

MECHANISMS OF INTESTINAL BICARBONATE SECRETION IN MARINE TELEOST FISH

Martin Grosell

Zoophysiological Laboratory, The August Krogh Institute, University of
Copenhagen, Universitetsparken 13, Copenhagen, DK-2100 Ø, Denmark.
Phone: +45 35 32 16 40/Fax: +45 35 32 15 67/email: mgrosell@aki.ku.dk.

Rod W. Wilson

Dept. of Biological Sciences, Hatherly Laboratories, University of Exeter
EX4 4PS, UK. Phone: + 44 01392-264652/Fax: +44 01392-263700/email:
r.w.wilson@exeter.ac.uk

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Bicarbonate is in many cases the dominant anion in the intestinal lumen of marine teleost fish and exceeds 100 mM in certain species (Fig. 1. and Walsh et al., 1991).

Considering the low extracellular bicarbonate concentrations in fish, such concentrations are in excess of electrochemical equilibrium across the intestinal epithelium (Grosell et al., 2001). This means that (secondary) active bicarbonate secretion must occur across the intestinal epithelium. Bicarbonate concentrations only increases slightly along the length of the intestine but in species investigated so far, all segments of the intestine can secrete bicarbonate at high rates.

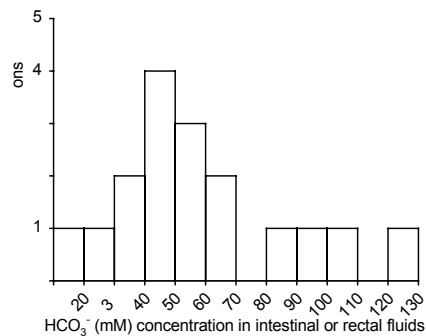


Figure 1. Frequency of 17 reported HCO₃⁻ concentrations from intestinal or rectal fluids from 14 marine teleost fish

The substantial intestinal bicarbonate secretion does not seem to play a role in digestion since most or all reported studies were performed on un-fed animals. High intestinal bicarbonate concentrations are present in euryhaline species residing in seawater but virtually absent when in freshwater,

suggesting that bicarbonate secretion is involved in osmoregulation (Wilson, 1999).

Intestinal bicarbonate secretion occurs in exchange with chloride absorption via an apical chloride/bicarbonate exchanger (Grosell & Jensen, 1999; Grosell et al., 2001) and contributes significantly to overall chloride (and thereby water) absorption by the intestine. Carbonic anhydrase is fueling at least part of the bicarbonate secretion through hydration of CO₂ (Wilson et al., 1996). Ongoing studies employing pH-stat titrations have demonstrated that CO₂ arising from epithelial metabolism is the main source of the secreted bicarbonate in the European flounder. The protons arising from the CO₂ hydration must be extruded by the epithelial cells to prevent reversal of this carbonic anhydrase mediated reaction and thereby sustain sufficiently high bicarbonate concentrations for the chloride/bicarbonate exchange. The carrier(s) involved in the proton extrusion is the subject of current studies but it is clear that the extrusion must occur across the basolateral membrane to explain the substantial net base secretion by this epithelium. A basolateral proton pump could be driving this proton extrusion (Grosell et al., 2001) but basolateral sodium/proton exchange is another possible mechanism. The latter exchange process would be possible due to the sodium gradient across the basolateral membrane of the intestinal epithelial cells. This sodium gradient is established by the basolateral sodium-potassium ATPase which thus indirectly could be fueling intestinal bicarbonate secretion. Bicarbonate secretion appears to depend on serosal sodium (Grosell et al., 2001) which support the proposed involvement of a basolateral sodium/proton exchange mechanism. The possible involvement of a basolateral sodium:bicarbonate co-transport system seems unlikely since current studies revealed substantial bicarbonate secretion even in the absence of serosal bicarbonate.

Ongoing studies on the European flounder have demonstrated that reduced luminal pH and elevated luminal calcium concentrations both stimulate intestinal bicarbonate secretion. The result of the substantial bicarbonate secretion is a highly alkaline environment (Walsh et al., 1991; Wilson, 1999; Grosell et al., 2001) which facilitate calcium-carbonate precipitation. The functional significance of this precipitation is discussed in a parallel presentation (Wilson & Grosell).

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