



**TGP Performance Measures for the Columbia River  
Water Use Planning Process  
A Review and Evaluation of Relevant Information and Data**

**Final**

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# **TGP Performance Measures for the Columbia River Water Use Planning Process A Review and Evaluation of Relevant Information and Data**

## **1.0: Introduction**

Water Use Planning was initiated by the Minister of Employment and Investment (MEI), the Minister of Environment, Lands, and Parks (MELP)<sup>1</sup>, and BC Hydro in 1998 as a public consultation approach to optimize water allocation and use associated with BC Hydro's facilities across a range of public, provincial, and federal issues. A Water Use Plan (WUP) is a technical document that, once reviewed by provincial and federal agencies and approved by the provincial Comptroller of Water Rights, defines how water control facilities will be operated. These operational constraints will reflect the multi-stakeholder decisions made to reflect power, environmental, recreation, and specific First Nations issues. One facet of the WUP develops performance measures to examine the merits of different hypothetical operational alternatives relative to each other and/or the current operations. Performance measures are developed for a broad range of interests and issues including, but not excluded to power, environmental, and recreation. Performance measure development and subsequent application to assess proposed operational alternatives are based on existing dam/powerplant operational and environmental constraints, data from the literature, and novel data collected specifically for performance measure development from the watershed involved. In most cases, operational alternatives are compared using performance measure results generated from simulated operations based on a common set of a continuous time series of historical inflows.

One of the WUP processes currently underway involves the Columbia River from the Mica Dam downstream to the Canada/US border. In terms of biological considerations, a major concern is the effect of dissolved gas supersaturation (DGS) produced by the Hugh Keenleyside Dam (HLK) on fish populations in the river below the dam. In general, Total Gas Pressures (TGPs, a measure of DGS – see Colt, 1983 and Fidler and Miller, 1997)<sup>2</sup> below the Hugh Keenleyside Dam often exceed the 1997 provincial and federal guidelines at various times of the year (Fidler and Miller 1997, Maxwell 1985, Hildebrand 1991, Aspen Applied Sciences Ltd. 1995 and 1998). A goal of the WUP process is to develop a performance measure that relates the operational alternative of the Hugh Keenleyside Dam to river TGP levels and associated biological impacts. However, the development of TGP performance measures involves a number of complicating factors. Specifically, TGP in this section of the Columbia River is influenced not only by the operations of the Hugh Keenleyside Dam, but also by operations of the Arrow Lakes Generating Station, the Brilliant Dam on the Kootenay River, other dams higher up on the Kootenay River, the Waneta Dam on the Pend Oreille River, and Pend Oreille River dams in the United States (Aspen Applied Sciences Ltd. 1995, 1998, 2001). In addition, Arrow Lakes Reservoir, upstream of the Hugh Keenleyside Dam may also influence TGP in the Hugh Keenleyside Dam discharge (Aspen Applied Sciences Ltd. 1995 and 1998).

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<sup>1</sup> MELP and its associated responsibilities has subsequently (2002) been reorganized into the Ministry of Water, Land, and Air Protection (MWLAP), Ministry of Sustainable Resource Management (MSRM), and the Crown Corporation Water and Land.

<sup>2</sup> For an explanation of the biological and physical principles of TGP and GBT along with terminology, see Appendix 1, Antcliffe, B.L., L.E. Fidler, and I.K. Birtwell. 2002a. Effect of dissolved gas supersaturation on the survival and condition of juvenile rainbow trout (*Oncorhynchus mykiss*) under static and dynamic exposure scenarios. *Can. Tech. Rep. Fish. Aquat. Sci.* 2370: 70 p.

To address these complications, a meeting was held at the B.C. Hydro offices in Castlegar B.C. on June 26, 2002. The meeting was attended by B.C. Hydro personnel, a representative from the B.C. Ministry of Water, Land and Air Protection (MWLAP), a representative from Canadian Columbia River Intertribal Fish Commission (CCRITFC), and consultants from Golder Associates and Aspen Applied Sciences Ltd. Given the complexity and unpredictability of various dam operations on the Columbia, Kootenay, and Pend Oreille Rivers along with unknown river flow patterns, it was decided to limit the development of TGP performance measures to the section of the Columbia River immediately below the Hugh Keenleyside Dam. Furthermore, it was agreed that the performance measures should be developed based on threshold TGPs and daily exceedance criteria relative to TGPs above the threshold (see Aspen Applied Sciences Ltd. 1998 and 2001 for explanations and examples).

Subsequent to the June 26<sup>th</sup> meeting, further discussions of TGP performance measures established that they should not only reflect TGP levels, but should also indicate the risk of GBT to fish in the Columbia River. Specifically, there should be GBT risk factors associated with the TGP levels produced by each operational configuration of the Keenleyside Dam. The importance in incorporating GBT risk factors into the performance measures lies in the non-linear relationship between risk of GBT and TGP. Specifically, as TGP rises from a threshold level (e.g., 115% for rainbow trout), the risk of GBT in fish (e.g., based on time to specific levels of mortality) rises rapidly in an inverse hyperbolic manner (Fidler and Miller 1997). Using this approach, the performance measures would then quantify the days of continuous exceedance of GBT risk factors associated with specific operational alternatives for the Keenleyside Dam. Although the days of continuous exceedance of risk factors is a new and more accurate approach to evaluating the performance of hydroelectric dams, the role of a threshold TGP remains central.

Ideally, the threshold TGP would be based on a Lowest Observed Effect Level (LOEL). That is, there would be no risk of GBT to fish below the LOEL threshold. Thus, all dam operations that produce TGPs below the threshold should be excluded from any comparative analysis based on TGP performance measures and their associated GBT risk factors. To include the range below the LOEL might distort the results of the analysis, once the performance measures were integrated over a yearly hydrograph.

One of the difficulties identified at the June 26<sup>th</sup> meeting was the uncertainty associated with establishing threshold TGPs. Specifically, should they be based on existing guideline values, literature data, or on observational information obtained directly from the Columbia River? The uncertainty in this issue was based, in part, on the fact that the existing guidelines for TGP were developed from laboratory experimental data and not field data; whereas, other information (Hildebrand 1991, Antcliffe et al. 2002a, b, and c) suggest that the guideline values may be overly conservative for some river applications. To resolve these uncertainties, it was agreed that the available information on TGP and gas bubble trauma (GBT) in fish should be re-examined in the context of identifying threshold TGPs specifically for the Columbia River below the Hugh Keenleyside Dam. This examination would include a review of the existing TGP guidelines, data from the literature upon which the guidelines were based, biological field data from the Columbia River, data on Columbia River TGPs, and more recent DFO Canada studies that address the issue of applying laboratory biological data to river conditions. The following summarizes the results of the review, recommends threshold TGPs to be used in developing Columbia River WUP performance measures, and suggests additional work that can augment and support the development of

performance measures. The review results are broken into four sections. The first section focuses on the B.C. Guidelines for DGS and contains the following sub-sections.

1. A review of the current B.C. guideline for DGS with assessment.
2. A review of recent DFO studies of DGS and GBT in fish (Antcliffe et al. 2002a, b, and c) with assessment
3. A re-examination of data from the literature in relation to the B.C. guideline and the DFO studies.
4. Summary

The second section examines GBT field data collected from the Columbia River with the following sub-sections.

1. A review of studies examining GBT effects in fish of the Columbia River with assessment.
2. A review of studies examining fish depth behaviour in the Columbia River with assessment.
3. A review of data sources for TGP levels in the Columbia River with assessment.
4. Summary

The third section contains an overall summary and recommendations for interim threshold TGPs for WUP performance measures while the fourth section provides recommendations for supporting field studies.

## 2.0 B.C. Guideline for DGS and Related Laboratory Studies

### 2.1. The B.C. Guideline for DGS

In 1997, the Province of British Columbia, in cooperation with DFO Canada and Environment Canada, developed water quality guidelines for dissolved gas supersaturation in Canadian waters (Fidler and Miller 1997). The guideline consisted of four parts (A, B, C, and D) to protect aquatic and marine organism in specific water environments. The four parts are:

- A. For Water Depths Greater than One Metre: Where local water depth at a given location in a water body exceeds one metre, the maximum  $\Delta P$  should not exceed 76 mmHg regardless of water  $pO_2$  levels. For sea level conditions, this corresponds to a TGP% of  $\approx 110\%$ .
- B. For Water Depths Less than One Metre: Where local water depth at a given location in a water body is less than one metre, the guideline should be based on Equation 4 which describes the threshold for swim bladder overinflation as a function of water depth and  $pO_2$  levels. However, the maximum  $\Delta P$  should not exceed 76 mmHg regardless of  $pO_2$  level.

$$\Delta P_{SB} = 73.89 \cdot h + 0.15 \cdot pO_2 \quad \text{Eq. 4}$$

where  $\Delta P_{SB}$  = water  $\Delta P$  required to initiate overinflation of the swim bladder in rainbow trout.

h = water depth at which the fish is located - metres.

$pO_2$  = partial pressure of dissolved oxygen (mmHg) in the environmental water.

The most conservative application of the guideline will be to use Equation 4 with  $h = 0$ . For example, at a water depth of zero metres and a  $pO_2$  of 157 mmHg, the  $\Delta P$  must not exceed 24 mmHg. This corresponds to a TGP% of  $\approx 103\%$  at sea level. This would apply to shallow water bodies and for stream margins, where the entire area less than one metre depth is used by juvenile fish.

