with fish moving up and down in the water column may result in grossly visible external GBD signs without producing detectable mortality.

## GBD SIGNS & MORTALITY

Mortality of fish due to TDG supersaturation appears to be poorly correlated with the externally visible GBD signs with exposure to very high or acute TDG levels, but may be correlated at moderate to high or chronic TDG levels (~120-130%). These conclusions are based on observations of fish exposed to TDG supersaturation in shallow (< 1 m) holding conditions.

In a laboratory investigation Mesa et al. (2000) found that the severity of GBD signs in Chinook salmon and steelhead is highly correlated with mortality at TDG levels of about 130%. However, extremely high levels of TDG together with confinement in shallow water may result in mortalities without the development of externally visible GBD signs.

VanderKooi et al. (2003) found GBD signs in resident fishes held in shallow water (26 cm) were greatest with chronic exposure (115% TDG) as compared to exposures to higher TDG level (125%, 135%). Mortalities did not occur at 115% TDG with up to four weeks exposure, but did occur with few GBD signs in hours to days at 125% and 135% TDG. This is another indication that with acute exposure internal bubbles may produce death prior to the development of peripheral GBD signs. The incidence of bubbles in the gill filaments and bubbles in fins did not correlate with mortality rates. Their results suggest that while bubbles in the gill filaments of resident fish may be related to mortality, they cannot be used to predict mortality risk to individuals. The inability to predict risk may be due to the rate at which internal lethal bubbles form during acute exposures. Ryan et al. (2000) also attempted to evaluate GBD-related mortality in non-salmonid fishes over the four years of net-pen holding experiments, but found a poor correlation of prevalence and severity of GBD signs with mortality.

Susceptibility to GBD may be affected by other stressful factors. Weiland et al. (1999) found juvenile Chinook with established bacterial kidney disease (*Renibacterium salmoninarum*) exposed to 120% TDG in a confined depth of 28 cm died earlier than fish without sign of BKD. The $LC_{20}$ for fish with BKD was about 37 h compared to fish without apparent BKD that survived for 96 h.

In live cage experiments Weitkamp et al. (2000b) found young rainbow and cutthroat trout held within 2 m of the surface in the lower Clark Fork River during exceptionally high TDG levels (+140%) all died within 4 d. Most fish surviving more then 2 d exhibited severe GBD signs, but many fish died during this acute exposure with minor or no visible GBD signs. Less than 20% of fish died during the 4 d exposure in a second test with TDG at 123-128% and GBD signs were observed in 0-28% of fish examined each day indicating the 2 m depth provided substantial compensation for the 123-128% TDG.

In their investigation of a pressure reduction to near zero (2 to 10 kPa) Becker et al. (2001) observed a 5% mortality of juvenile Chinook previously acclimated at 120% and 135% TDG and at a pressure of 191 kPa (~30 ft). Necropsies indicated massive gas bubbles in the heart (TDG 135%) and gas bubble blockage in the afferent lamellar arteries of the gills (TDG 120%) that blocked blood flow to the gills apparently caused the mortalities. This exposure represents acute conditions occurring with a sudden loss of compensating pressure producing internal bubbles that result in death without peripheral GBD signs.

## SPECIES VARIABILITY

Some investigations indicate some fish species are more susceptible to TDG supersaturation than other species, while other investigations have failed to find a difference among species. It is not clear from these reports that the observed differences in response to TDG supersaturation is due to inherent species differences or behavioral differences. Factors such as lateral line pore size and swim bladder structure may influence the appearance of external GBD signs and the affects of TDG supersaturation.

Nebeker et al. (1979) exposed juvenile Chinook, coho, and sockeye salmon, and steelhead to various TDG levels (115-120%) in shallow water at various temperatures (8-20 °C). They concluded increased temperatures caused a significant increase in mortality ($LC_{50}$) of Chinook and steelhead, but did not significantly affect coho and sockeye. Mesa et al. (2000) reported juvenile Chinook exposed to 130% TDG in a depth of 28 cm had $LT_{20}$ range of 3 to 6 h while the range for steelhead was 5 to 7 h. At 120% TDG, the Chinook $LT_{20}$ was 40 to 120 while the steelhead range was 20 to 35 h.

Fickeisen and Montgomery (1978) found mountain whitefish to be slightly more susceptible than cutthroat trout to TDG supersaturation when both were held in shallow (20 cm) cages at 124% and 128% TDG.

In exposures within laboratory pressure vessels Abernethy et al. (2001) found resistance to acute GBD from greatest to least, is bluegill > fall Chinook salmon > rainbow trout. Bluegills also had a lower incidence of chronic GBD signs than did fall Chinook salmon and rainbow trout. However, bluegills (a physoclistous fish), were extremely susceptible to swim bladder rupture when exposed to the sudden pressure change that represented turbine passage. In a following report on this research Becker et al. (2001) determined a pressure reduction to 2 to 10 kPa produced a 5% mortality of juvenile Chinook acclimated at 120% and 135% TDG and at a pressure of 191 kPa (~30 ft) within one hour, but young rainbow trout were not killed and showed no external signs of injury. However bluegill had a high morality and injury rate, apparently due to pressure changes rather than TDG. They concluded that species differences are related to differences in swim bladder structure.

Beeman et al. (2003c) found the times to 50% mortality ($LT_{50}$) in laboratory exposures showed variability among resident fish species. At 125% TDG the relative sensitivities of various species were northern pikeminnow ≥ largescale sucker > longnose sucker > redside shiver > walleye. At 130% TDG the $LC_{50}$'s were about half as long as at 125% TDG with species sensitivities similar except largescale sucker were more sensitive than northern pikeminnow. In similar laboratory tests Nebeker et al. (1980) determined speckled dace are more tolerant of TDG supersaturation in 25 cm depth than cutthroat trout, with a 2-week $LC_{50}$ of 129-131%, while the juvenile cutthroat

$LC_{50}$ was 115% and adult 118%.

