METABOLISM OF THE PERFUSED SWIMBLADDER OF THE
EUROPEAN EEL: OXYGEN, CARBON DIOXIDE, GLUCOSE
AND LACTATE BALANCE

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Accepted 7 February 1989

Summary

We have measured the metabolic activity in the vascularly isolated, saline-
perfused swimbladder of the eel (Anguilla anguilla) in order to investigate the
pathways for CO2 formation in the gas gland tissue. Concentrations of O2, CO2,
glucose and lactate were measured in the arterial inflow and venous outflow of the
swimbladder, and metabolic rates were calculated by the direct Fick principle.

1. Total CO2 production, averaging 55.8 nmol min⁻¹, was about 4.6 times the
O2 consumption (mean 12.0 nmol min⁻¹). This suggests that only about 22% of
the CO2 is formed by aerobic glucose metabolism.

2. CO2 formation from HCO3⁻ or CO2 washout does not appear to be
significant in our experiments with steady perfusion of a saline containing a low
level of HCO3⁻.

3. The ratio of lactate production to glucose uptake averaged 1.2, indicating
that only 60% of the glucose is converted to lactate. Since only 1-2% of the
glucose was found to be oxidized (2 nmol min⁻¹), the extra glucose appears to be
anoxidatively metabolized to CO2.

4. The anoxidative CO2 formation appears to be of functional importance for
producing the high gas partial pressures of both CO2 and O2 which are required for
secretion of these gases into the swimbladder.

Introduction

Many teleost fishes have developed a gas-filled swimbladder which enables them
to keep neutral buoyancy at varying water depths. It is generally accepted that the
gas enters the bladder from the blood by diffusion, and it is assumed that the high
gas partial pressures in the blood perfusing the swimbladder are generated by
acidification of the blood in the gas gland in conjunction with the countercurrent
gas exchange in the rete mirabile (Steen, 1970; Fänge, 1983). In fact, the gas gland
is known to produce lactic acid even at high O2 levels. This acid enhances, via the
Bohr and Root effects, the O2 partial pressure and, by the conversion of HCO3⁻ to
CO2, the CO2 partial pressure. The formation of lactate by gas gland cells has been
demonstrated both in tissue preparations (Ball et al. 1955; D'Aoust, 1970; Deck,

Key words: fish, gas secretion, metabolism, swimbladder.
1970) and in vivo (Kuhn et al. 1962; Steen, 1963b; Enns et al. 1967). In this model, CO₂ in the swimbladder would derive either from oxidative metabolism or from HCO₃⁻.

Oxidative metabolism has been estimated to contribute only 5% of the total CO₂ formed (Wittenberg et al. 1964). Ball et al. (1955), Wittenberg et al. (1964) and D’Aoust (1970) found that most, if not all the CO₂ was liberated from bicarbonate, presumably by lactic acid. This assumption neglects, however, the presence of nonbicarbonate buffers, which would buffer some of the H⁺ released from lactic acid, thus reducing the amount of H⁺ available for conversion of HCO₃⁻ to CO₂. Therefore, a lactate/CO₂ ratio of unity, found by Ball et al. (1955) and D’Aoust (1970), can only be explained if CO₂ derives from sources other than bicarbonate.

Such a source could be the decarboxylation reaction of the pentose phosphate shunt, which yields CO₂ from carbohydrates without consuming O₂. Boström et al. (1972) measured high activities of enzymes characteristic of the pentose phosphate shunt in the gas gland tissue of the cod, and increases in CO₂ content in blood passing through the gas gland have indeed been measured directly (Steen, 1963b; Pelster et al. 1988b). The contribution of CO₂ via this route has not been estimated yet.

In the present study, we have measured the metabolic activity in the saline-perfused eel swimbladder to evaluate the contributions of the various pathways to CO₂ production. The saline-perfused preparation was preferred to the more physiological, blood-perfused swimbladder, as it more easily allows measurement of the perfusion rate and quantitative estimation of metabolic rates.

Materials and methods

Specimens of the freshwater-adapted European eel (Anguilla anguilla; body mass 500–1100 g) were purchased from a local supplier and kept in a freshwater aquarium at 12–14°C until the experiment.

Animal preparation and experimental set-up

The animals were anaesthetized by adding MS 222 (0.1 g L⁻¹) to the water and were then placed into an ‘eel holder’, similar to that used by Steen (1963a). The gills were irrigated with well-oxygenated tap water (flow rate 4.5 L min⁻¹), containing MS 222 at a concentration of approximately 0.05 g L⁻¹, a level sufficient to keep the animal anaesthetized with stable ventilatory activity.