- C. For Natural Background Levels Higher than the Recommended Guideline: If natural background levels of DGS exceed the recommended guidelines, there should be no increase in the  $\Delta P$  or %TGP over the background levels. This recognizes that background levels which are higher than the recommended guidelines may be harmful to fish, and hence, any increase over background levels should not be tolerated for the protection of aquatic life.
- D. For Hatchery Environments: It is recommended that the DGS guideline for hatcheries be defined by Equation 4 with  $h = 0$ . This corresponds to a sea level TGP% of 103%. This guideline recognizes that fish in hatcheries may experience more stress due to higher densities and declining  $pO_2$  levels along the rearing facility. Also, fish feed near the surface and are held in shallow water containers. This guideline also allows for higher DGS in systems using oxygen supplementation. For example, using Equation 4, if  $pO_2$  is 250 mmHg (164% of saturation), then the maximum allowable DGS is 38 mmHg (TGP is 105%).

In terms of the Columbia River, guidelines A and B are the most relevant. Guideline A is the primary guideline, which limits the maximum sea level TGP to 110% saturation regardless of water depth. However, the more stringent guideline is part B that applies to organisms in water less than 1 m deep. Guideline B specifies a maximum sea level TGP that varies linearly from 103% to 110% for water depths ranging from 0 m to 1.0 m, respectively. This guideline was developed specifically to protect juvenile fish in shallow water environments from the effects of swimbladder overinflation, a sign of GBT (Fidler 1988, Shrimpton et al. 1990a and b, Fidler and Miller 1997).

## Assessment

In the development of the guidelines, it was recognized that the supporting data were developed in laboratories where fish were restrained in their movements and confined to shallow water environments. This was especially true for the data of guideline B where some of the laboratory data involved anesthetized and restrained fish. At the time, questions were raised as to how appropriate these data were when applying the guidelines to rivers and lakes where fish were unrestrained and often had significant water depths available. These questions became even more relevant when the province of British Columbia initiated the WUP process. If the guidelines were overly conservative, especially in relation to guideline B and swim bladder overinflation, they could severely impact the assessment of proposed operational alternatives.

Another consideration in applying the guidelines was the effects of dynamic exposure of fish to DGS. It was recognized that in rivers and lakes, fish utilize a range of habitats involving wide variations in water depths over time. With the corresponding variations in depth compensation (see footnote 1), the exposure of fish to DGS would be dynamic, both spatially and temporally. Specifically, there could be periods of GBT bubble growth followed by periods of bubble collapse. It was conceivable that, depending on fish depth behaviour, the harmful effects of GBT could be avoided, even under relatively high levels of DGS. A further consideration was that fish using depths much greater than

the compensation depth might acquire some added resistance to GBT (Aspen Applied Sciences Inc. 1998b).

These concerns led the DFO Canada to initiate a series of laboratory studies to determine if free-swimming fish actually displayed signs of DGS induced swim bladder overinflation, whether dynamic exposures delayed the onset of GBT and reduced the severity, and whether exposure to hydrostatic pressure influenced susceptibility to GBT. These experiments, which were conducted at the DFO Rosewall Creek Hatchery in 2000 and 2001, are described in Section 2.2 that follows.

It should be noted, that although parts A and B of the B.C. guideline focus on water  $\Delta$ Ps of 76 mmHg or less, additional discussion in the guideline document describes the threshold for cardiovascular bubble growth in fish, the most widely recognized cause of direct acute mortality from GBT. This threshold occurs at sea level TGPs ranging from 115% to 119%, depending on species (e.g.,  $\approx$ 115% for rainbow trout and  $\approx$ 119% for chinook salmon). This threshold will be discussed in subsequent sections.

## **2.2 DFO Canada Rosewall Creek GBT Experiments**

From January 2000 to May 2001, DFO Canada conducted a series of laboratory bioassays that examined the effects of DGS and GBT on fish. The research was supported by the province of British Columbia under the Water Use Planning Data Acquisition Fund. The studies were conducted in three phases with the results published in three DFO Canada reports. The primary purpose of these studies was to clarify certain aspects of the B.C. and Canadian water quality guidelines that apply to DGS in Canadian rivers and lakes. A brief summary of work performed in these bioassay studies follows.

**Phase I Studies:** In the first study (Antcliffe, B.L., L.E. Fidler, and I.K. Birtwell. 2002a), the effect of DGS on the survival and condition of juvenile rainbow trout under static (steady-state) and dynamic exposure scenarios was assessed in laboratory experiments between January and March 2000. Static exposures at 110%, 114%, 116%, 122%, and 140% TGP (at 0.25 m water depth and 10 °C) demonstrated an inverse dose-response relation between TGP and duration of exposure required to kill fish. The LT50 (time to 50% mortality) was 5.1 h for the 140% TGP exposure, compared to 55 h for the 122% TGP exposure. Cumulative mortality was 42% after a 9 day exposure to 116% TGP. All fish survived exposure to 114% TGP for 6 days, and 110% TGP for 9 days. Results are consistent with the bubble growth threshold equations in Fidler (1988), that predict the water TGP required to initiate bubble growth in the cardiovascular system or gill filaments of rainbow trout (115% TGP at sea level). Comparison of these results with others from the literature suggests that there are differences among stocks in their susceptibility to DGS. The high variability in time to mortality among the 180 fish exposed to 122% TGP revealed that large sample sizes are required to obtain a representative dose-response relation for any given fish stock.

Dynamic exposures examined the effects of fish use of variable water depths on the dose-response relation for DGS-induced mortality. At 122% TGP and 10 °C, fish were allowed volitional access to water depths ranging from 0 to 1 m, and from 0 to 2.5 m. Because fish behaviour in a laboratory deep tank may not be representative of that in the wild, other dynamic exposures held fish in cages, and the cages were cycled throughout the deep tank (0 to 2.5 m) according to a pre-determined duration/depth cycle. Precise information on the

behaviour of juvenile rainbow trout in the wild was not available at the time of this study to design the depth cycles based on known periods of time spent above or below the TGP compensation depth, the actual depths used, and the time spent at those depths. Therefore, fish were held in cages at the surface of the deep tank (in up to 0.25 m depth) where they were exposed to 122% TGP and 10 °C for a duration that killed 10% of the sample size. The cages were then lowered and held below the compensation depth for 3 h, before being returned to the surface. This depth cycle was repeated 4 times, such that cumulative mortality was from 0 to 10% during the first surface interval, and from 10% to 20%, 20% to 30%, and 30% to 40% during the second, third, and fourth surface intervals, respectively. Control fish were cycled to depth at the same time as exposed fish. Similar exposures occurred at 122% TGP and 124% TGP, with intervals at depth of 6 h. Results for dynamic and static exposures (which differed only in terms of fish use of water depth) were compared.

Fish volitional use of water depth significantly delayed the onset of mortality, and cumulative mortality over the exposure period. In the 0 to 1 m volitional exposure, the time to initiation of mortality was about 36 h, compared to 14 h for the static exposure at 0.25 m water depth, for 122% TGP and 10°C. Cumulative mortality was 22% in the volitional exposure, compared to 89% for the static exposure, over 96 h. Cyclical use of water depth, which represented possible diel behaviour in salmonids, also delayed the re-initiation of mortality when fish were cycled from below the compensation depth to the water surface, and in some cases, reduced the rate of mortality at the water surface (once mortality was re-initiated). Overall, the fish use of volitional or cyclical water depth had a significant and positive effect on the survival of the fish. Although the dynamic exposures were conducted over a relatively short period (from 96 h to 7 days), sustained or periodic use of water depth by fish over longer exposures should also provide benefits to survival.

The absence of mortality at 122% TGP with volitional water depth from 0 to 2.5 m provided evidence that small rainbow trout use water depth to compensate for the effects of GBT. However, other laboratory deep tank studies from the literature found that fish were unable (or unwilling) to use available water depth to compensate for exposure to elevated TGP. Notwithstanding this variability, the wide range of behavioural patterns exhibited by individual fish in natural environments will likely produce very different exposure histories, hence variability in biological responses among fish.

Results showed that roughly half of the fish that died from exposure to TGP greater than 120% had no external signs of GBT. Further, many of the survivors had no external signs of GBT. These findings may limit the use of external GBT signs monitoring programs in relation to assessing the exposure and biological effects of DGS.

Phase II Studies: In the second phase experiments ( Antcliff, B.L., L.E. Fidler, and I.K. Birtwell. 2002b), a series of laboratory experiments were conducted to determine the ecological consequences of swimbladder overinflation in juvenile rainbow trout and coho salmon (i.e., < 60 mm fork length) that had been exposed to various levels of dissolved gas supersaturation. The experimental design initially called for a predation challenge experiment to measure the effects of swimbladder overinflation between treatment fish and control fish. However, poor predator performance prevented those experiments from being conclusive. A time to seek cover challenge was substituted as a metric for measuring response to swimbladder overinflation. This challenge involved releasing treatment and control fish in a well-lit

environment a fixed distance from a darkened area of cover. The time to seek cover and the paths used were recorded. The experiments demonstrated that for shallow water exposures (0.1 – 0.25 m) and sea level TGPs ranging from 114% (7 day exposure) to 125% (36 hour), no effects of swimbladder overinflation could be detected. Essentially, treatment fish and control fish took about the same time to seek cover. Throughout both the predation and time to seek cover experiments, both treatment and control fish were closely observed to determine if there were any behavioural signs of swimbladder overinflation evident. Without exception, no behavioural differences could be observed between treatment and control fish, regardless of fish size. There was no evidence of venting the swimbladder or swimbladder ruptures. Nor were there differences in depth distribution, swimming orientation (i.e., head down), or swimming behaviour present. Nor was there evidence of bubble formation in the buccal cavity as reported by Jensen (1980 and 1988).