Striped bass larvae have been identified to be more susceptible than most species to TDG supersaturation. In laboratory exposures (0.4 m deep) the striped bass larvae were extremely sensitive to TDG supersaturation with swim bladder over inflation and bubbles in their intestinal tracks at TDG levels of 103-106% (Cornacchia and Colt 1984). They observed that the sensitivity of the larvae to TDG supersaturation decreased with age, possibly as a result of behavioral changes.

## LIFE STAGE VARIABILITY

Salmonid embryos have been previously found to be resistant to TDG supersaturation (Weitkamp and Katz 1980). Alderdice and Jensen (1985) determined that this resistance is explained by the hydrostatic pressure within the egg capsule that is higher than atmospheric pressure. Internal pressures are at least 15 mm Hg in eggs, increasing to 50 mm Hg in fertilized embryos, and to as much as 90 mm Hg near hatching. The internal pressures of 50-90 mm Hg are equivalent to about 107-112% saturation at atmospheric pressure. Krise and Meade (1998) reported rearing of lake trout embryos and alevins at 111% TDG in water 15 cm deep did not affect survival, condition factor or mean weight.

Jensen (1980, 1988) describe exposure of steelhead embryos, alevins, and fry to TDG levels of 102%, 106% and 110% in Heath trays (depth <4 cm). Embryos survival was not affected at any TDG level tested. Alevin survival was marginally affected at 110% with a 1.4% incidence of opercular deformities within a few days due to bubble growth in the buccal cavity. A few fry (2.6%) exposed to 111% TDG developed GBD signs in the form of burst swim bladders. They reported fry size was not significantly correlated with TDG after 21 days of feeding.

The influence of seawater on anadromous salmonids was investigated by Bouck and King (1983) who transferred groups of steelhead smolts to seawater (29‰) for two weeks. Subsequently half the fish were exposed to supersaturation of about 125% (ΔP of 190 mm Hg) in the water (0.3 m deep) and half the fish were returned to fresh water. There was not a significant difference in the mean times to death of fish in the two treatments. Krise and Herman (1991) found subyearling Atlantic salmon could tolerate slightly high TDG levels than yearlings.

Most adult salmonids appear to be relatively resistant to TDG supersaturation based on field monitoring of upstream migrants and resident adults (Dawley 1986, Fryer 1995, Backman and Evans 2002). This likely is the result of behavior that commonly keeps the adults sufficiently deep to avoid GBD (Weitkamp et al. 2003a, Johnson et al. 2005)

Although Cornacchia and Colt (1984) found striped bass larvae were very sensitive to TDG (103-106%) in shallow water, the older larvae were lese sensitive. They concluded behavioral changes were likely responsible for the decreased sensitivity with increasing larval age.

Tadpoles (*Rana catesbeiana*) exposed to TDG supersaturated shallow water (0.25 m) produced gas inflation of the gastrointestinal tract (Colt et al. 1984). Affected tadpoles floated either with their left sides elevated or on their backs on the surface. These signs were reversed by reducing the dissolved gas levels. A 4-day exposure to a ΔP of 160 to 170 mm Hg (~122 % TDG) had no effect on survival during 30 days post-exposure observation. A 10-day exposure increased mortality and the levels of systemic *Aeromonas hydrophila* bacteria, redleg disease. Bacterial levels returned to control levels after 6 days of recovery. Colt et al. (1987) reported adult bullfrogs, *Rana catesbeiana*, exposed to a ΔP of 128 mm Hg (TDG 116.8%) for four days had no effect on bullfrog mortality, but produced subcutaneous gas bubbles in the webbing and on body surfaces. However, exposure to a ΔP of 250 mm Hg (TDG 132.9%) resulted in 40% mortality within one day resulting from accumulation of gas in their vascular systems. No GBD signs developed in adult bullfrogs held at a ΔP of 67 mm Hg (TDG 108.8%) for 27 days.

## BEHAVIORAL RESPONSE – SUPERSATURATION DETECTION

Detection and avoidance of TDG supersaturation has been implicated but not clearly resolved. In a round tank with accessible compartments providing various TDG levels (115, 125, and 145%) Stevens et al. (1980) found young Chinook, coho, sockeye, and resident rainbow avoided 125% and 145% TDG. However, steelhead did not avoid any TDG level, which they attributed to the species aggressive behavior. Lund and Heggberget (1985) reported rainbow trout held in 1.6 m deep tank at 115-125% TDG showed no difference in depth distribution as compared to control fish held in saturated water.

During spill events at Libby Dam on the Kootenai River in 2002, the TDG supersaturation tended to remain along the left bank (Dunnigan 2002). Radio tagged rainbow trout did not appear to avoid the area of TDG supersaturation. Antcliffe et al. (2002) also found in laboratory experiments that rainbow trout failed to show behavioral effects as compared to control fish in all treatments (114% TDG, 10°C, 0.25 m; 118% TDG, 15°C, 0.25 m; 125% TDG, 18°C, 0.1 m). However, in 9-2.5 m cages the young trout tended to school at depths greater than 1m.

