

# AIR GULPING<sup>1</sup> IMPROVES BLOOD OXYGEN TRANSPORT DURING AQUATIC HYPOXIA IN THE GOLDFISH *CARASSIUS AURATUS*<sup>1</sup>

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Buccal movements at the air-water interface causing surface water and air bubble inhalation ("surface breathing") and the resultant effects on blood O<sub>2</sub> transport have been quantified as a function of inspired water PO<sub>2</sub> in the goldfish, *Carassius auratus*. At 26 C, surface breathing occurred below a PO<sub>2</sub> of about 70 mmHg and increased to a peak of 180 surface breathing periods/h at PO<sub>2</sub> 20 mmHg. At this severe level of hypoxia, surface breathing resulted in a significant elevation of arterial PO<sub>2</sub> by 1.2 mmHg above that in control goldfish, which were not surface breathing. This very small rise in arterial PO<sub>2</sub> caused a sharp rise in arterial blood O<sub>2</sub> saturation from 16% to 33%, because of the high oxygen affinity (P<sub>50</sub> = 2.6 mmHg) and steep slope of the blood O<sub>2</sub> equilibrium curve. The net effect of surface breathing at the air-water interface during severe aquatic hypoxia is a 2.5 times increase in the arterial-venous O<sub>2</sub> saturation difference compared with strictly aquatic ventilation. Surface breathing with a nitrogen rather than air atmosphere significantly increased surface-breathing frequency and significantly decreased arterial blood oxygenation at a constant water PO<sub>2</sub>. Surface breathing, which includes both "air gulping" and irrigation of the gills with the surface water layer, is thus demonstrated to have a significant respiratory function in the goldfish.

## INTRODUCTION

Adaptations for air breathing that allow particular fish to survive in severely hypoxic waters have received much attention (see reviews by Johansen [1970]; Datta Munshi [1976]; Randall et al. [1981]). Yet, often the greatest proportion of the piscine fauna to be found in typical hypoxic environments are entirely dependent on aquatic respiration. In many respects, the adaptations that have allowed these solely aquatic fish to survive hypoxic exposure are as fascinating as those of the air-breathing fish.

The goldfish, *Carassius auratus*, is an aquatic cypriniforme that thrives in habitats subject to extended periods of low oxygen. A large oxygen extraction from a high branchial water flow (Dejours, Armand, and Verriest 1968) and hemoglobin with very high oxygen affinity (present study) apparently aid oxygen uptake and transport under such conditions. How-

ever, as hypoxia becomes progressively more severe, the metabolic cost of maintaining a convective flow of hypoxic water over the gills eventually exceeds the benefits of the oxygen obtained. Under severe conditions of oxygen depletion the goldfish increasingly depends upon anaerobic metabolic pathways and may even produce CO<sub>2</sub>, ammonia, and ethanol rather than lactate as metabolic end products (see Hochachka 1980). As with any anaerobic pathway, there is an attendant drop in high-energy phosphate compound yield compared with continued aerobic processes.

The air-water interface may provide partial means for the goldfish to avoid hypoxia in the underlying water column and thus avoid either the high cost of maintaining aerobiosis in hypoxic water or the lowered energy yield of switching to anaerobic metabolism. Not only do the top few millimeters or centimeters of severely hypoxic or even anoxic natural bodies of water have an appreciable oxygen content (Carter 1931; Hutchinson 1957; Dusant 1963; Wetzel 1975; Dehadrai and Tripathi 1976; Burggren, unpublished), but the air itself may also be potentially important as a direct source of oxygen for aquatic fish not generally perceived as "air breathers," as suggested by the very common behavior

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of air gulping at the surface exhibited by the goldfish, bullhead catfish, and other strictly aquatic species (see Lewis 1970; Steen 1971; Kramer and McClure 1982). In spite of the long-standing speculation about its benefits to respiration, little data on the physiological effects of "air breathing" in the goldfish or any other aquatic fish lacking specific morphological structures for air breathing are available. The present study was designed to quantify the use of the air-water interface by *C. auratus* in hypoxic water and the effects of this behavior on in vivo blood oxygenation.

#### MATERIAL AND METHODS

Experiments were performed on a total of 17 goldfish (body mass =  $178 \pm 87$  g, mean  $\pm$  SD) captured from the university pond in Amherst, Massachusetts. All animals were acclimated for 1 wk–1 mo at 25–26 C before use. Because nonventilatory behaviors resembling surface breathing are exhibited by long-term captive goldfish inadvertently trained to seek floating food items, great care was taken to use only naive, wild goldfish fed during the acclimatory period by dense trout pellets deposited on the bottom of the holding tank.

