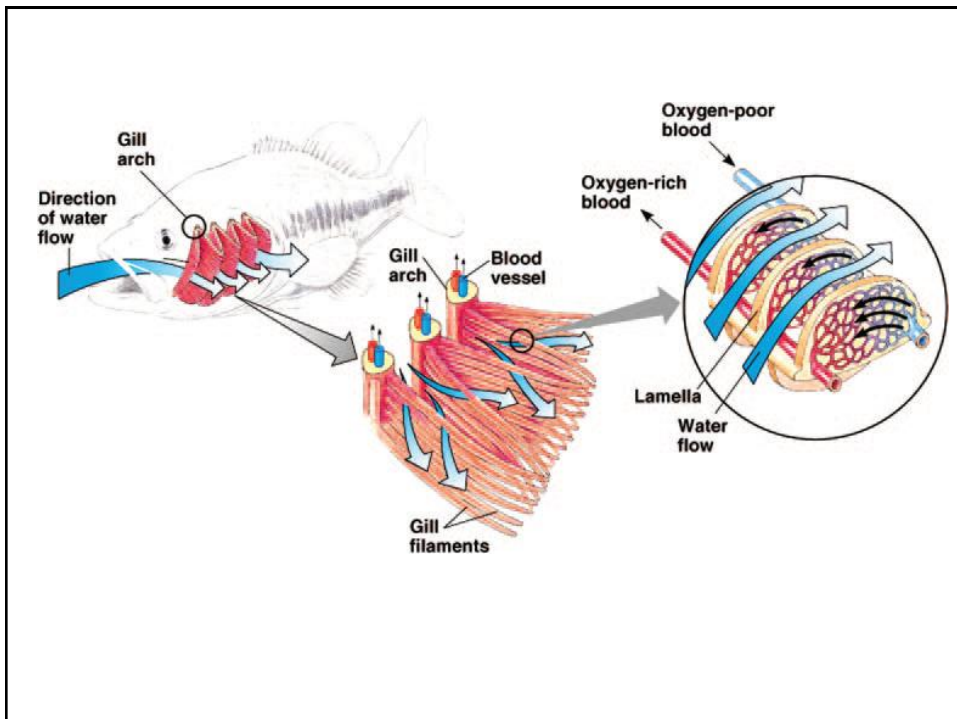
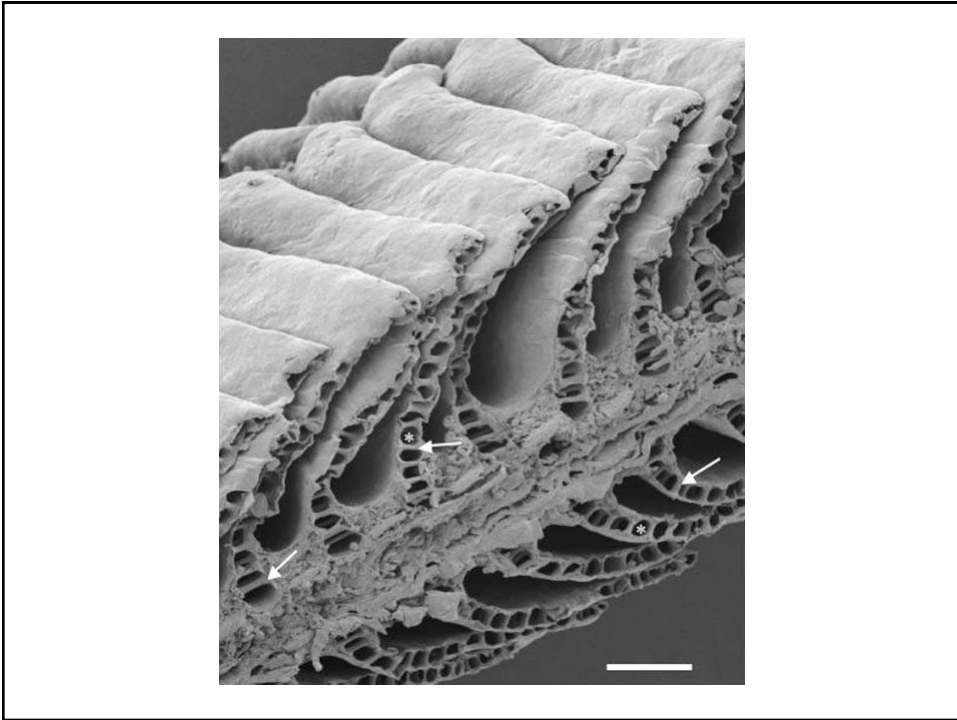


# Gills

- Diagram the blood flow
- Name some of the gill functions?