Kokanee fry in 9 m deep live-cages at the edge of Lake Pend Oreille by Weitkamp et al. (2000b) during a period of average TDG levels >120%. Hydroacoustic monitoring indicated that the kokanee spent considerable time at depths greater than 1 m. However, during pre-dusk hours the fry were observed to slowly move closer to the surface where they remained for several hours before dispersing to depths of about 4 m. During the pre-dusk hours the fry were not avoiding the TDG supersaturated surface conditions

TDG supersaturation can produce higher pressures in a fish’s swim bladder that may result in behavioral changes. Shrimpton et al. (1990a) observed rainbow trout released gas through the pneumatic duct with greater pressures developing prior to release of the gas in larger fish. Those fish experiencing substantial changes in density resulting from increased swim bladder pressure compensated by increasing their depth within a 2 m water column Shrimpton et al. (1990b).

Exposure to high TDG supersaturation for the depth they occupy may affect the vulnerability of smaller fish to predation. Mesa and Warren (1997) found juvenile Chinook exposed to 130% TDG in a depth of 28 cm for 3.5 h showed a significant increase in vulnerability to predation by northern pikeminnow in laboratory tests. However, fish exposed to only 112% and 120% TDG for longer periods did not show increased vulnerability to predation.

Potential predators may also be affected by TDG supersaturation. The average daily prey consumed by northern pikeminnow decreased in proportion to increased TDG levels when they were exposed in shallow (0.25 m) laboratory tanks (Bentley and Dawley 1981). At 100% TDG saturation the pikeminnows consumed 14.2 g/d, at 117% consumption decreased to 6.2 g/d, and 2.3 g/d at 126%. VanderKooi et al. (2003) reported resident fishes (redside shiner, northern pikeminnow, largescale sucker, longnose sucker, and walleye) held in shallow water (26 cm) tended to have decreased activity and settle to or swim near the bottom as they developed GBD. The behavioral response of carp and black bullhead to TDG supersaturation was investigated by Gray et al. (1982) and Gray et al. (1983a). In water 30 cm-deep the fish only avoided supersaturation at the extreme levels (146% TDG).

Chamberlain et al. (1980) observed the behavior of Atlantic croaker (*Micropogon undulatus*) to be affected by TDG supersaturation. Initially the croakers moved upward within a 2.5 m-deep test tank in response to supersaturated water (145% TDG). The croakers subsequently moved down within the water column after 2-4 h through a series of up and down movements of unequally amplitude. They suggested that the fish's response was the result of changes in swim bladder volume.

## PREVIOUS EXPOSURE TO TDG SUPERSATURATION

There is some evidence that prior exposure to TDG supersaturation may affect susceptibility to subsequent exposure. Antcliffe et al. (2003b) examined the potential effects of initial exposure of rainbow trout to hydrostatic exposure of 2.5 m for 4 h prior to exposure to 122% TDG in water 0.25 m deep for 48 h. Survival of fish previously exposed to the higher hydrostatic pressure was not markedly greater than fish held in 0.25 m water prior to exposure to TDG supersaturation.

Knittel et al. (1980) described the recovery of young steelhead with GBD signs resulting from exposure to TDG supersaturation in shallow water (10, 50, and 100 cm) for near-lethal periods. These juveniles recovered completely in about 2 hr when lowered to a depth providing hydrostatic compensation. Longer holding times at depth increased survival times during reexposure to high TDG at surface conditions.

# TDG CONTROL

Controlling or reducing TDG supersaturation has been accomplished by a variety of methods depending on the water volume involved and its ultimate use. Hatchery water supplies are relatively small compared to rivers allowing various control measures not applicable to the large volumes of rivers. Hatchery water supplies also have more rigid TDG limits than rivers and lakes due to the extremely shallow conditions (0.1 - 1 m), and therefore limited hydrostatic compensation, under which young fish are typically reared. Controlling TDG supersaturation resulting from discharge of large rates of flow over dam spillways is more challenging and has greater potential environmental consequences.

## DAM SPILLWAYS

Discharge of river flow through dam spillways is the primary cause of TDG supersaturation reported in most literature dealing with field conditions. Most of the information available on spillway modification designs and other alternatives to control TDG and their effectiveness has not been published in generally available reports. Miller and Heaton (1994) describe TDG management measures and monitoring in the Lower Snake and Columbia Rivers. The U.S. Army Corps of Engineers, Walla Walla District, conducted the DGAS Phase II Study (2001) feasibility level analysis of the eight Corps projects on the lower Columbia and lower Snake Rivers to identify, evaluate, and recommend structural and or operational measures which will reduce TDG levels in the rivers. This report identified design criteria, identified alternatives, provided a feasibility evaluation of alternatives, and estimated costs for implementation of alternatives.

At Noxon Rapids Dam on the Kootenai River, Montana, Sullivan et al. (2004) found that discharging through gates over the central portion of the spillway could reduce TDG levels by 6-12% of saturation as compared to spill through the end gates equipped with flip bucket flow deflectors. The combination of greater air entrainment with the flip bucket design together with entrainment of air bubbles in the powerhouse discharge, resulted in the higher TDG levels when a gate close to the powerhouse was used. Downstream at Cabinet Gorge Dam discharging water was reduced by up to 13% of saturation by using spill gates that produced less entrainment of bubbles in the powerhouse discharge.

## TDG MONITORING

Monitoring of TDG supersaturation during the last 20-25 years has been conducted with standard commercial water quality monitoring devices that use semi-permeable membranes to measure TDG *in situ*. Earlier investigations generally used the van Slyke blood gas analyzer, Winkler titration, and/or gas chromatograph to measure dissolved gases in water samples. Bouck (1982) describes a forerunner of the commercially available semi-permeable membrane devices. D’Aoust and Clark discuss the various means of measuring dissolved gases that have been used in the earlier years (pre-1980) of TDG research.