For experimental purposes, individuals were placed in a 10-liter chamber containing dechlorinated tap water at 25–26 C. The oxygen partial pressure ( $PO_2$ ) of the water was closely regulated by continuously pumping water from the chamber through a gas-exchange column bubbled with various nitrogen-air mixtures. Water  $PO_2$  was constantly monitored by using a small peristaltic pump to draw water from the open tip of a catheter in the chamber through a cuvette containing an Instrumentation Laboratory (I.L.)  $O_2$  electrode connected to an I.L. 113 meter. A magnetic stirring bar gently spinning on one wall of the chamber completely mixed the water column, providing a constant  $PO_2$  at nearly all depths. Of particular interest, however, was the thickness of the thin oxygenated surface layer of water that would exist at the top of even a hypoxic water column covered by air. This thickness was determined by using a micromanipulator to carefully position the tip of the sampling catheter at various

levels below and above the air-water interface (fig. 1). Even when  $PO_2$  in the chamber was as low as 15–20 mmHg, the oxygenated surface layer of water was much thinner than  $\frac{1}{2}$  mm.

In the first series of experiments, individual goldfish were given 24 h to acclimate to the experimental apparatus (inspired  $PO_2$ ,  $PI_{O_2}$ , initially 140–150 mmHg), after which the water  $PO_2$  was progressively reduced to approximately 20 mmHg, at a rate of decrease of about 2 mmHg/min. Exposure to hypoxic conditions in this and all other experiments was confined to under 2 h because of recent experimental evidence indicating that the  $O_2$  transport characteristics of the blood of cyprinid fish may begin to change after as little as 2 h of hypoxic exposure (Lykkeboe and Weber 1978). Gill ventilation and surface-breathing frequency were counted by direct observation at  $PI_{O_2}$  increments of 10 mmHg. Surface breathing, which was always accompanied by an easily recognized orientation of the fish to the surface (fig. 1), was defined as ventilatory movements that occurred when the lower jaw was within 1 mm of the water surface and the upper jaw was air exposed, including those ventilatory movements when the entire mouth was elevated above the water surface. Single ventilatory cycles during surface breathing were often erratic and punctuated with frequent coughing, making them difficult to count individually. Instead, the frequency of *periods* of surface breathing,

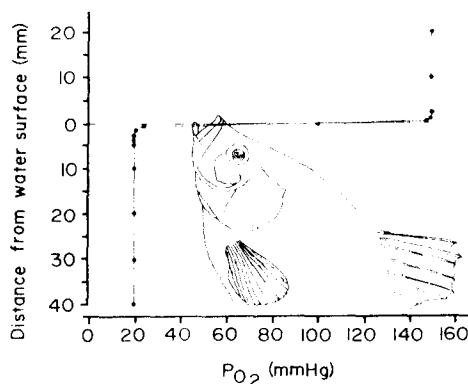


FIG. 1.—A  $PO_2$ -depth profile through a stirred hypoxic water column and the air above the experimental chamber. Also indicated is the posture of a goldfish actively engaged in surface breathing. See text for details.

ation as a complete series of uninterrupted ventilatory cycles that were wholly at or above the water surface as described above, was recorded.

In the second series of experiments, goldfish were anesthetized in tricaine methanesulfonate, buffered to pH 7.5. A 2-cm incision over the lateral line and 2–4 cm anterior of the base of the tail was made to expose the caudal artery and caudal vein below the vertebral column (Cech and Rowell 1976). A polyethylene 10 cannula filled with heparinized saline was implanted into either the dorsal aorta or caudal vein and secured in place with cyanoacrylate tissue adhesive. The incision was closed with interrupted sutures, and the fish was allowed to recover overnight in air-saturated water in the experimental apparatus. Implanted catheters generally remained patent for 2–4 days, during which no evidence of tail necrosis or other signs of interrupted circulation developed.

Blood samples (120  $\mu$ l) were drawn during normoxic conditions and at two levels of aquatic hypoxia ( $P_{iO_2}$  approximately 30 and 18 mmHg). Samples were taken both when normal surface breathing had occurred consistently for at least 2 min and when it was prevented by suspending a wire screen 1 cm below the water surface. Arterial and venous blood  $P_{O_2}$ , and in some instances  $P_{CO_2}$  and pH, were determined on 100- $\mu$ l samples with an Instrumentation Laboratories  $\mu$ 13 blood gas analyzer. Blood oxygen content was measured using the method of Tucker (1967) and a radiometer O<sub>2</sub> electrode and pHM 71 meter.