Preparation of animals was essentially the same as described elsewhere (H. Kobayashi, B. Pelster & P. Scheid, in preparation). Briefly, the body wall was opened ventrally, the swimbladder was exposed and carefully freed from connective tissue. The duct connecting the absorption and secretion part of the bladder was tied off, and small blood vessels and anastomoses bypassing the rete mirabile were carefully ligated without damaging rete vessels or the swimbladder wall. The gas secretion rate was too low to be measured accurately by a catheter inserted
CO₂ production in swimbladder tissue

Fig. 1. Diagram of the two retia in the eel swimbladder. The arterial inflow (ai) and venous outflow vessels (ve) at the heart pole were occlusively cannulated for perfusion. For serial perfusion (type S), ai of one and ve of the other rete were blocked (see Materials and methods).

into the swimbladder (less than 0.1 ml h⁻¹). All experiments were carried out at 20–22°C.

The artery and vein at the heart pole (ai and ve in Fig. 1) were occlusively cannulated using PE 50 and PE 100 catheters, respectively, and the rete vessels were perfused with saline solution at 0.13–0.40 ml min⁻¹ using an infusion pump (Precidor, Type 5003, Infors AG, Basel, Switzerland). This solution was a heparinized (100 i.u. ml⁻¹) Ringer’s solution which contained (in mmol l⁻¹) NaCl, 129; KCl, 5; MgSO₄, 0.9; CaCl₂, 1.1; glucose, 15; insulin, 8 i.u. The colloid osmotic pressure was adjusted to that of eel plasma by adding 5 g l⁻¹ Dextran FP 70 (Serva, Heidelberg, FRG). The saline contained no bicarbonate and was equilibrated with air so as to remove nearly all CO₂.

**Experimental protocol**

Samples of the perfusate were collected at the venous outlet (ve), and their composition was compared with that of the perfusate at the inflow (ai). Venous collection was about every 30 min for 3–4 h. For calculating metabolic rates from perfusion rate and concentrations in inflow and outflow, steady-state conditions have to prevail. Since countercurrent exchange in the rete during normal flow conditions (perfusion type C) retards attainment of steady state, a serial type of rete flow (type S) was achieved in some experiments by occluding the afferent artery of one and the efferent vein of the other rete at the heart pole (Fig. 1), this flow type enhanced attainment of steady state. Measurements were made in these experiments during both type C and type S flow.

**Analytical procedures**

Saline samples were analysed for oxygen content (CO₂), using the method of
Tucker, 1967) and total CO₂ content (C_{CO₂}; using the method of Cameron, 1971) immediately after collection. For analysis of glucose and lactate concentrations, part of the sample was deproteinized with perchloric acid and neutralized with K₂CO₃ or KOH. Employing this procedure, no extrusion of proteins from the tissue into the perfusate was detected. The assays were performed enzymatically as outlined by Bergmeyer (1974).

Calculations

The rates of production of CO₂ (M_{CO₂}) and lactate (M_{La}) and the rates of consumption of O₂ (M_{O₂}) and glucose (M_{Glu}) were calculated by mass balance from the perfusion rate (Q) and the concentration difference between inflow and outflow. Student's t-tests were used to test for statistical significance, and P < 0.01 was accepted as the limit of significance.

Results

At the onset of perfusion, there were high concentrations of lactate and CO₂ in the venous outflow (ve) which gradually decreased to a steady-state level. For both substances this was higher than the arterial level (ai; Fig. 2). For calculation of metabolic rates only steady-state values were taken into account.

Table 1 shows concentrations of O₂, CO₂ and lactate in inflow (ai) and outflow (ve) of the rete during perfusion at two flow rates when there was no countercurrent flow (type S). The observed differences in these concentrations

![Graph showing concentrations of total CO₂ (open circles, continuous line) and lactate (closed circles, dashed line) in the venous outflow (ve) of a saline-perfused swimbladder (perfusion type C) plotted against time from onset of perfusion. Concentrations in the perfusate at the entrance to the rete (ai) are presented as horizontal lines. Values of ve for t > 120 min were averaged in this experiment to yield the steady-state value from which metabolic activity was calculated.](chart.png)
Table 1. Arterial inflow (ai) and venous outflow (ve) concentrations of $O_2$ ($C_{O_2}$), $CO_2$ ($C_{CO_2}$) and lactate measured in saline-perfused swimbladders at two perfusion rates during type S perfusion (serial flow, no countercurrent).