Colt (1983) discusses the physics and terminology of TDG monitoring. Although he recommends reported TDG in terms of excess pressure (ΔP), the majority of reports dealing with biological effects continue to report TDG as a percent of saturation. Colt (1984) is a handbook that provides useful detailed information and relevant tables for the various factors involved in calculating and reporting TDG levels.

Tanner et al. (2002) reported that field checks of TDG sensors with a secondary standard showed eight Columbia River monitoring stations were recording TDG levels within 1% of saturation. They found measured barometric pressures and water temperatures were usually within 1 mm Hg and 0.05 ºC respectively of secondary standards.

Schneider and Barko (2006) found that breaking wind waves can greatly increase the rate of TDG supersaturation reduction in the Columbia River as spilled water travels downstream. They also reported temperature changes of 1.5 ºC can produce about a 3.5% change in TDG levels at spring temperatures near 15 ºC. This can be a natural source of increases in TDG levels when water becomes warmer as it flows slowly through reservoirs and slow moving rivers.

There are numerous gray literature reports providing monitoring data for many of the rivers in the Pacific Northwest as well as a few other locations.

## HATCHERY SUPPLIES

Monk et al. (1980) used small diameter siphons (5.1, 10.2, and 15.2 cm) to reduce dissolved gas. They concluded turbulence within the siphon was the primary factor producing a reduction in dissolved gas. Reduction of TDG supersaturation in hatchery water supplies was investigated by Bouck et al. (1984) using plunges, screens and columns packed with Glitch Ballast rings (1.58, 2.54 and 3.81 cm outside diameter), Tri-pac spheres (4-6 cm outside diameter), or Olin 12-gauge shotcups. They concluded that packed columns can generally produce biologically acceptable levels of dissolved gases ($\Delta P \leq 15$ mm Hg) when they are adequately designed, properly operated and regularly monitored. Ballast rings of 2.54 cm size proved more effective than the other sizes or the Tri-pac spheres or shotcups. Colt and Bouck ((1984) provide mass transfer model analysis for the packed columns, together with detailed information on the operational characteristics for degassing as a function of environmental and operating conditions. They concluded column heights in the range of 1-3 m are commonly needed to meet the criteria of $\Delta P = 20$ mm Hg and DO of $\geq 90\%$. Hargreaves and Tucker (1999) also describe packed column aerators as an effective means to reduce TDG supersaturation and raise low DO levels. They indicated the packed column should have 90% void or empty space per unit volume, and should cause the water flow to break up randomly into a thin film.

Colt and Westers (1982) discuss the potential production of TDG supersaturation in hatcheries using submerged aerators. They describe the factors involved in determining and calculating the degree of TDG supersaturation, primarily depth. Marking (1987) recommended considering oxygen injection systems for hatchery water supplies that may have a TDG supersaturation problem.

# NATURAL TDG SUPERSATURATION

It has long been recognized that TDG supersaturation can occur naturally as a result of water falls, rapids and ground water changes (Weitkamp and Katz 1980). A few recent reports have added to the records of naturally produced TDG supersaturation.

Bouck 1984 monitored TDG supersaturation in four spring-fed Oregon streams over a year. He measured strongly seasonal variation in TDG supersaturation with hyperbaric pressures ranging from a maximum ΔP of 40-70 mmHg in May-September to lows of 5-30 mmHg in winter. Each stream had the same trend in TDG levels, but hyperbaric pressures were substantially different among the streams. Seasonal temperature differences (6-11 ºC) contributed to the seasonal changes in TDG pressure in several of the streams.

A combination of naturally produced and man caused TDG supersaturation (air entrainment, solar heating, and photosynthesis) was reported in the American River, California during 1982-1983 and 1985-1986 by Colt et al. (1991). During February 1986, TDG levels reached 200-240 mm Hg (~ 126-130% TDG) apparently due to upstream air entrainment at Folsom Dam. Schisler and Bergersen (1999) also found photosynthesis by aquatic plants produced TDG supersaturation in the Colorado River.

The occurrence of naturally produced TDG supersaturation in aquaculture ponds was described by Boyd et al. (1994). Phytoplankton photosynthesis ($O_2$) together with solar heating of the water produced supersaturation with TDG (ΔP) during afternoon hours (1300 to 1500 h) increased TDG levels by a mean of 111 mm Hg (range -46 to 334 mm Hg) or to about 115% of saturation (94-144%).

Mathias and Barica (1985) reported TDG supersaturation produced by reduced liquid volume in a lake resulting from ice formation that confined the total volume of dissolved gas. Fish held in cages placed at near-surface depths, where high oxygen levels were measured, died showing GBD signs. They estimated dissolved nitrogen levels were near or greater than 200% of saturation and TDG >130%.

Recently TDG levels downstream from Niagara Falls (Figure 8) were monitored (Parametrix Inc. 2005). Recorded TDG levels averaged 126% between Niagara Falls and two downstream power stations (Canadian and U.S.). The discharge from the U.S. power station averaged 103%, reducing TDG levels in the lower Niagara River well below the natural TDG levels produced by Niagara Falls. The lower TDG levels in power station discharge, which were withdrawn upstream from the falls, reduced TDG levels in the lower Niagara River by 8-18% of saturation. Other locations such as Kettle Falls and Celilo Falls on the Columbia River likely produced high levels of TDG (110-130%), however these falls were submerged by reservoirs long before TDG monitoring was initiated. Figure 9 shows an early photo of Celilo Falls with obvious air entrainment downstream. Many other rivers have falls and major rapids followed by plunge pools that likely produce naturally high TDG levels.