Phase III Studies: These experiments, which were conducted in May, 2001 (Antcliffe, B.L., L.E. Fidler, and I.K. Birtwell. 2002c), examined whether prior exposure to hydrostatic pressure (depth), imparted any resistance to the development of cardiovascular bubbles in fish that were subsequently exposed to DGS in shallow water. The hypothesis of the experiments was that elevated hydrostatic pressure could collapse nucleation sites from which bubble growth originates (see Fidler 1988 for discussion of nucleation sites and Aspen Applied Sciences Inc. 1998a and b for discussion of the effects of hydrostatic pressure on bubble growth). The hypothesis was supported in part by experiments conducted by Aspen Applied Sciences Inc. (1998b). In these studies, the time to initiation of mortality for juvenile chinook salmon exposed to a sea level TGP of 140% and temperature of 10° C near the water surface was 3.7 hours longer for a group of fish that had received hydrostatic pressure pre-treatment compared to a group of fish without pre-exposure to hydrostatic pressure. The treatment fish were held in 3 m of water depth at sea level TGP of 100% for 3 hours. The fish that had no hydrostatic pre-exposure started to die at about 3 hours while the fish with hydrostatic pre-exposure began to die at about 6.7 hours. The experiments also showed that as mortality proceeded, the rate of mortality for treatment fish that died later was lower than that of the fish that had not received the hydrostatic pre-exposure. Although only one replicate of this experiment was performed, and there was a difference in the mean fork length of the fish in the treatment and control groups (139 mm versus 154 mm), the results suggested that pre-exposure to hydrostatic pressure may have had a beneficial effect in terms of delaying the onset of mortality from exposure to elevated TGP.

In the Rosewall Creek experiments, two groups of rainbow trout (8 replicates of 20 fish each) were exposed to a hydrostatic pressure equivalent to 2.75 metres of water depth for 3 hours before exposure to a TGP of 122% at the water surface. The corresponding control groups of fish received no hydrostatic pressure exposure prior to exposure to a TGP of 122%. Although there was an increase in time to mortality for treatment fish of the first group pre-exposed to hydrostatic pressure, compared to the control fish without pre-exposure, there was a statistically significant difference only after 48 hours. In the second group of fish pre-exposed to hydrostatic pressure, there was no statistically significant difference from the corresponding control group without pre-exposure to hydrostatic pressure. Although there appeared to be a delay in the onset of mortality for the first group, it was not as large as was observed with the chinook salmon of the Aspen Applied Sciences (1998a) experiments. The reduced response in the first group and the absence of a statistically significant difference between the treatment and control fish of the second group may be due to differences in

susceptibility to GBT between the chinook salmon of the Aspen Applied Sciences Inc. (1998b) experiments and the DFO Canada rainbow trout. Numerous studies have reported that chinook salmon appear to be more resistant to GBT than steelhead or rainbow trout (Dawley and Ebel 1975, Dawley et al 1976, Stroud and Nebeker 1976, and Meekin and Turner 1974, Aspen Applied Sciences Inc. 1998). In addition to the difference in species, the fish of these experiments were smaller than the ones in the Aspen Applied Sciences (1998b) experiments (91 – 97 mm FL versus 139 – 154 mm FL). It is not known how this difference in length might affect the response of exposure to hydrostatic pressure and susceptibility to GBT.

Alternatively, given the lower TGP of the Rosewall Creek experiments (i.e., 122%) compared to the 140% of the Aspen Applied Sciences Inc. (1998) experiments, the differences may be the result of the longer time to initiate mortality at the lower TGP. Although nucleation site collapse may have occurred during the pre-exposure to hydrostatic pressure, the nuclei would have re-grown to near their original size at some time after the fish were returned to the surface. However, the time required for the nuclei to re-grow to their original size would be a smaller percent of the total time required to initiate mortality at 122% TGP (18 hours) compared to 140% TGP (3 hours). If true, then it would be expected that the effects of pre-exposure to hydrostatic pressure would be most pronounced at high TGPs compared to low TGPs. These experiments were limited to a hydrostatic pressure equal to 2.75 metres of depth. Greater depths would be expected to collapse nucleation sites even further and perhaps delay their re-growth even more. This might produce a more significant delay in the onset to mortality for any given TGP and perhaps reduced rate of mortality. Additional time at depth may also provide a beneficial effect. Additional experimental work would be required to examine these effects in detail.

## **Assessment**

**Phase I Studies:** These studies illustrated that application of the primary TGP guideline of 110%, which protects all species and life-histories from the acute effects of DGS, would be conservative in some situations. It would be conservative for short exposures, since the literature data indicate that long exposure periods are required to elicit mortality at low TGP, even in shallow water. It would also be conservative at 110%, or even at higher TGP levels, if fish periodically use sufficient water depth, relative to the compensation depth, to reduce or eliminate bubble growth processes. Both volitional and non-volitional dynamic exposures clearly show increased time to mortality and reduced rate of mortality. Unfortunately, little is known about juvenile fish depth behaviour in the Columbia River. Although it is not possible to apply these results directly, the general features of dynamic TGP- depth exposures is an important consideration in the development of WUP performance measures.

**Phase II Studies:** From these studies it was concluded that swimbladder overinflation for TGPs up to 125%, if present, was not severe enough to affect time to seek cover performance. There was no evidence of altered fish behaviour, swimming orientation, seeking deeper water, venting of bubbles from the swim bladder. It was evident that the primary B.C. guideline A, which limits TGP to 110% regardless of water depth, would protect juvenile fish in shallow water environments from the effects of swimbladder overinflation.

Phase III Studies: These studies provided very contrasting results with earlier studies of Aspen Applied Sciences Inc. (1998) that examined the effects of hydrostatic pressure pre-exposure on susceptibility of fish to GBT. However, the two studies involved different fish species, fish sizes, and very different TGP levels. As a result, the 2001 Rosewall experiments may have raised more questions than they resolved and clearly identify the need for additional inquiry. Also, the depths of hydrostatic exposure in both of these experiments were quite shallow in comparison with the depths available to fish in the Columbia River. Similarly, times of exposure at depth were short in relation to what fish in the Columbia River might experience. Without a more substantial deep tank (i.e., 10 m or more deep), it will be difficult to address these issues. In the mean time, it is not clear how fish use of hydrostatic pressure and any GBT inhibiting effects that may result can be factored into WUP TGP performance measures.

### **2.3 Re-examination of the Literature in Relation to the B.C. Guideline and the DFO Studies.**

Guideline B was derived primarily from scientific data developed by Wright and McLean (1985) and Shrimpton et al. (1990a and b). The data of Wright and McLean (1985) showed that chinook salmon fry held in shallow hatchery raceways (approximately 0.4 m deep) at a sea level TGP of about 105% for 122 days developed a differential mortality about 2% greater than that of control fish held in the similar raceways at a sea level TGP near 100%. Although the exact cause of the differential mortality could not be identified, it was speculated that TGP induced swimbladder overinflation and overbuoyancy were the probable causes.

The work of Shrimpton et al. (1990a) confirmed the threshold equations of Fidler (1988) that predicted the swimbladders of physostome fishes such as rainbow trout would begin to over-inflate at sea level TGPs of about 103%. This work also showed that swimbladder overinflation was a problem primarily for rainbow trout 35 to 100 mm in length.

Although the work of Shrimpton et al. (1990a and b) formed the primary basis for the linearly varying TGP with depth that formed Guideline B, there remains the question as to its applicability in aquatic environments other than those of the laboratory. Specifically, the unique laboratory conditions under which the thresholds for swimbladder overinflation were determined (i.e., anaesthetized and restrained fish) do not exist for freely swimming fish in rivers and lakes. These questions, in part, formed the basis for the Phase II bioassay work summarized above.

As described, the Phase II bioassays failed to show any evidence that swimbladder overinflation was a problem for free swimming rainbow trout and coho salmon fry for sea level TGPs ranging to well over 110% (the upper boundary of Guideline B). Treatment fish performed the same as control fish as far as observable behaviour and when subjected to challenges of finding cover. Based on these results, it can be concluded that although swimbladder overinflation could occur in anaesthetized and restrained laboratory fish, it does not pose a problem to free swimming fish.

If swimbladder overinflation was the cause of the differential mortality observed by Wright and McLean (1985), it was unique to hatchery environments where fish were held in shallow water for periods of 4 months or more. Such a combination of depth and duration of exposure is unlikely to occur in rivers and lakes. Thus, for free-swimming fish in Canadian rivers and lakes, the Phase II

experiments demonstrated that fish do not require protection from swimbladder overinflation caused by low level dissolved gas supersaturation in the range of 103 – 110% TGP.

Other signs of acute or chronic GBT below 110% TGP that have been reported in the literature were examined to determine whether a low-level guideline is required to address signs of GBT other than swimbladder overinflation. With the exception of the observations reported by Wright and McLean (1986), no conclusive evidence could be found that acute or chronic effects exist for any of the fish species examined (see Fidler and Miller 1997). The only reported signs of GBT at TGP levels below 110% are those of Dawley et al. (1976). In these studies, a variety of signs ranging from emphysema of external skin surfaces to bubbles in gill filamental arteries (but no swim bladder overinflation) were reported for steelhead smolts at TGP levels as low as 105%. However, further reviews of the scientific literature revealed that these observations are unique and have not been reported by other investigators examining fish exposed to TGPs less than 110%. Most likely, the results of Dawley et al. (1976) were due to the fact that the experimental fish were captured directly from the Snake River (personal communication with the primary author), where TGP levels were known to be high (e.g., >125%). If so, the uniqueness of the Dawley et al. (1976) observations are probably be due to the fish having had previous exposure to high TGP. When emphysema and cardiovascular bubbles have occurred previously, the re-initiation of GBT signs under subsequent exposures may occur more rapidly and at lower TGP levels (Aspen Applied Sciences 1998a and b). The 1976 data of Dawley et al. contrast not only with other data from the literature (Meekin and Turner 1974, Stroud and Nebeker 1976), but also with earlier work by Dawley and Ebel (1975), where neither signs of GBT nor GBT related mortality were detected at TGPs of 110%.