<table>
<thead>
<tr>
<th></th>
<th>$\dot{Q} = 0.2 \text{ ml min}^{-1} \ (N = 2)$</th>
<th>$\dot{Q} = 0.4 \text{ ml min}^{-1} \ (N = 4)$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$ai$</td>
<td>$ve$</td>
</tr>
<tr>
<td>$C_{O_2}$ (mmol l$^{-1}$)</td>
<td>0.22 ± 0.01</td>
<td>0.14 ± 0.01</td>
</tr>
<tr>
<td>$C_{CO_2}$ (mmol l$^{-1}$)</td>
<td>0.15 ± 0.05</td>
<td>0.54 ± 0.41</td>
</tr>
<tr>
<td>[Lactate] (mmol l$^{-1}$)</td>
<td>0.07 ± 0.04</td>
<td>0.83 ± 0.22</td>
</tr>
</tbody>
</table>

$N$, number of preparations.
Table 2. Rates of oxygen consumption ($\dot{M}_{O_2}$) and CO$_2$ excretion ($\dot{M}_{CO_2}$) in saline-perfused swimbladder tissue and the corresponding respiratory exchange ratio, $R$

<table>
<thead>
<tr>
<th>Expt no.</th>
<th>Perfusion type</th>
<th>$\dot{M}_{O_2}$ (nmol min$^{-1}$)</th>
<th>$\dot{M}_{CO_2}$ (nmol min$^{-1}$)</th>
<th>$R$</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>C</td>
<td>13.6</td>
<td>60.0</td>
<td>4.4</td>
</tr>
<tr>
<td>6</td>
<td>C†</td>
<td>12.0</td>
<td>82.0</td>
<td>6.8</td>
</tr>
<tr>
<td>7</td>
<td>C</td>
<td>3.7</td>
<td>10.4</td>
<td>2.8</td>
</tr>
<tr>
<td>8</td>
<td>C</td>
<td>15.5</td>
<td>66.0</td>
<td>4.3</td>
</tr>
<tr>
<td>8</td>
<td>S</td>
<td>13.4</td>
<td>26.2</td>
<td>2.0</td>
</tr>
<tr>
<td>9</td>
<td>S</td>
<td>17.6</td>
<td>130.8</td>
<td>7.4</td>
</tr>
<tr>
<td>11</td>
<td>C</td>
<td>11.2</td>
<td>48.4</td>
<td>4.3</td>
</tr>
<tr>
<td>11</td>
<td>S</td>
<td>10.0</td>
<td>38.8</td>
<td>3.9</td>
</tr>
<tr>
<td>13</td>
<td>C</td>
<td>8.8</td>
<td>63.6</td>
<td>7.2</td>
</tr>
<tr>
<td>13</td>
<td>S</td>
<td>15.6</td>
<td>55.6</td>
<td>3.6</td>
</tr>
<tr>
<td>13</td>
<td>C</td>
<td>10.8</td>
<td>32.4</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Mean ± s.d.  

12.0 ± 3.8*  
55.8 ± 32.1*  
4.5 ± 1.8**

Perfusion type C, perfusion with countercurrent flow; type S, serial perfusion, no countercurrent flow.

* Significantly different from zero ($P < 0.01$).
** Significantly different from unity ($P < 0.01$).
† Only one rete mirabile perfused.

between ai and ve were clearly dependent on the flow rate. Doubling the flow rate resulted in a reduction of the (ve-ai) differences by a factor of about 2.

The rates of O$_2$ consumption ($\dot{M}_{O_2}$) and CO$_2$ production ($\dot{M}_{CO_2}$), calculated from concentration differences in individual experiments, are listed in Table 2. There were no differences between the flow types. In each experiment the CO$_2$ production rate by far exceeded the O$_2$ consumption rate. The resulting values of the respiratory exchange ratio, $R = \dot{M}_{CO_2}/\dot{M}_{O_2}$, are significantly above unity. The rates of glucose uptake ($\dot{M}_{Glu}$) and lactate release ($\dot{M}_{La}$) are presented in Table 3. On average, the rate of glucose consumption was higher than the rate of lactate production.