In a final series of observations, X-ray plates (1/30–1/10-s exposures) were made of ventilatory movements in goldfish exposed to various  $P_{iO_2}$ 's. Exposures were made during strictly aquatic breathing and during surface-breathing periods to assess the extent of air movement with the buccal cavity.

Treatment effects on blood oxygenation parameters were assessed by ANOVA, while differences between individual means were assessed with Student's independent *t*-test, using a significance level of .05. All data reported are mean values  $\pm$  1 SD.

## RESULTS

### VENTILATORY PATTERNS

Gill ventilation began to increase almost immediately with decreasing  $P_{iO_2}$ , rising 140% over control values at a  $P_{iO_2}$  of 20 mmHg (fig. 2A). Large increases in ventilation stroke volume as well as frequency usually develop in hypoxic fish (Shelton 1970), so total ventilation volume doubtlessly rose by an even greater extent.

Although occasional ventilatory movements occurred within 0.5 cm of the surface, periods of distinct surface breathing were very infrequent in goldfish in air-saturated water (fig. 2B). Below a  $P_{iO_2}$  of about 70 mmHg, however, periods of surface breathing became increasingly frequent, rising to 180 periods/h at  $P_{iO_2}$  20 mmHg. It should be emphasized that a single surface-breathing period at a higher  $P_{iO_2}$  usually consisted of only 1–2 ventilatory cycles, whereas at a lower  $P_{iO_2}$  each surface-breathing period often consisted of 10–40 ventilatory cycles with the mouth at the air-water interface and in a few goldfish was the only form of gill ventila-

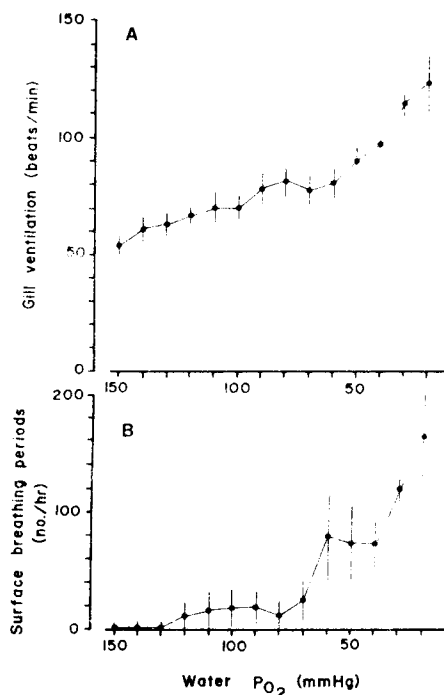


FIG. 2.—The relationships between gill ventilation (A) and surface-breathing periods (B) with variation in inspired water  $P_{O_2}$ . Mean values  $\pm$  SD from eight goldfish are plotted.

tion of severe hypoxia. The shift from strictly aquatic to surface air breathing during hypoxia was thus even greater than revealed simply by the increasing frequency of surface-breathing periods.

X-ray and visual observation revealed that depression of the buccal floor during surface breathing resulted in a large bubble of air entering the dorsal region of the buccal cavity, along with a variable amount of water from the surface layers. Forceful ventilatory movements frequently caused propulsions of the air bubble back and forth within the buccal cavity, often to the extent that the bubble was momentarily thrust against the anterior aspects of the branchial arches before returning toward the mouth. Water and air within the buccal cavity thus were constantly mixed during surface breathing. When surface breathing was suspended and a horizontal posture for normal aquatic breathing was resumed, a small air bubble was often trapped in the upper regions of the buccal cavity, eventually expelled underwater by a forceful "cough."

BLOOD HEMOGLOBIN-OXYGEN AFFINITY

Figure 3 presents the in vivo Hb-O<sub>2</sub> equilibrium curve determined by plotting in vivo blood P<sub>O<sub>2</sub></sub> versus blood O<sub>2</sub> content from seven goldfish. Blood oxygen capacity varied from 6.0 to 9.3 (mean = 7.6 ± 1.4) ml O<sub>2</sub>/100 ml blood, so all oxygen con-