Although there are no bioassay data that demonstrate any acute or chronic signs of GBT for TGPs below 110%, there remains the question of what happens to fish exposed to TGPs of 110%. In developing Guideline A (Fidler and Miler 1997), it was observed that, excluding the Dawley et al. (1976) and Wright and McLean (1986) data, the lowest TGP level for any acute or chronic effects of GBT reported in the literature occurred at sea level TGPs of 110%. That is, the lowest observed effects level (LOEL) was 110%. At TGPs near 110%, several investigators have reported both acute and chronic effects, albeit some under unique environmental conditions. For example, Jensen (1988) found that after five days exposure to a TGP of 111% and a temperature of 10 °C, 1.4% of a population of small (25 mm fork length) steelhead trout held in 2.5 cm water depth developed a bubble in the buccal cavity. Although there were no mortalities and the bubble eventually disappeared, it left a permanent deformity of the buccal cavity. Jensen speculated that this would reduce the chances of survival for these fish. Also noted, at 50% O<sub>2</sub> saturation (TGP = 111%), ruptured swim bladders occurred in 2.6% of the population (water depth = 2.5 cm). No swimbladder ruptures were observed at O<sub>2</sub> saturation levels of 75% or greater.

In yet earlier work, Jensen (1980) reported 13% mortality in steelhead alevins held in Heath trays (2.5 cm deep) caused by bubble formation in the buccal cavity at a TGP of 110% and a temperature of 12 °C. However, these experiments involved very soft water (10 mg/l CaCO<sub>3</sub>). At lower water temperatures and higher levels of water hardness (100 mg/l CaCO<sub>3</sub>), no mortalities or signs of buccal deformities were observed.

The results of Jensen (1980 and 1988) indicate that in order to produce buccal cavity deformities in alevin size steelhead trout, a TGP of 111% or greater, or a TGP of at least 110% combined with very soft water (10 mg/l CaCO<sub>3</sub>) and a temperature of at least 12 °C, are required. However, this GBT condition was restricted to hatchery Heath trays where the water depth is 2.5 cm. Although a few

rivers and lakes may have very soft water, it is unlikely that alevins would be found in water of 2.5 cm deep for extended periods. In the DFO Phase II experiments involving exposures in Heath trays, there was no indication of buccal cavity bubbles in coho salmon 35 mm in fork length or in rainbow trout 44 mm in fork length at TGPs up to 114%, suggesting that the problem is unique to alevins under the conditions examined by Jensen (1980 and 1988). For these reasons, the GBT observations of Jensen (1980 and 1988) apply to hatchery environments only and are not likely to have relevance to rivers and lakes where alevins will be found in deeper water. The shallow water depths used by Jensen (1980 and 1988) might correspond to conditions of stranding or potential dewatering in natural environments.

In other work related to TGPs of 110%, Meekin and Turner (1974) reported that for temperatures ranging from 9.4° C to 12.8° C, a minimum of 4 – 10 days (depending on fish species and length) is required to produce 10% mortality for exposures in shallow (0.17 m) water environments. However, Meekin and Turner (1974) give no indication of the cause of mortality (i.e., cardiovascular bubbles, emphysema, etc.) A somewhat disturbing feature of the Meekin and Turner (1974) work is that for one experiment, they report signs of GBT at TGP exposures of 98%. Stroud and Nebeker (1976) noticed emphysema on the operculum and haemorrhaging from gills of steelhead smolts at a TGP of 110% and a temperature of 10 °C after 6 days of exposure in vessels 0.6 m deep. They did not report whether they used control fish in the experiments, much less whether control fish were examined for gill haemorrhages or any other physiological abnormalities that were observed. It should be noted that no other investigators have reported haemorrhaging from the gills at this low of a TGP. Interestingly, Stroud and Nebeker (1976) did not observe any mortalities at either 110% or 115% TGP after up to 17 days exposure (water temperature = 10 °C and depth = 0.6 m). In the DFO Phase I, II, and III studies, no evidence of gill haemorrhages was found even at TGPs up to 122%.

Dawley and Ebel (1975) reported minor signs of GBT but no GBT related mortality in 130 mm steelhead trout and 125 mm Chinook salmon after 35 days exposure at TGPs of 110% and a temperature of 15 °C (water depth = 0.25 m). Nebeker et al. (1979) reported no mortalities in sockeye salmon after 21 days exposure to a TGP of 110% in 0.28 m of water and a temperature of 12.5 °C. Nebeker and Brett (1976) found no GBT mortalities in 160 mm coho salmon, 140 mm sockeye salmon or 200 mm steelhead trout after 25 days exposure at a TGP of 110% and a temperature of 12 °C (water depth = 0.6 m); however, some emphysema of external body surfaces was observed in the steelhead trout. Nebeker and Brett (1976) did not report any haemorrhaging from the gills.

The above observations are somewhat contradictory in that some investigators observe mortalities at a TGP of 110% while others find no mortalities up to a TGP of 115%. In the case where mortality was observed, it was always in shallow water environments involving long exposure times and there was no indication as to the cause of mortality. Similarly, some investigators observe external signs of GBT at a TGP of 110% while others find few if any signs. If the reported observations of acute or chronic signs of GBT are correct, then the primary guideline A may be inadequate to protect aquatic organisms from the effects of GBT. However, it is not clear at this time how to derive a guideline for TGPs near 110% given the contradictory nature of the data. Clearly, these contradictions must be resolved before a guideline can be derived. This is especially true in the case of the Meekin and Turner (1974) observations since these are the only observed mortalities at a TGP of 110%.

The problem becomes complicated further, when the effects of fish depth behaviour and hydrostatic pressure are considered. For example, the mortalities reported in the literature at a TGP of 110%

occurred in laboratory environments where fish were confined to shallow water (0.17m) for lengthy exposure periods. Furthermore, the exposure vessels were of a size that prevented fish from swimming freely. Fish in rivers and lakes are not likely to spend days confined to the water surface and limited in their swimming freedom. Furthermore, the results of the DFO Canada Phase I and II experiments show that fish use of water depth can significantly reduce their susceptibility to GBT even at TGP levels well above the maximum (110%) specified by Guideline A. Thus, periodic excursions to depths of 1 metre or more may be sufficient to avoid any signs of GBT at TGPs above 110%.

A concern regarding mortality at sea level TGPs between 110% and 115% is that the cause of mortality has not been identified in the literature. The DFO Canada Phase I, II, and III experiments along with data from Dawley and Ebel (1975), Stroud and Nebeker (1976), Nebeker et al. (1976) clearly show that sea level TGPs of 115% or greater are required to illicit bubble growth in the cardiovascular systems of fish. Importantly, these results are for shallow water laboratory environments. Some of the other signs of GBT reported for TGPs between 110% to 115% are emphysema of external body surfaces including the lining of the buccal cavity (Stroud and Nebeker 1976, Weitkamp and Katz 1980, Fidler 1988), exophthalmia (Dawley and Ebel 1975), spinal flexures (Dawley and Ebel 1975, Shrimpton 1985), and extracorporeal bubbles between gill filaments (Fidler 1988). However, it is not clear how these signs might lead to the mortality observed between TGPs of 110% and 115%. The cause of mortality is important because, as in the case of swimbladder overinflation, one must be sure that mortality is not an artifact of the experimental environment. For example, in the mortalities reported by Meekin and Turner (1974) at a TGP of 110%, the fish were confined to small exposure containers (about 30 cm square), while the fish of Stroud and Nebeker (1976) that showed no mortality at TGPs up to 115% were in 12 ft diameter exposure vessels. The added freedom of movement in the Stroud and Nebeker (1976) exposure vessel may have been a factor in the absence of mortalities. Although the depths of the exposure vessels differed, the added 0.3 m of depth in the Stroud and Nebeker (1976) exposure vessels would not have fully compensated for the 115% TGP versus the 110% TGP of the Meekin and Turner exposure vessels. Regardless of these speculations, the cause or causes of mortality in the 110% to 115% TGP range are unknown and it is not clear whether mortality would occur in free-swimming fish exposed to these TGP levels.

Another study that has some relevance to GBT mortalities near 115% is that of Birtwell et al. (2001) in which juvenile chum salmon were exposed to seawater at a temperature of 20.7 °C and TGPs of 115% and 120%. Following these exposures, the fish were subjected to a predation challenge. For an exposure of 48 hours at a TGP of 115%, Birtwell et al. (2001) found no increase in rates of predation in treatment fish over control fish. However, after a 24 hour exposure to a TGP of 120% the predation rates in the treatment fish were significantly higher than with the control fish. Since a TGP of 115% is at the lower threshold for cardiovascular bubble growth (115% - 119%, depending on species – Fidler 1988, Fidler and Miller 1997), it is possible that fish in this treatment were not stressed by GBT and behaved no different than the control fish. However, at a TGP of 120% bubble growth in the cardiovascular system was probably the key factor in the increased rate of predation. Given that the fish were chum salmon, the experiments were conducted in sea water, and there was a thermal stress component, it is not clear how these results can be transferred to fish in the Columbia River. However, the Birtwell et al. (2001) results are consistent with the threshold equations of Fidler (1988) and demonstrate that fish stressed by GBT are more susceptible to predation.