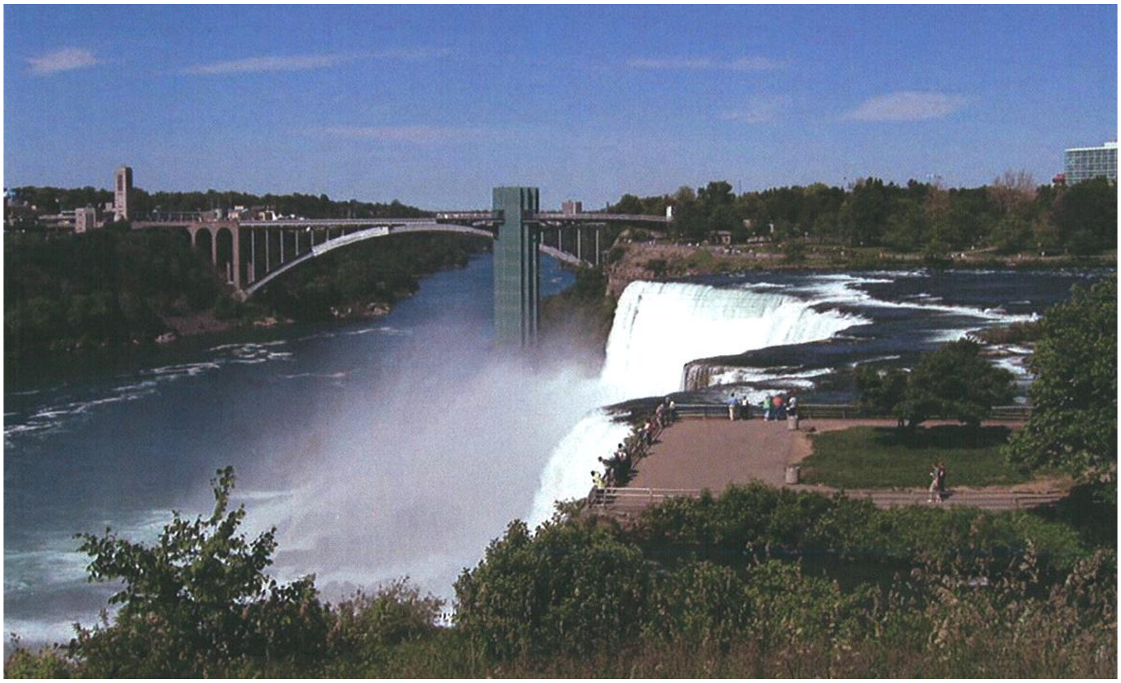

**Figure 8. Niagara Falls and lower Niagara River (by Gary Emond, TRC Solutions).**

**Figure 9. Celilo Falls, Columbia River circa 1890 (source: Salem Public Library).**

## HEATED EFFLUENTS

Water can become supersaturated by increasing its temperature without changing the amount of dissolved gas as a result of the decreased solubility of gases as the temperature increases. With substantial increases in temperature the TDG supersaturation can be sufficient to produce GBD. McInerny (1990) identified largemouth bass, white bass and bluegill inhabiting the heated discharge canal of Duke Power Company's Marshall Steam Station, Lake Norman, North Carolina, showed GBD signs. During February to May the levels of dissolved gases in the intake water were sufficient to produce supersaturation when heated. Peak TDG levels of 118-121% in the discharge water produced GBD signs in 12-28% of white bass, bluegill and largemouth bass collected from the discharge canal by electrofishing.

## MARINE CONDITIONS

Marine waters are potentially as susceptible to TDG supersaturation as fresh water, however there are apparently fewer actions by man that lead to recognized TDG supersaturation. However, there have been recorded instances resulting from thermal discharges (Weitkamp and Katz 1980) that have resulted in research on marine species. Gray et al. (1995) exposed post larvae and fingerling sea bass (*Dicentrarchus labrax*) and striped mullet (*Bugil cephalus*) to TDG supersaturation in aquaria 30 cm deep. They determined the mortality threshold to be 115% TDG with 90 h $LC_{50}$ values of 127% TDG for sea bass post larvae and 129% for striped mullet post larvae at 20° C.

Birtwell et al. (2001) exposed juvenile chum salmon to TDG supersaturation experimentally in marine water. They produced TDG supersaturation in heated seawater to reproduce conditions in a cooling water effluent. Juvenile chum were exposed in seawater at 115% for 48 h, 120% for 24 h, and 130% for 12 h exposures at 20.7 °C and then exposed to potential predation by Copper and quillback rockfish (*Sebastes caurinus, Sebastes maliger*), kelp greenling (*Hexagrammus decagrammus*), and Pacific staghorn sculpin (*Leptocottus armatus*). Test fish died only in the 130% treatment, in which all fish showed GBD signs. At both 120% and 130% TDG exposures they did not observe a significant difference in predation on treated and control fish in individual tests, but did detect significantly greater predation on treated fish in the pooled data.

The vertical responses of juvenile Atlantic croaker (*Micropogon undulatus*) to acute nitrogen and oxygen supersaturation with changing temperatures were observed in a 2.5-m-tall test cylinder by Chamberlain et al. (1980). Supersaturation of $N_2$ caused an initial upward movement of fish, although a compensatory downward response seemed to occur at 2-4 h of exposure. Dissolved oxygen supersaturation resulted in an almost immediate downward movement of fish. Similarities between the croaker’s behavior and the behavior of other physoclists after swimbladder volume manipulation suggested that gas supersaturation caused the swim bladders of test fish to inflate, resulting first in upward drift and then in downward swimming to restore neutral buoyancy.