**Discussion**

For a quantitative evaluation of the metabolic activities of the gas gland its perfusion rate had to be known. Since blood flow rate cannot easily be obtained in the eel swimbladder without altering its function, we had to apply the technique of saline perfusion of the vascularly isolated tissue. The CO$_2$ and lactate values measured in this study, however, are similar to values that we obtained earlier by non-obstructive blood collection in intact eel swimbladder (Pelster et al. 1988b). We, therefore, assume the values measured in this study to be representative for eel swimbladder under physiological conditions.
Table 3. Rates of lactate production ($\dot{M}_{La}$) and glucose consumption ($\dot{M}_{Glu}$) in saline-perfused swimbladder tissue

<table>
<thead>
<tr>
<th>Expt no.</th>
<th>Perfusion type</th>
<th>$\dot{M}_{La}$ (nmol min$^{-1}$)</th>
<th>$\dot{M}_{Glu}$ (nmol min$^{-1}$)</th>
<th>$\dot{M}<em>{La}/\dot{M}</em>{Glu}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>C</td>
<td>109</td>
<td>36</td>
<td>3.0</td>
</tr>
<tr>
<td>2</td>
<td>C</td>
<td>86</td>
<td>44</td>
<td>1.9</td>
</tr>
<tr>
<td>3</td>
<td>C</td>
<td>208</td>
<td>83</td>
<td>2.5</td>
</tr>
<tr>
<td>4</td>
<td>C</td>
<td>451</td>
<td>204</td>
<td>2.2</td>
</tr>
<tr>
<td>5</td>
<td>C</td>
<td>106</td>
<td>112</td>
<td>1.0</td>
</tr>
<tr>
<td>6</td>
<td>C</td>
<td>117</td>
<td>432</td>
<td>0.3</td>
</tr>
<tr>
<td>7</td>
<td>C†</td>
<td>59</td>
<td>78</td>
<td>0.8</td>
</tr>
<tr>
<td>7</td>
<td>C</td>
<td>19</td>
<td>189</td>
<td>0.1</td>
</tr>
<tr>
<td>8</td>
<td>C</td>
<td>99</td>
<td>242</td>
<td>0.4</td>
</tr>
<tr>
<td>8</td>
<td>S</td>
<td>115</td>
<td>112</td>
<td>1.0</td>
</tr>
<tr>
<td>8</td>
<td>C</td>
<td>62</td>
<td>180</td>
<td>0.4</td>
</tr>
<tr>
<td>9</td>
<td>S</td>
<td>189</td>
<td>158</td>
<td>1.2</td>
</tr>
<tr>
<td>9</td>
<td>C</td>
<td>46</td>
<td>250</td>
<td>0.2</td>
</tr>
<tr>
<td>10</td>
<td>C</td>
<td>150</td>
<td>276</td>
<td>0.5</td>
</tr>
<tr>
<td>10</td>
<td>S</td>
<td>148</td>
<td>400</td>
<td>0.4</td>
</tr>
<tr>
<td>10</td>
<td>C</td>
<td>46</td>
<td>104</td>
<td>0.4</td>
</tr>
<tr>
<td>11</td>
<td>C</td>
<td>243</td>
<td>256</td>
<td>1.0</td>
</tr>
<tr>
<td>11</td>
<td>S</td>
<td>236</td>
<td>156</td>
<td>1.5</td>
</tr>
<tr>
<td>12</td>
<td>C</td>
<td>166</td>
<td>56</td>
<td>3.0</td>
</tr>
<tr>
<td>12</td>
<td>S</td>
<td>155</td>
<td>108</td>
<td>1.4</td>
</tr>
</tbody>
</table>

Mean ± s.d. 141 ± 97* 174 ± 110* 1.2 ± 0.9**

Perfusion type C, perfusion with countercurrent flow; type S, perfusion without countercurrent flow.

* Significantly different from zero ($P < 0.01$).

** Significantly different from 2 ($P < 0.01$).

† Only one rete mirabile perfused.

Sources for CO$_2$ formed in the gas gland

The important results of this study are the demonstration of the high rate of CO$_2$ production in the swimbladder tissue and the high respiratory exchange ratio, which significantly exceeded the value of unity expected for oxidative glucose breakdown. The values were calculated assuming no gas secretion into the swimbladder, which was indeed below the detection level. In our experiments the CO$_2$ may derive from three sources: washout from the tissue, oxidative metabolism and anaerobic decarboxylation reactions.