## 2.4 Summary of TGP Guideline, DFO Canada Studies, and Literature Data Review

From the previous discussions, the key elements in relation to WUP performance measures are:

- The B.C. Guideline B for DGS was developed using laboratory data for fish exposed to DGS in shallow water environments where extremely long exposure times (months) were required to elicit a presumed overbuoyancy response. In other laboratory data used in developing the guideline, fish were either restrained, anesthetized, or both.
- The DFO Canada Phase I, II, and III experiments show that swimbladder overinflation is not a problem in free-swimming juvenile rainbow trout and coho salmon for water TGPs up to 125%, which is well above the B.C. guideline B values of 103% to 110%.
- Thus, the B.C. Guideline B should have little relevance to the development of Columbia River WUP performance measures.
- The DFO Canada Phase I, and III experiments show that intermittent exposure to DGS resulting from periodic increases in hydrostatic pressure can prolong the initiation of mortality and reduce the rate of mortality in populations of fish. These results were obtained for hydrostatic exposures of 2.75 m. Added benefits might result from deeper and longer hydrostatic exposures.
- No conclusive evidence for other signs of GBT at TGPs below 110% has been reported in the literature.
- The B.C. Guideline A (sea level TGP = 110%) was developed from laboratory data involving fish exposed to DGS in shallow water environments and often in confined spaces.
- There is considerable contradictory evidence for signs of GBT and mortality at TGPs of 110% to 111% - some literature reporting signs and mortality while other literature reporting neither signs nor mortality.
- There is no clear understanding of the cause of GBT mortality for fish exposed to TGPs between 110% and 115%.
- Cardiovascular bubble growth is the cause of mortality in fish exposed to TGPs greater than 115%, in shallow water environments (depth = 0.25m).
- It is not clear how to apply guidelines developed from laboratory data to rivers and lakes where water depth and fish behaviour may significantly alter susceptibility to GBT.
- Fish stressed by GBT display behaviour changes that make them more prone to predation.

In general, the B.C. guideline parts A and B are supported by questionable data from the literature. Conditions unique to laboratory environments are, in part, responsible for these shortcomings. However, inconsistencies between results reported by various investigators add to the uncertainties in the data. Essentially, if the guidelines were applied only to shallow water laboratory environments, there would still be questions as to their validity. Clearly, it would be difficult at this time to defend extending the guidelines to rivers and lakes, where environments are dramatically different and fish behaviour becomes a dominant factor. With these key points in mind, the current review moves to an examination of field studies in the Columbia River that have attempted to identify more realistic relationships between river TGP and signs of GBT in fish.

### **3.0 Studies of TGP and GBT in the Columbia River Below the Hugh Keenleyside Dam**

A number of studies have attempted to establish the relationship between TGP and GBT in fish of the Columbia River. Early work (Ash et al. 1981, R.L. & L. 1984) did not find any signs of GBT in dead fish; however, the levels of TGP were not known at the time of the examinations. Two, more recent studies, were designed to examine the TGP/GBT relationship specifically. The first of these is the Hildebrand (1991) study.

#### **3.1 Lower Columbia River Fisheries Inventory Studies 1990 (Hildebrand 1991)**

The 1990 studies of Hildebrand reported some of the general characteristics of Columbia River fish populations compared to earlier studies. Specifically:

1. Mountain whitefish abundance in the upper section of the river had decreased since the 1980's.
2. Rainbow trout abundance since the 1980's had increased.
3. Walleye populations increased considerably since the 1980's. This may be responsible for decline in juvenile mountain whitefish and red side shiner populations.
4. Kokanee abundance similar to 1980's.
5. White sturgeon less than 1000 mm length had decreased since the 1980's.
6. Burbot were in low abundance in the upper section of river.

It is unknown how DGS has contributed to these observations over the period of comparison. However, the increases in rainbow trout populations suggest that it has had little effect on this species.

In the Hildebrand (1991) work, two methods were used to assess the effects of elevated river TGP on fish. In the first method, free swimming fish were captured by boat and backpack electro-shocking and angling. About 7500 fish of 4 different species including fry through adult age classes were examined for external signs of GBT. Of these fish, 45 out of 1443 (3.1%) largescale suckers showed signs of GBT. In addition, 6 out of 226 (2.6%) longnose suckers, 2 out of 4577 (0.04%) of mountain whitefish, and 10 out of 1343 (0.7%) rainbow trout showed signs of GBT. As well, angling near the tailrace of the Hugh Keenleyside Dam captured 13 rainbow trout of which 3 (23.1%) had external signs of GBT. Of all the fish examined, the severity of GBT signs ranged from mild to severe.

In addition to these studies, live cage experiments were conducted where fish were held in cages at various depths in the Columbia River. Adult rainbow trout, mountain whitefish, and walleye were the principal species used in these experiments. Throughout both the river capture studies and the live cage studies, maximum TGP levels were recorded in the 120% – 125% range. An inconsistency was observed in the results of the live cage studies when compared with data from the literature. Essentially, the fish exposed in the Columbia River were dying much too quickly for the TGP and temperature levels recorded. LT100s of 5.5 hours or less were observed for all three species held in water depths between 1.0 and 1.5 m. Based on data from the literature, an LT 50 should not be reached in less than 30 hours for a TGP of 125% in 0.25 m of water (Dawley et al. 1976, Fickeisen and Montgomery, 1978, Antcliffe et al. 2002a).

#### **Assessment**

In consideration of performance measures for the Columbia River WUP, the Hildebrand (1991) report was re-examined to establish if the data from the 1990 study provided information that would help in the selection of a reference or threshold TGP. The issue of the rapid rates of mortality in the live cage studies became an immediate concern. Initially it was thought that electro-shocking of the fish during their capture may have played a role in the high mortality rates. It was speculated that muscle spasms induced by electro-shocking might have activated nucleation sites in the muscle and cardiovascular systems of the fish. However, after further consideration, it was thought more likely that the TGP monitoring instruments were at fault and the actual TGPs in the river were considerable higher than those recorded. To check this hypothesis, the operational records for the Hugh Keenleyside Dam for 1990 were examined. It was found that the sluice gates were used exclusively to release water from the dam between May 18 and December 31, 1990 (i.e., during the entire early summer, late summer, and fall portions of the study). It is well known that the dams' sluiceways produce the very high TGPs in the Columbia River with levels in excess of 140% being recorded (Maxwell 1985, Hildebrand 1991, Aspen Applied Sciences Ltd. 1995 and 2000). To validate the measured river TGP levels, the HLK/TGP/GBT computer model was used to predict what river TGP levels should have occurred during the study period. Using 1990 recorded discharge levels, forebay elevations, tailwater elevations, and sluice gate openings (Kath Bowie, B.C., personal communication – July 18, 2002), the model predicted river TGPs immediately below the Hugh Keenleyside Dam of between 135% and 137% for the entire period from May 18 through November 30, 1990. This is considerably high than what was recorded during the Hildebrand (1991) study. Although the higher TGP levels are more consistent with the rates of mortality observed in the cage studies, there remained concern that the rate of mortality was still too high. Specifically, at a depth of 1 metre where the live cages were placed, the  $\Delta P$  should have been about 200 mmHg for a TGP of 137%. This would be somewhat comparable to fish being exposed at the water surface to a TGP of 127%; in which case, the observed LT100 would have still been much too short. However, it was noted by Hildebrand (1991) that the cage fish were captured in shallow river water and were held in a shallow live box in the boat before being placed in the cages. Columbia River water circulated through the live box. Since the river water would have been at about 137% during this period, the fish would have had a pre-exposure to a higher  $\Delta P$  (278 mmHg) than they had in the live cages. The pre-exposure could have lasted for several hours and would then explain the rapid rate of mortality once the fish were placed in the live cages. Based on the high mortality rates in the live cage studies and the predictions of the HLK/TGP/GBT model, it is likely that TGP monitoring instruments were in error and that the actual TGP levels in the river during the entire early summer, late summer, and fall portions of the 1990 study were between 135% to 137%.

During the last 2 days of the 1990 winter session (conducted 12 – 21 February), the low level ports were used in combination with the sluiceways to release water from the Hugh Keenleyside Dam. Again, using the HLK/TGP/GBT computer model and data from B.C. Hydro, the predicted Columbia River TGPs would have been about 112%. Prior to February 19, the sluiceways were the only release facilities used and river TGPs would have been in the 121% - 125% range. During the entire spring session (conducted 11 April - 11 May), the low level ports were the only release facility and river TGPs would have been between 101% and 103%.

The above discussion applies to that portion of the Columbia River between the Hugh Keenleyside Dam and the confluence with the Kootenay River. One of the problems in assessing Columbia River TGPs below the confluence is that neither Kootenay River flows nor TGPs were monitored during most of the 1990 study. Only one TGP measurement of about 120% was recorded in the Kootenay

River on 6 June, 1990. Thus, it is difficult to determine what TGP levels existed below the Columbia/Kootenay confluence for the entire study period. However, this may not be so important when considering where most of the sampling of free-swimming fish for signs of GBT took place. The sampling was conducted in three sections of the Columbia River; one above the Columbia/Kootenay confluence and two below. These were designated the Upper, Middle, and Lower sections. Most of the sampling took place in the Upper section between the Hugh Keenleyside Dam and the Columbia/ Kootenay confluence. All of the rainbow trout and mountain whitefish sampling took place in this section. About 85% of the sampling of longnose suckers took place in the Upper section and about 27% of the sampling for largescale suckers. Thus, most of the incidence of GBT signs was observed under conditions where the river TGP was in the 135% - 137% range.