Outbreaks of GBD in cultured saltwater tilapia (*Oreochromis spilurus*) and brackish water tilapia (*Oreochromis niloticus*), as well as groupers (*Epinephelus fuscogutiatus*) are described by Saeed and Al-Thobaiti (1997). The depths of holding conditions are not reported (apparently shallow water). They measured TDG levels of 111-113% during the outbreaks resulting in mortalities of 10-30% of the fish.

Gas bubble disease was observed in pink shrimp (*Penaeus brasiliensis, P. paulensis*) held in indoor tanks (depth not identified) supersaturated by air leaks allowing air to mix with the water under pressure of a pumped water supply (Brisson 1985). He reported DO levels were 9.5 mg/l at 21-22° C (~130% saturation) shortly following the incident, but TDG levels were not reported. The shrimp developed air-bubbles within their body cavities and fluids that produced convulsions. Other marine invertebrates reported to have suffered mortality include gastropods (*Bulla striata*, *Fissurella* sp.), bivalves (*Loevicardium* sp.), an echinoderm (*Lytechinus variegatus*), small barnacles, small crabs, and other shrimps.

Elston (1983) reported GBD signs in red abalone, *Haliotis rufescens*, exposed to water with supersaturated oxygen of about 150–200% in shallow culture conditions (0.4 L glass bowls) for 3-12 h. The abalone became lethargic with tissue swelling and loss of pigmentation within 3 h. The shellfish showed gas emboli through out their muscular and connective tissues and in the vascular system

# MODELING TDG SUPERSATURATION

Considerable effort has been devoted to modeling TDG produced by spillway discharge at hydroelectric facilities and to the affects of TDG supersaturation on fish.

## BIOLOGICAL MODELING

Using data derived from literature for Chinook, coho, sockeye and steelhead exposed to TDG supersaturation, generally in shallow water (<1 m), Jensen et al. (1986) examined 18 models that explore different factors influencing the effects of TDG supersaturation. For the shallow water conditions they concluded apparent safe levels for 50 day exposures are 104-115% TDG.

Ryan et al. (2000) used observations of GBD in large samples of non-salmonids and invertebrates collected from the Snake and Columbia Rivers along with observations in net-pen holding experiments to develop a model to predict the extent of GBD signs. They developed a mathematical equivalence model for TDG saturation duration and level of exposure that was strongly correlated with the prevalence of GBD signs ($r^2$=0.79). Signs of GBD were rare when TDG did not exceed 120% of saturation. Severity of GBD signs provided weak or variable relationships with the TDG data and was not used for the model. They concluded that when TDG exceeded 120% the model reliably predicted the extent to which fish displayed external GBD signs.

A numerical model of juvenile salmonid movements in the Columbia and Snake Rivers called Fish Individual-based Numerical Simulator (FINS) was developed by Scheibe and Richmond (2002). This model employs a discrete, particle-based approach to simulate the movements and history of exposure of individual fish to TDG. FINS is linked to a two-dimensional (vertically-averaged) hydrodynamic simulator that quantifies local water velocity, temperature, and dissolved gas levels as a function of river flow rates and dam operations. Simulated TDG exposure histories can be input to biological mortality models to predict the effects of various river conditions on fish injury and mortality due to TDG supersaturation.

## PHYSICAL MODELING

Weber et al. (1982) discuss the use of air bubble entrainment, numerical gas saturation, supersaturation and flow simulations for building 2D and 3D models of TDG production associated with dam spillways. Hibbs and Gulliver (1997) used a weighted average or effective bubble depth to predict gas transfer at spillways. They developed a theory for estimating the effective bubble depth given the spillway angle of inclination, velocity, and depth of flow at jet impact and tailwater depth. The physically based relationships of TDG supersaturation to dams were used by Geldert et al. (1998) to predict supersaturation downstream from spillways. They apply a predictive technique to the TDG supersaturation using conditions within the stilling basin and the river reaches immediately downstream from the structure. The model was calibrated using extensive field data from three spillways on the Columbia and Snake Rivers to fit coefficients that describe predictive relationships. Orlins and Gulliver (2000) provide further modifications and applications of this model.

Shaw (1998) provides new equations for the production of TDG from spill in the CRiSP.1.6 model previously developed by the U.S. Army Corps of Engineers Waterways Experiment Station. The equations are an empirical fit of spill data and monitoring data collected by the Corps.

Another modeling approach was taken by Politano et al. (2004). They predict TDG levels using a two-phase flow model that is based on a two-fluid model to calculate the gas volume fraction and velocity of the bubbles. They solve a transport equation for the TDG considering the mass exchange with the bubbles and assume one variable bubble size, which may change due to local mass transfer and pressure. Their simultaneous solution of a bubble number density equation provides prediction of the bubble size.

## PROMULGATION OF 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 available scientific information was derived primarily from laboratory investigations that restricted fish and invertebrates to shallow water (30 cm to 1 m total depth). These 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.

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

The literature reviewed in this paper is presented to provide a summary of information on gas bubble disease (GBD) and total dissolved gas (TDG) supersaturation that has become available since 1980. The information is provided to address the significance of GBD to populations of fish and invertebrates inhabiting waters where TDG occurs through anthropogenic processes. The information from the literature is provided to address the necessity of the 110% or a more appropriate criterion, based on field studies that have evaluated the actual effects of TDG supersaturation in the field where the criterion is applied.