Washout of HCO$_3^-$/CO$_2$ from the tissue

As the CO$_2$ content in the perfusing solution was kept low, to ensure a high sensitivity for arteriovenous CO$_2$ differences, the swimbladder tissue had probably been cleansed of most of the CO$_2$ and HCO$_3^-$ during the perfusion period before
measurements were performed. An enhanced conversion of HCO$_3^-$ to CO$_2$ is to be expected as a result of lactic acid formation.

The exact amount of HCO$_3^-$/CO$_2$ washed out from the tissue is difficult to estimate. Let us consider first the conversion of HCO$_3^-$ to CO$_2$ in the tissue, effected by the protons liberated from lactic acid, and its subsequent release into the perfusate. The amount of lactate produced (Table 1) would, indeed, suggest a large contribution to the CO$_2$ release into the perfusate, but there are two other possibilities for the H$^+$ apart from combining with HCO$_3^-$: One is binding to nonbicarbonate buffers in the tissue, the other binding to any buffer in the perfusate and washout with it. The nominal buffer value of the perfusate was very low, and the pH change in the perfusate from $ai$ to $ve$ of 1–2 units suggests that the amount of H$^+$ washed out is probably low. We do not know the buffer value of the swimbladder tissue and, thus, cannot estimate the amount of CO$_2$ washed out from it by considering the H$^+$ liberated from lactic acid.

There is another reason to suggest that the contribution of HCO$_3^-$/CO$_2$ washout from tissue was probably small. Assuming a bicarbonate concentration of approximately 2 mmol l$^{-1}$, typical of muscle tissue, the tissue mass of the swimbladder (about 0.4 g for an 800 g eel; Ball et al. 1955; Wittenberg et al. 1964) would contain less than 1 µmol of HCO$_3^-$. Under steady-state conditions (see Fig. 2) this cannot contribute very much to the observed rate of CO$_2$ formation.

**CO$_2$ formation by aerobic glucose metabolism**

The rate of CO$_2$ production by glucose oxidation can best be estimated in our experiments from the O$_2$ consumption rate, which suggests that 12 nmol min$^{-1}$ or about 20% of the CO$_2$ derives from this pathway. It thus appears that the major fraction of the CO$_2$ formed must stem from the third source.

**CO$_2$ formation by anaerobic decarboxylation**

An example of anaerobic CO$_2$ formation is the pentose phosphate shunt. This pathway allows formation of CO$_2$ by decarboxylation of 6-phosphogluconate formed enzymatically from glucose. The pentose phosphate shunt is known to occur in ocular tissues of fish, where it contributes significantly to the glucose metabolism (Hoffert & Fromm, 1970). Boström et al. (1972) found that in the cod glucose-6-phosphate dehydrogenase and 6-phosphogluconate dehydrogenase, key enzymes of the pentose phosphate shunt, displayed higher activities in gas gland cells than in muscle cells. In preliminary measurements we found activities of these two enzymes in the eel gas gland similar in magnitude to those in liver tissue and about 10 times higher than their activities in muscle tissue. It is, thus, to be expected that part of the glucose should be diverted to the pentose phosphate shunt to form CO$_2$ by decarboxylation catalysed by 6-phosphogluconate dehydrogenase. In this case, an enzymatic reaction reoxidizing the NADPH produced in the pentose phosphate shunt is necessary. This reoxidation might occur by chain elongation of fatty acids.

Another possibility for anaerobic CO$_2$ formation has been described for goldfish.
CO2 production in swimbladder tissue

muscle (Van den Thillart & Verbeek, 1982; Mourik, 1982). This CO2 formation, which is combined with ethanol formation (Shoubridge & Hochachka, 1980), is predominantly located in the mitochondria of red muscle cells. The observed activities of the pentose phosphate shunt enzymes, and the histological observation that gas gland tissue contains only few mitochondria (Dorn, 1961), lead us to favour the pentose phosphate shunt as a possible pathway for anaerobic CO2 formation.

Glucose as a source of CO2 formation

Since the pentose phosphate shunt requires glucose, more glucose should be degraded in our experiments than would be expected from lactate formation and oxidative metabolism. The balance of glucose metabolism is, therefore, of interest in our study.