This places the results of the in-river sampling in a completely different perspective. That is, given the very high levels of TGP in the river during the early summer, late summer, and fall monitoring sessions, it is surprising that the incidence of GBT signs in captured river fish was so low. It is even more surprising when one considers that the exposure to TGPs between 135% and 137% was continuous for more than 200 days. The low incidence of signs may be due, in part, to fish making use of deeper water in the river and the corresponding benefits of hydrostatic pressure compensation. If so, the use of deeper water may have been due to normal depth behaviour and not any specific attempt to avoid DGS. In a subsequent study sponsored by B.C. Hydro (Prince et al., 2000) the use of deeper water by adult rainbow trout was confirmed along with confirmation that this was not driven by DGS avoidance (Section 3.3).

One of the issues that must be addressed in the Hildebrand (1991) study is whether, in spite of the low incidence of signs, GBT mortalities were occurring. In the extreme case, given the 200+ days of continuous TGP between 135% and 137%, every fish in the river that ventured into shallow waters for more than a few hours should have been killed. Yet, no dead fish with or without signs of GBT were found. Still, finding dead fish in large rivers, regardless of the cause of death, is extremely difficult. Aquatic predators can home in on live fish stressed from signs of GBT (Birtwell et al. 2001), making it impossible to detect these indirect acute mortalities. Dead fish can sink to the bottom and go undetected. Avian predators can pick up dead fish floating on the water surface or stressed fish swimming near the water surface. In general, large numbers of dead fish are found in rivers only when mortality has been massive (Wes Ebel., U.S. National Marine Fisheries Service, retired – personal communication, 1999). In the case of the Hildebrand (1991) study, one would expect that the live, free-swimming fish that were captured should have shown higher levels of GBT signs if large mortalities were occurring. However, given the low incidence of GBT signs, it is likely that the levels of GBT mortality were also low. Certainly, in the extreme case described above, the entire river fishery would have been severely impacted for years and would not have gone undetected. Given the extreme conditions of 1990, the Hildebrand (1991) report suggests that Columbia River fish may be able to tolerate higher levels of TGP than suggested in the B.C. Guideline without developing signs of GBT.

One of the original goals of the Hildebrand (1991) live cage studies was to determine if Columbia River fish showed increased resistance to GBT over fish that had been examined in other studies. The hypothesis was that because fish in the Columbia River below the Hugh Keenleyside Dam have been exposed to high TGP over many generations, evolutionary changes may have occurred that result in more TGP resistant populations. This resistance, if present, may be the result of physiological adaptations (e.g., fish with smaller nucleation sites or less body fat), behaviour adaptations (e.g., the use of deeper water), or both. Because of the high TGPs, the live cage studies

would not have been able to answer the question regarding any added resistance to high TGP resulting from physiological adaptation. That is, TGPs of 135% to 138% would have killed all fish regardless of any inherited resistance at lower TGP levels. Nevertheless, the issue of increased resistance of Columbia River fish to TGP remains important in terms of WUP performance measures. Specifically, if there is increased resistance, it may allow the TGP thresholds used to develop WUP performance measures to be higher than those specified in the B.C. TGP guideline.

### **3.2 Depth Distribution Patterns of Telemetered Columbia River Rainbow Trout (Prince et. al. 2000)**

In a July 1998, B.C. Hydro funded study, adult rainbow trout (N = 18) were equipped with sonic transmitters containing pressure sensitive depth sensors and released into the Columbia River below the Hugh Keenleyside Dam. Although TGP levels below the dam during much of the summer were above provincial water quality guidelines, only one experimental fish, a kelt, showed signs of GBT when captured. Tagged fish did not move into tributary systems where TGP levels were lower, nor did they tend to occupy confluence areas within the Columbia mainstem. Rather, the most common type of habitat used was deep-water eddy habitat. During the summer months when TGP levels are high, the slow velocities provided by eddies (average 0.3 m/s, range 0.0 to 0.5 m/s) reduce swimming demand. Although this behaviour may have nothing to do with DGS, swimming stress is known to decrease time to GBT mortality (Stroud and Nebecker 1976). In addition, eddies generally collect invertebrates, provide habitat for small fish, and offer compensating depth.

Rainbow trout activity was greatest during the summer as fish moved among feeding areas. In contrast to lake residents that move offshore at night to feed on prey fish, large riverine rainbow trout moved near shore at night, presumably to feed on smaller fish inhabiting the shoreline.

The depths occupied by adult Columbia River rainbow trout appeared to be strongly influenced by foraging strategy and food availability. The study showed two distinct depth modes for adult rainbow trout: one at or just below the maximum compensation depth of 5m (summer) and another at 15-20m depth (winter). Seasonal distribution patterns showed that more fish occupy non-compensating depths (< 5 m) during the summer months than at other times of the year. The high proportion of fish (76.5%) that spent time near the surface in summer compared with other seasons (50.0% in fall and 27.3% in winter) probably reflects the ability of invertebrate hatches to draw adult rainbow trout to feed at the water's surface.

The vertical movements of adult Columbia River rainbow trout may be characterized by periods of frequent sounding interspersed with extended periods of inactivity (hours to days). While average daily depth (mean depth 10.4 m, range 3.91 to 20.3 m, n=17) was well below the maximum compensation depth required in the Columbia River, some individuals spent hours and days in shallow (<1m) depths regardless of TGP level. It is hypothesized that intermittent-sounding to depth may offset the effects of GBT altogether for these fish, despite spending lengthy periods near the surface.

In 1998, operations at the HLK Dam that delivered flow through the sluices resulted in high TGP levels in the Columbia River below the dam. Records of fish behavior (N=8) during the operations experiment showed no significant difference in fish depth across varying levels of total dissolved gas (108-128% saturation, Friedman ANOVA 0.25 <P<0.50). Adult fish did not alter their depth distribution and some continued to spend prolonged periods at depths less than one meter even when

exposed to gas levels that exceeded cardiovascular bubble thresholds (*i.e.*, TGPs greater than 115% – 119%). These preliminary results suggest that adult rainbow trout either cannot detect DGS in their environment or, if capable of detecting it, do not avoid it. Regardless, given their frequent diving behavior, average depth (>5m), and seasonal activity, Columbia River rainbow trout appear, for the most part, to be compensating behaviorally for some of the effects of DGS.

## Assessment

The 1998 adult rainbow trout depth behaviour studies show that during the summer months, these fish alternate between shallow feeding activities and deep water holding periods. The depths involved in the holding periods are sufficient to reverse any cardiovascular bubble growth that may have occurred near the water surface during feeding periods (Aspen Applied Sciences Ltd. 1998a and b). However, it is not known if cardiovascular bubble growth actually occurred. Given the long periods over which the fish were tracked, it is unlikely that significant cardiovascular GBT occurred. This assessment is based on data from the literature which clearly show that, once cardiovascular bubble growth begins, fish generally become disoriented and swim in circles near the water surface, usually on their sides. None of this behaviour was observed in the experimental animals. The behaviour bias to deeper water when insect hatches are not occurring should improve their condition as far as GBT is concerned. It is not known if the observed depth distribution is unique to Columbia River adult rainbow trout. The fish that were examined have lived in the system for 3 to 5 years and are both survivors of their brood line (*i.e.*, did not succumb to DGS) and the product of selection under an altered TGP regime.

Although these data do not address the behaviour of juvenile fish, they do support the 1990 observations of Hildebrand (1991) in terms of adult fish. Specifically, the low incidence of GBT signs in rainbow trout is consistent with fish use of deeper water habitats that afford hydrostatic pressure compensation. It is important to note that little is known about the dynamic depth behaviour of other fish species in the Columbia River.

### 3.3 Columbia River TGP Data and Analyses Sources

Along with the various biological studies of DGS and its relationship to GBT in fish, a number of other studies have attempted to both record and predict TGP levels in the Columbia River below the Hugh Keenleyside Dam. These are described briefly as follows.

**DFO Canada Access Database of British Columbia TGP Measurements:** This database was developed by Aspen Applied Sciences Ltd. (Fidler and Miller 1997c). Recorded TGP data from the literature, government agencies, and corporations were assembled into a Microsoft Access Database that covered all data for the province of British Columbia through 1995. The database covered rivers, lakes, streams, as well as the marine environment. Both spot measurements and continuous data were included to yield a database of over 45,000 records. Data from the Columbia River below the Hugh Keenleyside Dam made up the bulk of the database.

**CRIEMP Total dissolved gas pressure database for the Columbia, Kootenay, and Pend d'Oreille Rivers 1995 – 2000:** This database, developed by Golder Associates (2002), picks up where the DFO Canada database ended and extends it through 2000. As noted in the title, it is restricted to TGPs in the Columbia, Kootenay, and Pend d' Oreille Rivers. Most of the data in the database involves continuous measurements. In addition to TGP data, the database contains

corresponding dam operations information (i.e., discharges, by facility, gate openings, total head, etc.).

**B.C. Hydro Operations Database:** Although this database does not contain TGP information specifically, it does contain complete information on the operations of the Hugh Keenleyside Dam including forebay elevations, tailwater elevations, discharge by facility, and individual gate openings. When coupled with the Hugh Keenleyside TGP/GBT Computer Model (see below), past river TGP levels can be predicted with reasonable accuracy.