Review of the substantial literature now available from field investigations makes it obvious that the GBD incidence and severity observed in shallow laboratory and cage conditions is not representative of field or river conditions. Fish behavior in field conditions, together with available water depths, commonly results in substantially lower exposure to supersaturation than occurs with laboratory conditions for a measured TDG level. The literature now substantiates that fish commonly experience TDG levels of 120% and higher in field conditions without experiencing GBD.

## HYDROSTATIC COMPENSATION

It has been obvious since GBD was first recognized that water pressure available at depths of several meters (hydrostatic compensation) reduces the level of TDG saturation substantially. This hydrostatic compensation has the potential to greatly reduce the incidence and severity of GBD in fish exposed to TDG supersaturation under field conditions as compared to those exposed in shallow water laboratory conditions, generally <1 m. The issue has been, does the available hydrostatic compensation actually avoid biological effects of TDG supersaturation in field conditions at TDG levels greater than 110% of saturation?

Hydrostatic compensation appears to be the primary factor that produces differences in laboratory results and field observations at various TDG levels. The available literature now contains a substantial variety of records of field monitoring that provide reliable information on the biological affects of TDG supersaturation in regulated waters.

## FIELD CONDITIONS

Under typical field conditions of free-flowing rivers, reservoirs, and lakes, fish have the opportunity to select the depth of their choice within the range of available depths. Commonly these depths are several meters or more where TDG supersaturation occurs, providing potential hydrostatic compensation that is not available in the confined depth conditions of most laboratory and cage investigations. It also appears that fish confined in cages that provide depths of several meters often do not have the same depth distributions as fish in open field conditions.

There is also a conservative factor in field observations of GBD. Fish observed under field conditions are commonly collected by means that are more likely to over estimate rather than under estimate the incidence and severity of GBD in the populations as a whole. Fish are commonly collected by electrofishing or beach seines that sample that portion of the population present in shallow water where they are most likely to develop GBD in TDG supersaturated waters. Juvenile salmon are also commonly collected from dam bypass systems that divert fish from substantial depths into collection and holding systems, which often have less than 1 m of depth. These diverted fish commonly spend hours in the shallow diversion and holding conditions prior to being examined.

Generally, waters where TDG supersaturation occurs in excess of 110% of saturation have substantial water depths. Although fish commonly occur in shallower streams and ponds, these shallower waters are generally not situations where man-caused TDG supersaturation occurs. Rivers, reservoirs and lakes where dam spillways produce substantial levels of TDG over large areas generally have depths of several meters or more.

Water depths available to fish commonly increase during periods of high TDG levels because these conditions are usually the result of high flow rates that result in spill at dams. The high flow rates raise river and reservoir levels increasing depths where fish and invertebrates are exposed to the TDG supersaturation. Simultaneously water velocities in rivers increase making it more likely resident fish will remain near the bottom where they encounter lower velocities.

During periods of high TDG levels the main flow path and shallow backwater areas do not necessarily have the same levels of supersaturation resulting in lower TDG in the shallower backwater areas. Schrank et al. (1997) reported they measured 109% TDG in backwaters when TDG levels were 131% in the swift main current. Thus, those fish most likely to be exposed in shallower water may experience lower TDG levels than those in deeper water areas.

## FISH MORTALITIES

In general low incidences and severities of GBD do not appear to produce detectable long-term effects to individual fish or to fish populations. It also appears that most individuals recover from low severity of GBD. Low severity of GBD signs generally indicates low severity of GBD with the exception of acute TDG conditions where some fish die without showing GBD signs or minor signs. The recorded incidences of substantial GBD mortalities in field conditions have occurred where TDG levels have substantially exceeded 120% TDG resulting in actual supersaturation at depths of 2 m or more or where fish were restricted by very shallow depths (~1 m).

The various field investigations of the biological effects of TDG supersaturation demonstrate that GBD and mortalities have been recognized in a number of instances, but that detectable mortalities are uncommon in river and reservoir conditions. The few investigations that have examined local populations have not detected an effect resulting from TDG supersaturation. It is clear that the depth compensation which substantially reduces the exposure of fish and invertebrates to actual TDG supersaturation results in greatly reduced incidences and severity of GBD in field environment conditions as compared to laboratory conditions.

## BEHAVIOR

In natural conditions (free flowing rivers, reservoirs, lakes, etc.) where fish are generally exposed to TDG supersaturation, the fish are rarely restrained in shallow water. In these field conditions the behavior of the fish relative to depth becomes and over riding factor in determining their actual exposure to TDG supersaturation and the resulting biological effects.

Evidence of the behavior of organisms exposed to TDG supersaturation is provided by two types of evidence. There are direct investigations of depth distribution, primarily through observation of fish implanted with depth sensitive tags that provide direct recordings of the depths occupied by the fish. There is also inferential evidence from the incidence of GBD recorded in sample populations of fish recovered following exposure to known TDG levels, such as the juvenile salmonids monitored at many Columbia River dams.

The available depth recordings of fish in field conditions show that most commonly occupy average depths of about 2 m or more, where these depths are available.

Johnson et al. (2005) documented that upstream migrating adult salmon remained deeper than 2 m the majority of the time, thereby avoiding exposure to supersaturation at TDG levels of 120% and less.

There is the potential that fish mortalities have been caused by TDG supersaturation that have not been observed. However, the available information indicates this risk is minor.

- Various investigations have actively sought mortalities without finding either direct or indirect evidence of dead fish attributable to GBD.
- Most of the fish with observed signs of GBD in natural/field conditions have a relatively minor degree (rank) of GBD, indicating they are not likely to suffer mortality.
- Fish obviously have the capacity to recover from GBD.
- Investigations of the rate of disappearance of GBD signs indicate that the externally visible signs should remain visible for several days in fish that have died, even those held in water with low TDG.
- There have been several situations where substantial mortalities attributed to TDG supersaturation have been recognized. These instances indicate that it is likely that substantial fish mortalities resulting from GBD will be recognized where they do occur.