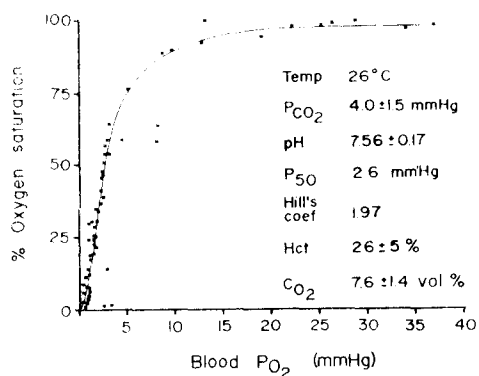


FIG. 3.—Relationship between blood P<sub>O<sub>2</sub></sub> and percentage of blood oxygen saturation in *C. auratus*. Both arterial and venous samples from seven goldfish are plotted. Also indicated are the mean conditions of P<sub>CO<sub>2</sub></sub>, pH, and temperature (see text for further details). The single curve describing the Hb-O<sub>2</sub> equilibrium curve has been drawn by eye.

tents were expressed in the conventional fashion as a percentage of oxygen saturation to allow comparison between experimental conditions. The P<sub>CO<sub>2</sub></sub> and pH of arterial blood were not significantly different ( $P > .1$ ) from venous blood, nor were these two variables different among the various experimental conditions. Therefore, all experimental data have been included in figure 3 and the mean P<sub>CO<sub>2</sub></sub> and pH indicated.

The in vivo Hb-O<sub>2</sub> affinity of the blood of the goldfish under the stated mean condition of temperature and pH was extremely high for goldfish, with half O<sub>2</sub> saturation (P<sub>50</sub>) occurring at a blood P<sub>O<sub>2</sub></sub> of only 2.6 mmHg.

BLOOD OXYGEN PARTIAL PRESSURES

Without surface breathing.—Table 1 and figure 4 present data for arterial and

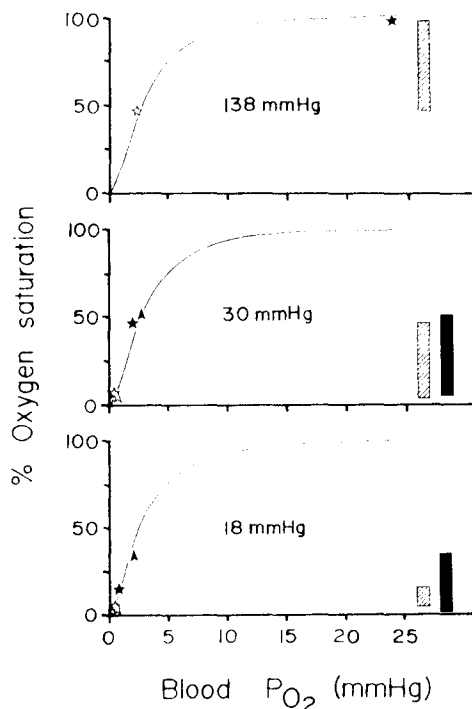


FIG. 4.—Effects of surface breathing at three different levels of P<sub>iO<sub>2</sub></sub> on arterial (solid symbols) and venous (open symbols) blood P<sub>O<sub>2</sub></sub> and percentage of oxygen saturation. The mean values of measured blood P<sub>O<sub>2</sub></sub>'s (see table 1) have been transposed onto the in vivo Hb-O<sub>2</sub> equilibrium curve redrawn directly from fig. 3. The vertical bars at side of figure portray the arterial-venous difference in oxygen saturation at each P<sub>iO<sub>2</sub></sub> both in the absence (striped bars) and presence (solid bars) of surface breathing.

TABLE 1  
EFFECTS OF HYPOXIA AND PRESENCE OR ABSENCE OF SURFACE BREATHING ON IN VIVO BLOOD OXYGENATION OF "CARASSIUS AURATUS" AT 26 C

Condition, Surface-breathing Status, and No. of Samples	P <sub>I</sub> O <sub>2</sub> (mmHg)	P <sub>A</sub> O <sub>2</sub> (mmHg)	P <sub>V</sub> O <sub>2</sub> (mmHg)	% O <sub>2</sub> Saturation	Arterial-Venous O <sub>2</sub> Difference (% O <sub>2</sub> Saturation)
Normoxia.					
Surface breathing absent:					
12.....	139±9	23.8±11.9	.....	97±4	52
22.....	136±7	.....	2.2±1.6	45±17	
Intermediate hypoxia:					
Surface breathing absent:					
8.....	31±1	1.8±1.2	.....	47±11	43
10.....	33±8	.....	.7±.1	4±4	
Surface breathing present:					
6.....	28±2	2.6±.3	.....	51±13	45
6.....	31±1	.....	.2±.1	6±3	
Severe hypoxia:					
Surface breathing absent:					
5.....	18±3	.9±.6	.....	16±4	11
5.....	19±1	.....	.2±.3	5±8	
Surface breathing present:					
8.....	19±4	2.1±1.1	.....	33±11	29
5.....	18±1	.....	.3±.5	4±3	