The glycogen stores in swimbladder tissue are too small to fuel glycolysis for more than a few minutes (D’Aoust, 1970), and blood (or saline) glucose is expected to be the main source for metabolism. Three pathways of glucose degradation have to be considered in our study: aerobic oxidation to CO2, anaerobic fermentation to lactic acid and anaerobic decarboxylation reactions (for example, the pentose phosphate shunt).

Aerobic metabolism does not seem to be of great importance in gas gland cells. In histological studies Dorn (1961) and Morris & Albright (1975) found only thin and irregularly distributed mitochondria. Boström et al. (1972) measured only a very low activity of cytochrome oxidase in gas gland tissue of the cod, Gadus morhua. This is in line with the low rate of aerobic glucose metabolism in our experiments, which was estimated from the O2 consumption rate to be 2 nmol min⁻¹ or only 1–2% of the total glucose metabolized.

Swimbladder tissue is known to produce lactic acid even in the presence of oxygen (Ball et al. 1955; D’Aoust, 1970), and its rate of production allows us to estimate the rate of glucose metabolized by this route. Since two lactic acid molecules are formed from each glucose molecule, a \( \frac{M_{La}}{M_{Glu}} \) ratio of 2 would indicate complete degradation of glucose to lactate. The average value in our study of 1.2 indicates that only 60% of the glucose was used for anaerobic glycolysis. Considering also the oxidatively metabolized glucose, about 38% of the glucose metabolized could have entered the pentose phosphate shunt. It is not possible to predict the ratio of CO2 to glucose in this pathway, but our data seem to indicate that most of the CO2 was formed by the pentose phosphate shunt.

Physiological significance of anaerobic CO2 formation

Anaerobic formation of CO2 appears to have several advantages for gas secretion into the swimbladder, which requires generation of high gas partial pressures, particularly of O2 and CO2, in blood (Fänge, 1983). Let us first consider a situation in which high O2 and CO2 gas partial pressures are formed in a swimbladder by aerobic CO2 and anaerobic lactic acid formation, but with no extra CO2 production (Fig. 3). \( P_{CO2} \) would mainly be increased by aerobic CO2
Fig. 3. (A) Diagram of metabolic processes in gas gland cells contributing to the formation of CO₂ and to the delivery of oxygen from the haemoglobin (Hb) in the capillaries. The anaerobic decarboxylation reaction was assumed to occur in the pentose phosphate shunt (PPS). Open arrows (⇐) indicate an influence on haemoglobin oxygen-affinity. TCA, tricarboxylic acid cycle. (B) The diffusion gradients for the gases CO₂ and O₂ shown schematically.

formation, and countercurrent enhancement in the rete (Kuhn et al. 1963) would further increase P_{CO₂}. The ensuing combined respiratory and metabolic acidosis (lactic acid formation) would release O₂ from the haemoglobin bond via the Bohr and Root effects (see Steen, 1970), and the increased P_{O₂} would be further enhanced in the rete countercurrent system.

It should, however, be noted that both CO₂ and O₂ secretion into the swimbladder would counteract the increases in their partial pressure. Moreover, any CO₂ molecule formed would consume one O₂ molecule, thus also reducing P_{O₂}. And, even if some CO₂ were formed from HCO₃⁻, this would reduce the total salt concentration which is required for raising the partial pressures of inert gases (e.g. N₂ and Ar) by the salting-out effect (Gerth & Hemmingsen, 1982; Enns et al. 1967; Pelster et al. 1988a).

In the presence of an anaerobic route for CO₂ formation (Fig. 3), the efficiency of raising all partial pressures would be significantly increased. P_{CO₂} itself ca
CO₂ production in swimbladder tissue

thereby attain higher values, and this would not reduce PCO₂ levels. The highest PCO₂ values will be found in the gas gland cells, where CO₂ is formed, and CO₂ will diffuse from the tissue to the swimbladder and to the blood (Fig. 3). The concomitantly more pronounced reduction in pH would yield higher PO₂ levels in the blood by the Root and Bohr effect. Furthermore, HCO₃⁻ could be formed from CO₂ despite the lactacidosis, and this would increase the salt concentration for the salting-out effect.

It is thus evident that anoxidative CO₂ formation, for example via the pentose phosphate shunt, may be of great significance for the formation of high gas partial pressures in the swimbladder.

The authors wish to thank Mrs G. Ryfa and Mr S. Röhr for expert technical assistance. Financial support by the Ministerium für Wissenschaft und Forschung des Landes Nordrhein-Westfalen (Grant no. IV B 4-10200687) is gratefully acknowledged.

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