Together these factors indicate that fish in field conditions are unlikely to suffer mortality or other identifiable long-term effects from TDG supersaturation of 120% or less. Population effects have not been detected where TDG levels have exceeded 120% for substantial periods of time.

Signs of GBD do not equate to mortality. Most fish exhibiting GBD signs in field conditions apparently recover, with some showing scars in their fins. However, at least with extremely high TDG level producing acute conditions with restraint in shallow depths, fish can die without showing externally visible GBD signs.

## TDG CRITERION

It has become obvious to regulators and dam operators that TDG supersaturation is challenging to control and limit within the existing criterion of 110%. Exemptions have been granted routinely in the Columbia River System to allow substantial levels of spill for fish passage. Efforts to reduce spill at dams and change spillways to reduce air entrainment have had limited success. The TDG conditions have substantially improved for most dams during the last 30 years by reducing the frequency, duration and amount of spill, except when required for fish passage. Many dams also have spillway modifications (flip lips) and operating procedures that limit the degree and duration of TDG levels exceeding 110%. However, there are still numerous situations where it is not possible or practical to meet the 110% criterion. Thus, it is appropriate to evaluate the necessity for meeting the 110% TDG criterion and the biological risks involved in not meeting the criterion.

There are a number of reports that question the need for a TDG criterion lower than 120% of saturation. Mesa and Maule (1997) concluded it appeared GBD was not a threat to migrating juvenile salmonids when TDG levels do not exceed 120%. McGrath et al. (2006) concluded short-term exposure of up to 120% TDG does not produce significant effects to migratory juvenile or adult salmonids when compensating water depths are available. However, they raised concern for several issues due to limited available information: 1) sensitive and vulnerable species or life stages, 2) long-term chronic or multiple exposure, 3) vulnerable habitats and reaches, 4) incubating fish in hyporheic habitats, and 5) community and ecosystem impacts. Each of these issues is addressed to some degree by the literature discussed in earlier sections of this review.

Schneider (2000) provides an assessment by the National Marine Fisheries Service (NMFS) of the 120% TDG criterion for the Columbia and Snake Rivers during juvenile salmon migration periods. The NMFS assessment concluded that the risk associated with a managed spill program to the 120% TDG level is warranted by the projected 4-6% increase in survival of juvenile salmonids. They found recent research and biological monitoring results predict that TDG in the 120% to 125% range, together with the vertical distribution of fish (most fish migrate at depths providing some gas compensation) would not cause juvenile or adult salmon mortalities exceeding the expected benefits of spillway passage.

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# FRESHWATER FISH SPECIES INCLUDED

| common name | Scientific Name |
|---|---|
| **white sturgeon** | *Acipenser transmontanus* |
| **gizzard shad** | ***Dorosoma cepedianum*** |
| **Chinook salmon** | ***Oncorhynchus tshawytscha*** |
| **coho salmon** | ***Oncorhynchus kisutch*** |
| **chum salmon** | ***Oncorhynchus keta*** |
| **steelhead (anadromous)** | ***Oncorhynchus mykiss*** |
| **rainbow trout** | ***Oncorhynchus mykiss*** |
| **sockeye salmon** | ***Oncorhynchus nerka*** |
| **kokanee** | ***Oncorhynchus nerka*** |
| **cutthroat trout** | ***Oncorhynchus clarki*** |
| **westslope cutthroat trout** | ***Oncorhynchus clarki lewisi*** |
| **brown trout** | ***Salmo trutta*** |
| **bull trout** | ***Salvelinus confluentus*** |
| **lake trout** | ***Salvelinus namaycush*** |
| **mountain whitefish** | ***Prosopium williamsoni*** |
| | |
| **carp** | *Cyprinus carpio* |
| **northern pikeminnow** | ***Ptychocheilus oregonensis*** |
| **chiselmouth** | ***Acrocheilus alutaceus*** |
| **peamouth chub** | ***Mylocheilus caurinus*** |
| **redside shiner** | ***Richardsonius balteatus*** |
| **tench** | ***Tinca tinca*** |
| **longnose sucker** | ***Catostomus catostomus*** |
| **largescale sucker** | ***Catostomus macrocheilus*** |
| **bridgelip sucker** | ***Catostomus columbianus*** |
| **yellow bullhead** | ***Ameiurus natalis*** |
| **black bullhead** | ***Ameiurus melas*** |
| | |
| **three-spine stickleback** | ***Gasterosteus aculeatus*** |
| **sand roller** | *Percopsis transmontama* |
| **striped bass** | ***Morone saxatilis*** |
| **white bass** | ***Morone chrysops*** |
| **smallmouth bass** | ***Micropterus dolomieu*** |
| **largemouth bass** | ***Micropterus salmoides*** |
| **pumpkinseed** | *Lepomis gibbosus*) |
| **bluegill** | ***Lepomis macrochirus*** |
| **white crappie** | ***Pomoxis annularis*** |
| **crappie** | *Pomozis* spp. |
| **yellow perch** | ***Perca flavescens*** |
| **perch** | ***Perca*** *fluviatilis* |
| **walleye** | ***Stizostedion vitreum*** |
| | |
| **freshwater drum** | ***Aplodinotus grunniens*** |
| **mottled sculpin** | *Cottus bairdi* |
| **sand roller** | *Percopsis transmontana* |