**Hugh Keenleyside TGP/GBT Computer Model:** This model, developed by Aspen Applied Sciences Ltd (1995, 1996, 1998, 2001) for B.C. Hydro, predicts Columbia River TGP levels from below the Hugh Keenleyside Dam to the Canada/US border. The computer program integrates Hugh Keenleyside, Brilliant, and Waneta Dam operations, dissolved gas production, Columbia, Kootenay, and Pend-Oreille River hydraulics, water levels and dissolved gas dissipation with the potential biological impacts associated with gas bubble trauma in fish and fish habitat de-watering.

**Analysis of TGP Reduction at the Hugh Keenleyside Dam as a Result of Power Production:** This study by Aspen Applied Sciences Ltd (1998) used the Hugh Keenleyside TGP/GBT Computer Model to evaluate the reductions in Columbia River TGP that would result from the installation of a power plant at the Hugh Keenleyside Dam. The analysis was based on historic daily average river flows for the years 1987 and 1991 through 1996. River TGPs, with and without the power plant, were predicted using daily average flows. From the daily TGP information, exceedence tables were constructed that showed days of the year that a range of TGPs were exceeded. The tables were used to assess the TGP benefits derived from the installation of the power plant.

**Dissolved Gas Reduction at the Brilliant Dam Due to an Expansion to the Power Plant:** A study similar to that for the Hugh Keenleyside Dam, but with TGP predictions for the Kootenay River and the Columbia River at Birchbank (mixed Brilliant Dam, Hugh Keenleyside Dam, and Arrow Lakes Generating Station flows). The study, performed by Aspen Applied Sciences Ltd (2000, 2001) predicts Columbia and Kootenay River TGPs using daily average flows from 1991 through 1999. The study re-examined the earlier Hugh Keenleyside analysis and added the flow years 1997 through 1999. Again TGP exceedence analyses were used to assess the TGP benefits derived from the expansion of the Brilliant Dam power plant.

**Fisheries Resource Information and TGP Risk Assessment for the Canadian Portion of the Lower Columbia River Basin:** This study, performed by R. L. & L. Environmental Services Ltd (2002 – now Golder Associates) examines the GBT risk to fish in the Lower Columbia Basin. It includes assessments of the mainstem Columbia River, the Kootenay River (from Kootenay Lake downstream) and the Pend d’Oreille River (from the Canada/US border downstream). Part of the mainstem Columbia River assessment is the river reach between the Hugh Keenleyside Dam and the confluence with the Kootenay River, which is most relevant to the Columbia River WUP analyses. TGPs were assessed from the previously described analysis of the Brilliant Dam expansion by Aspen Applied Sciences Ltd (2000, 2001). TGP risk assessments were based on the percent of time river TGPs exceeded specific levels (i.e., 110%, 115%, 120% and 130%). The choice of TGPs above 115% was somewhat for convenience rather than for any specific biological response. The 110% level corresponds to part A of the B.C. guideline, while the 115% corresponds to the threshold for cardiovascular bubble growth. The percent of exceedence time was calculated over a nine year interval (1991 – 1999). These numbers were then overlain on specific fish habitats within the river

reach and risks assessed based on time of habitat use by species, life stage, and preferred depth. Risks were rated as low, medium, and high and were assigned during a fisheries workshop in Castlegar, B.C. on September 6 – 7, 2000.

Because the risk assessments are so wide-ranging, they involve numerous tables that are highly detailed by habitat location, period of use, species, and age class. However, the tables do not account for the diversity in GBT susceptibility of different species and age classes or the depth behaviours of different species and age classes (i.e., dynamic river exposures versus static exposures). The latter becomes more important at lower TGPs than at high TGPs. At high TGPs (e.g. greater than 130%), short exposure times in shallow water can kill fish in hours. In this situation, the risks assessments are probably realistic. However, at lower TGPs (e.g., 115%), days may be required to kill fish, allowing more opportunity for fish, through normal depth behaviour, to periodically access deeper water where bubble growth can be halted or reversed (Aspen Applied Sciences Inc. 1998a and b, Antcliffe et al. 2002a). In these situations, the risk assessments may be overly conservative. There remains the problem of associating projected risks from the tables with actual observations of GBT in fish from the Columbia River. Given the low incidence of GBT signs in the study by Hildebrand (1991) and the extreme TGP conditions that existed in the river, the tables would appear to over estimate the risks. Perhaps the tables are best viewed in terms of relative risk and not absolute risk. In this context, the risk assessments at TGP levels of 115% and 120% may be useful to the WUP process (Section 4.0).

### **3.4 Summary of Columbia River Field Studies and TGP Data Sources**

From the previous discussions, the key elements in relation to WUP performance measures are:

- The observations from the field studies of Hildebrand (1991) in which fish were sampled directly from the river (i.e., electro-shocking, seining, etc.) contrast sharply with observations reported in the literature.
- Specifically, very low levels of GBT signs were found in a range of fish species that had been exposed to TGPs between 135% and 137% for a period of over 200 days.
- No dead fish were found; whereas, data from the literature for shallow water exposures at these TGP levels would have indicated 100% mortality in 10 – 24 hours (Fidler and Miller 1997).
- This level of mortality was plainly evident in Hildebrand's (1991) live cage studies.
- Clearly, fish depth behaviour, perhaps coupled with some selective resistance, must play a role in the low levels of GBT that existed in the Columbia River in 1990.
- The depth behaviour of adult rainbow trout, as observed by Prince et al. (2000), is consistent with depth compensation affording fish significant levels of protection from GBT.
- Two databases (Miller and Fidler 1997c and Golder Associates 2002) contain all of the TGP data that has been recorded for the Columbia River below the Hugh Keenleyside Dam through 2000. Dam operations data from 1995 through 2000 are also included in the Golder Associates (2002) database.
- Coupling of the Hugh Keenleyside TGP/GBT Computer Model with B.C. Hydro operations data allows river TGPs to be predicted for periods not covered by the Fidler and Miller (1997c) and Golder Associates (2002) databases.

- The risk assessment analysis by R. L. & L. Environmental Services (2002) may contain relative risk information that can be utilized in the development of WUP performance measures.

#### **4.0 Review Summary and TGP Threshold Recommendation**

This review has identified a number of important experimental observations that will significantly affect the development of WUP TGP performance measures for the Columbia River below the Hugh Keenleyside Dam. The first of these deals with the relevance of the B.C. guidelines for DGS in the Columbia River WUP process. The results of the DFO Canada Rosewall Creek experiments (Antcliffe et al. 2001, 2002a and b) have shown that TGPs up to 125% do not elicit behavioural changes in juvenile rainbow trout and coho salmon that would suggest overbuoyancy caused by swim bladder overinflation. The low level TGPs specified in Part B of the B.C. guideline are the result of unique conditions in a hatchery experiment (extremely long exposure periods at shallow depths, Wright and McLean 1985) or artifacts resulting from unique laboratory conditions (i.e., restrained and/or anesthetized fish, Shrimpton et al 1990a and b). The DFO Canada experiments suggest that Part B of the B.C. guideline is not relevant to the Columbia River WUP process. That is, Part A of the guideline will protect juvenile fish from any effects of swim bladder overinflation, if present. Furthermore, even Part A of the guideline is conservative in terms of swim bladder overinflation.

There are added concerns about Part A of the guideline based on inconsistencies in the scientific literature. Specifically, at TGPs of 110%, some researchers report signs of GBT and mortality while other researchers report neither signs nor mortality. In addition, there is concern over the fact that the cause of mortality between TGPs of 110% and 115% is unknown. The contrasting observations coupled with uncertainty as to the cause of mortality allow the possibility that other laboratory artifacts may be present in the data from which Part A of the B.C. guideline was derived. Essentially, the basis for Part A of the guideline is in question.

The DFO Canada experiments further show that fish intermittent use of water depth can significantly prolong the onset of mortality in a population of fish and reduce the rate of mortality. However, it is important to note that these results were obtained for depth exposures of up to 2.75 m and exposure times of between 3 and 6 hours. It is not known what additional benefits might be derived from much deeper exposures and longer exposure periods, as indicated in the Prince et al. 2000 studies. This is important because Part A of the guideline (with its contrasting observations and uncertainty as to the cause of mortality) was derived from steady state exposures in shallow water. In deep rivers such as the Columbia River, Part A of the guideline may not be relevant to WUP TGP performance measures if fish make regular use of deep water.

The results of the DFO Canada experiments are certainly consistent with the 1990 field studies of Hildebrand (1991). Examination of free-swimming fish from the Columbia River found low levels of external signs of GBT and no GBT mortality, even though TGP levels were extreme (135% - 137%) and exposure periods were extensive (greater than 200 days continuous exposure). These results clearly indicate that fish use of water depth, and perhaps some resistance due to evolutionary selection, are important factors in the low levels of chronic signs of GBT that were observed.

The use of deep water is confirmed, at least for adult rainbow trout, by the 1998 field studies of Prince et al. (2000). The studies show that fish regularly use water depths much greater than those in the studies from which Part A of the B.C. guideline was derived. The greatest risk appeared to be

associated with insect hatches in the summer and fall that brought the fish to the water surface. However, holding in water depths well below the compensation depth for extended periods followed these excursions to the water surface.

### **Recommended TGP Thresholds for Columbia River WUP Performance Measures**

This review has not only shown strong contrasts between Columbia River field GBT data and laboratory GBT data, but also strong contrasts within the laboratory data upon which the B.C. guidelines for DGS were derived. Yet, there is consistency between the DFO Canada Rosewall Creek experiments and the various field studies that have been conducted on the Columbia River. Thus, it is not evident that Parts A and B of the B.C. guideline are, at this time, applicable to the Columbia River WUP process and the selection of performance measure thresholds. An alternate basis using the 1990 field studies of Hildebrand (1991) is recommended for selecting performance measure TGP thresholds – specifically, that portion of the study involving the monitoring of free-swimming fish.