NOTE.—Mean values ± SD are given. Data are from seven goldfish.

venous blood PO<sub>2</sub> in *Carassius auratus* under different experimental conditions. The PO<sub>2</sub> of arterial blood (P<sub>A</sub>O<sub>2</sub>) at a P<sub>I</sub>O<sub>2</sub> of 140 mmHg was only 24 mmHg, which is low compared with most other teleosts but similar to that reported for the carp, *Cyprinus carpio* (Itazawa and Takeda 1978). However, the Hb-O<sub>2</sub> affinity of the blood of the goldfish is so high that arterial blood was fully oxygen saturated. Venous blood was only 45% O<sub>2</sub> saturated, indicating that about half of the arterial blood oxygen store was extracted in transit through the tissue capillary beds.

At a P<sub>I</sub>O<sub>2</sub> of about 30 mmHg and with strictly aquatic gill ventilation, P<sub>A</sub>O<sub>2</sub> fell greatly, and arterial O<sub>2</sub> saturation was reduced to only 47% (table 1, fig. 4). However, the small drop in venous PO<sub>2</sub> occurred over the steepest region of the Hb-O<sub>2</sub> equilibrium curve, and venous oxygen saturation at this level of hypoxia fell to only 4%. The venous oxygen reserve was thus heavily exploited, and as a consequence there was no significant change ( $P > .1$ ) in the arterial-venous O<sub>2</sub> saturation difference between that evident at a P<sub>I</sub>O<sub>2</sub> of 140 and that at 30 mmHg.

When P<sub>I</sub>O<sub>2</sub> fell from 30 to 18 mmHg in the presence of aquatic ventilation alone, severe disruption of blood oxygenation oc-

curred. Blood leaving the gills was only 16% oxygen saturated, and the arterial-venous oxygen saturation difference fell to just 11%. Goldfish were apparently very tolerant of this short-term exposure to serve hypoxia and invariably recovered when the P<sub>I</sub>O<sub>2</sub> was returned to air-saturated levels.

*With surface breathing.*—At a P<sub>I</sub>O<sub>2</sub> of 30 mmHg, unrestricted goldfish spent a major portion of their ventilatory activity on periods of surface breathing (see fig. 2). However, with the exception of a significant ( $P < .005$ ) but quite small rise in P<sub>V</sub>O<sub>2</sub>, there were no significant changes from blood variables measured in goldfish relying strictly on aquatic ventilation (table 1, fig. 3).

At the lowest P<sub>I</sub>O<sub>2</sub>, however, major differences emerged between goldfish able to surface breathe and those that were not. The P<sub>A</sub>O<sub>2</sub> was significantly higher ( $P < .025$ ) during surface breathing, and consequently arterial blood reached an oxygen saturation twice as great as that achieved with aquatic ventilation only. The arterial-venous oxygen difference was reduced from that in normoxia by only one-half and was 2.5 times greater when compared with strictly aquatic ventilation at the same low inspired oxygen level.

These data strongly indicate that surface breathing makes an important contribution to blood oxygenation. To test further this hypothesis, the experiments were repeated for six goldfish in water with a  $P_{O_2}$  of about 22 mmHg and with unrestricted surface access. In the first instance the atmosphere over the water was air, and after recording experimental variables, oxygen-free nitrogen gas was substituted for air. Gill ventilation frequency was unchanged in the two conditions, but surface breathing increased by about 50% when nitrogen rather than air was present (table 2). Concomitantly,  $PA_{O_2}$  fell by a third, and the oxygen saturation of arterial blood fell by half to values very similar to those in goldfish denied surface access while in severely hypoxic water.

#### DISCUSSION

Surface breathing can clearly make a substantial contribution to arterial blood oxygenation in *Carassius auratus* subjected to aquatic hypoxia and becomes increasingly important as hypoxia becomes more severe. At a  $PI_{O_2}$  of 18 mmHg, for example, the rise of  $PA_{O_2}$  afforded by this respiratory behavior is not large, but it has profound effects because it acts over the steepest region of the Hb- $O_2$  equilibrium curve, where small increases in  $PA_{O_2}$  produce major increases in blood oxygen content. Clearly, air breathing does not raise the percentage of oxygen saturation of

arterial blood to normoxic levels, or even to those levels produced by aquatic ventilation alone at a higher  $PO_2$  (fig. 4). Cardiac output was not measured, so the net effect on tissue  $O_2$  uptake cannot be estimated. However, in the presence of surface breathing at  $PI_{O_2}$  18 mmHg, the arterial-venous oxygen saturation difference was reduced by only half from normoxic levels, compared with a reduction to one-fifth when surface breathing is absent. Even if a reduction in cardiac output occurred, the difference in the aerobic scope permitted by surface breathing and strictly aquatic breathing lower in the water column during aquatic hypoxia must be considerable.

Description of this respiratory behavior as surface breathing rather than "air breathing" is intentional (see Kramer and Mehegan [1981] for other descriptive terms that have been used). The oxygenated, unstirred water layer was effectively reduced to less than  $\frac{1}{2}$  mm in thickness (fig. 1), but it is almost certain that such an oxygenated layer, albeit very thin, still existed in the experimental apparatus, as it does in natural bodies of hypoxic water (Carter 1931; Hutchinson 1957; Dusan 1963; Wetzel 1975). In addition to the inspiration of large air bubbles, some of this thin surface layer, along with underlying hypoxic water, appeared to be drawn also into the buccal cavity during surface breathing. Thus the respiratory im-

TABLE 2  
EFFECT OF AIR VERSUS NITROGEN ATMOSPHERE DURING SEVERE AQUATIC  
HYPOXIA ON FREQUENCY OF GILL VENTILATION AND SURFACE-  
BREATHING PERIODS AND ON ARTERIAL BLOOD OXYGENATION IN  
SIX "CARASSIUS AURATUS" AT 26 C

	Air Atmosphere	Nitrogen Atmosphere	% Change
$P_{O_2}$ of water (mmHg) . . . . .	23 ± 4	21 ± 6	...
Gill ventilation frequency (beats/min) . . . . .	116 ± 22	120 ± 20	3% increase ( $P > .1$ , NS)
Surface-breathing frequency (periods/h) . . . . .	130 ± 65	197 ± 34	52% increase ( $P < .05$ )
$PA_{O_2}$ (mmHg) . . . . .	2.1 ± 1.1	1.4 ± .7	33% decrease ( $P < .05$ )
$O_2$ saturation of arterial blood (% of total $O_2$ capacity) . . . . .	34 ± 11	16 ± 15	53% decrease ( $P < .05$ )

NOTE: Mean values ± 1 SD are given.

the oxygenated surface layer remote to the inspired air bubble cannot be quantified. However, given the large size of the air bubble, its continual mixture and contact with inspired buccal water (as revealed by X-ray analysis), and its frequent renewal, it would appear likely that the gas phase makes a large contribution to the effectiveness of surface breathing. The air bubble remains within the buccal cavity and appears to make little if any direct contact with the respiratory surface areas of the gills, suggesting that it is the oxygenation of buccal water subsequently irrigating the gills that is directly responsible for elevating  $P_{A_{O_2}}$  in severely hypoxic conditions.

The air-water interface thus provides *C. auratus* with a means for partially escaping from the detrimental effects of severe aquatic hypoxia on oxygen transport by the blood. While the anaerobic capacities of many fish are extraordinary (see Blazka 1958; Hochachka 1980), aerobic metabolism is clearly more effective in terms of ATP yield. This may favor the selection of even quite energetically expensive behavior that helps to support continued aerobic metabolism, as does surface breathing. However, surface breathing is not without negative features, or perhaps many other

fully aquatic fish might be expected to regularly use this respiratory behavior. For example, the metabolic cost of surface breathing in the goldfish or any of the other fish that do display this respiratory behavior is unknown but may be higher than suspected (Kramer and Mehegan 1981). Certainly the repeated trips to the air-water interface by air-breathing fish are known to have a large impact on their daily energy budget, even when in oxygenated water (Arunachalam, Vivekanandan, and Pandian 1976; Pandian and Vivekanandan 1976; Kramer and McClure 1981). Also, movement in the top regions of the water column for respiratory purposes may greatly increase the risk of alerting aerial or terrestrial predators, particularly for brightly colored fish like *C. auratus*.

In summary, a combination of inspired atmospheric air and oxygenated surface water is used by goldfish to help maintain blood oxygen transport when in severely hypoxic water. Such surface breathing has been observed in other fish that migrate toward the upper regions of a hypoxic water column (Kramer and Mehegan 1981) and probably is of similar importance in supporting continued aerobic metabolism.

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