When compared to the manner in which the Hugh Keenleyside Dam is currently being operated (i.e., minimization of TGP), the 1990 Columbia River environment represents extreme TGP conditions. Given the TGP levels at the time (135% - 137%) and the 200+ days of continuous exposure, the incidence of chronic signs observed in the Hildebrand (1991) study are surprising low and more consistent with LOELs associated with much lower TGPs and considerably shorter exposure periods. Thus, it would be expected that TGPs on the order of 115% – 120%, coupled with much shorter exposure periods (e.g. 20 – 30 days maximum), may not be harmful to Columbia River fish in terms of acute or chronic GBT. It is therefore recommended that, for the time being, two TGP thresholds be considered in the Columbia River WUP performance measures. The first is 115%, which is the recognized threshold for cardiovascular bubble growth, albeit for shallow water environments. The second is 120%, which recognizes that fish can avoid the effects of GBT through their normal use of water depth. An additional consideration in this TGP value is the significant depths of the Columbia River (30 m or more) that are available to fish. However, as recommended below, supporting field studies should be conducted.

In addition to identifying the biological factors that may affect the derivation of Columbia River WUP performance measures, this review has provided a bibliography of existing Columbia River TGP analyses, database sources, and computer modeling tools that may be helpful in the WUP process. The TGP/GBT risk assessment analysis by R. L. & L. Environmental Services Ltd. (2002) may also provide useful information for the Columbia River WUP process.

### **TGP Effects on White Sturgeon**

Before considering the experimental programs required to support the derivation of Columbia River WUP performance measures, one additional issue needs to be examined. At the June 26<sup>th</sup> meeting in Castlegar, questions were raised as to the effects of TGP on white sturgeon in the Columbia River, especially in the vicinity of the Waneta Eddy where important spawning takes place. The first issue was to what extent discharges from the Hugh Keenleyside Dam affect TGPs in the Waneta Eddy. After some discussion, it was recognized that TGP levels in the eddy were mainly dependent on the TGPs associated with discharges from the Waneta Dam and to a lesser extent, discharges from the Brilliant Dam on the Kootenay River and discharges from the Hugh Keenleyside Dam. In terms of deriving performance measures for the Columbia River WUP, the TGPs produced by the Waneta

Dam are, at this time, unpredictable. This is due, in part, to the lack of information on TGPs produced by the dam's spillways and, in part, due to the unpredictability of TGPs produced by other dams upstream on the Pend d'Oreille River, especially those in the United States. The effect of the upstream dams is that their TGPs pass through the Waneta Dam by way of the generators and thereby influence the downstream TGP environment. Furthermore, the operations of the Waneta Powerplant are such that they do not allow long term predictions of spill discharges versus generator discharges. Finally, it is not clear to what extent the Keenleyside and Brilliant Dam discharges mix with the Waneta Dam discharges in the eddy. Based on these factors, it was concluded that the derivation of performance measures for the Columbia River WUP could not account for the effects of TGP produced by the Hugh Keenleyside Dam on the TGP environment of the Waneta Eddy.

A second issue is the limited amount of data on larval sturgeon susceptibility to elevated TGP. The only data that define a response to TGP are those of Counihan et al. (1998) in which larval sturgeon were exposed to two levels of TGP in a shallow (0.25 m deep) static water environment. These data show that bubbles form in the buccal cavity and nares at TGPs of 118% and 131%. Although the bubbles appeared within 15 minutes of exposure, mortality occurred only at the 131% TGP level, reaching 50% mortality in 13 days. However, the absence of signs of blood flow in the gills and in other components of the circulatory system, combined with distended pericardiums, suggested bubble formation in the cardiovascular system was the cause of death. Although bubbles form in the buccal cavity and may affect buoyancy and swimming ability, the actual cause of death at a TGP of 131% is the formation of bubbles in the cardiovascular system. On the other hand, if bubble formation in the buccal cavity affects swimming behaviour, predation may cause death before cardiovascular bubble formation proceeds to a lethal stage. This may be true regardless of the TGP. Although these data identify two responses to elevated TGP (i.e., bubbles in the buccal cavity and nares at TGPs of 118% and 131% and cardiovascular bubble formation at a TGP of 131%), they do not provide information that is directly applicable to the Columbia River WUP. This is because the direct mortality data are for a single TGP and the susceptibility of larval sturgeon (with buccal cavity bubbles) in the Columbia River is unknown.

A third issue was the question of the TGP exposures that white sturgeon larvae might experience in the Waneta Eddy and downstream. The first consideration was that, due to their size, the swimming velocities of larval sturgeon were low (millimeters per second) in relation to Columbia River water velocities (meters per second). Secondly, the flows in the Columbia River are turbulent (i.e., Reynolds numbers  $> 10^4$ ) and larval sturgeon positions in the water column are more a function of this turbulence than their swimming ability. Because of the general nature of turbulence, it is likely that the larvae are randomly cycled up and down in the water column repeatedly over time with periods both above and below the compensation depth. Given the depths of the Columbia River in the vicinity of the Waneta Eddy and downstream, it is unlikely that any significant portion of the larvae population is exposed to TGP near the water surface for extended periods (e.g., 15 minutes or greater). Any larvae that develop buccal cavity bubbles would eventually, through turbulence, be cycled to depths where the bubbles would collapse. Based on the above considerations, it was concluded that TGP was not likely to be a significant factor in the survival of larval white sturgeon in the Waneta Eddy and downstream. Apart from this consideration, it is not possible to assess TGP impacts on larval sturgeon in the context of the Columbia River WUP due to the unpredictability of river TGP levels at the Waneta Eddy and downstream.

## 5.0 Supporting WUP Experimental Studies

At present, there are no bioassay data that define GBT effects in Columbia River fish at TGPs of 115% and 120%. For this reason and the need to have experimental data to support a TGP threshold for the WUP performance measures, the Hildebrand live cage studies should be repeated as early as possible. However, the new studies should be conducted at a time when the Columbia River TGP is between 120% and 125%. Cages similar to those used by Hildebrand (i.e., 0.5 m deep) should be placed in the river below the Hugh Keenleyside Dam with the top of the cage 0.5 m below the water surface. The cages should be located where the river TGP can be measured continuously for a period of up to five days or until GBT mortality reaches 50%. Both juvenile and adult rainbow trout and mountain whitefish should be examined. Between 20 and 30 fish of each species and age class (80 – 120 fish total) should be included in the study. Multiple cages may be required to accommodate this number of fish. If it is not possible to get collection permits for this many fish, then the maximum number that permitting will allow should be used. Captured fish should be held in 100% TGP water before they are placed in the cages. Alternatively, they should be held at a depth of at least 3 m. Care should be taken to minimize stress on the fish from handling and caging. Water velocities through the cages should be kept to a minimum to avoid any swimming stress induced GBT (Stroud and Nebeker 1976). During the exposure period, dead fish should be collected as often as possible and examined for signs of GBT. A hand held magnifying scope (10x) should be used for examining external signs and for establishing the presence of bubbles in the filamental arteries of the gills. A severity of signs scale like that used by Hildebrand (1991) should be employed to grade the GBT signs observed. Fish that survive the five days of exposure should be sacrificed and examined for signs of GBT. If possible, the live cage studies should be completed before October 1, 2002. If this is not possible, they should be completed as soon as possible, river TGP conditions permitting. The key information required from this study is whether Columbia River rainbow trout and mountain whitefish can survive TGP levels of 120% to 125% without showing signs of GBT. If so, it will be easier to justify a threshold TGP of 120% for the WUP performance measures. If the fish in the cage studies do show signs of GBT, then an interim TGP threshold of 115% should be adopted until further field studies can be performed (see below).

In addition to the cage studies, it is recommended that the 1990 field studies of free swimming fish, conducted by Hildebrand (1991), be repeated during 2003 for a period of about three weeks and during a time when Columbia River TGPs are between 120% and 125%. This should be preceded by an experimental design study to identify methods of fish capture, methods of examination for signs of GBT, methods for protecting and holding captured fish, numbers of fish to be captured and examined, and opportunities for manipulating Hugh Keenleyside Dam operations to achieve desired TGP levels. These studies, although not in the current Columbia River WUP time frame, would provide further confirmation of the threshold TGPs that are used in the performance measure studies and perhaps confirm the results of the 2002 cage studies.

To adopt a lower WUP TGP threshold, such as 110%, would require that the contradictions and uncertainties in the data from the literature be resolved. This would require a laboratory study that repeated many of the conflicting experiments from the literature and clearly identified signs of GBT and the cause of mortality in the 110% - 115% TGP range. The experiments would have to be designed to assure that artifacts created by the experimental environment were not the cause of GBT signs and mortality. These experiments might also form a basis for revising the B.C. TGP guideline A to a higher level (e.g., 115%). Although, the uncertainty regarding the application of shallow water experimental results to river environments would remain, a higher guideline value would greatly

relieve the pressures to address TGP issues at lower levels (e.g. 110% to 115%). For example, the Hugh Keenleyside Dam north and south low-level ports are currently operated to minimize TGP regardless of the level. This was due, in part to the B.C. Guideline B, which restricted TGP levels to protect juvenile fish from the effects of swim bladder overinflation. With Guideline B removed and Guideline A raised to 115%, the north and south low level ports could be operated in a much less restrictive manner, giving more flexibility to the overall dam operations.

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