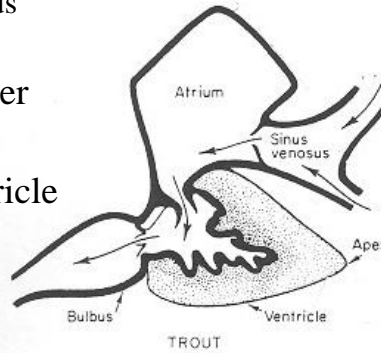


## Fish Cardiovascular system

- Branchial Heart – 4 chambers (different counting)
  - Sinus venosus, atrium, ventricle, and bulbus arteriosus/conus arteriosus
- Sinus venosus has pacemaker tissues to initiate heart beat
- Atrial contraction fills ventricle
- Ventricle largest and main pressure

# Fish Cardiovascular system

- Branchial Heart – 4 chambers
  - Sinus venosus, atrium, ventricle, and bulbus arteriosus or conus arteriosus
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- Atrial contraction fills ventricle
- Ventricle largest and main pressure



All blood goes to gills!

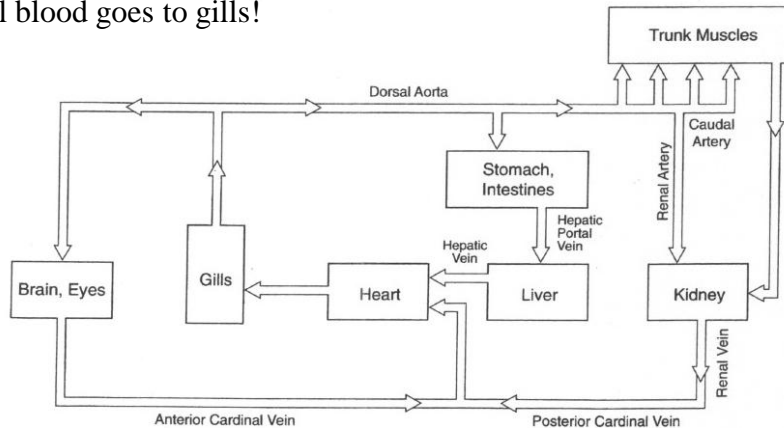


Figure 2.5. Simplified block diagram showing the major features of the fish circulatory system. Organs and tissues further from the heart must function with progressively less oxygen. Note the separate circulation of freshly oxygenated blood from the gills to the brain and return.

## Adaptive Radiation

- Differences in blood pressures in fish lineages
- Highest in active teleosts
- Cardiac output (Q) is distributed to respiratory circulation via afferent branchial arteries – does not return to heart

## Regulation of Q- Cardiac Output

- Changes in heart rate (Fh) and stroke volume (SVh) that are controlled by intrinsic neural and humoral control mechanisms
- Cardiac cycle is a coordinated event with contraction /relaxation cycles of atrium or ventricle separated into systolic (raised blood pressure from contraction) and diastolic periods (relaxed ejection phase)

## Activity and Temperature

- Fish that have high levels activity have higher resting than sluggish forms
- Temperature has profound effect on Q and increases with temperature.

**TABLE 4**  
**Myocardial Power Output in Selected Fishes at Rest and While Swimming**

	Power output (mW/g)		Temperature (°C)	Body Mass (kg)	Source	
	Rest	Exercise				
Temperate-water fishes						
hagfish	<i>Myxine glutinosa</i> <sup>+</sup>	0.08	0.27	11	0.08	a
houndshark	<i>Triakis semifasciata</i>	1.71	3.30	14–24	1.93	b
Cat shark	<i>Scyliorhinus stellaris</i>	1.43	2.46	19	2.6	c
	<i>Gadus morhua</i>	1.77	3.29	10.5	0.4–0.8	d
lingcod	<i>Ophiodon elongatus</i>	1.18	3.21	10	4.2	e
Sea raven	<i>Hemitripteris americanus</i>	1.16	3.13	10	1.2	f
	<i>Anguilla australis</i>	1.08	2.19	16–20	0.9–1.1	g
	<i>Oncorhynchus mykiss</i>	1.53	7.03	11	1.0	h
	<i>Oncorhynchus kisutch</i>	1.22	5.97	5	1.4	i
Tropical fishes						
Skipjack tuna	<i>Katsuwonus pelamis</i>	4.70	—	26	1–2	j
	<i>Thunnus albacares</i>	5.60	—	26	1–2	j
Antarctic fishes						
Black fin	<i>Chaenocephalus aceratus</i>	0.98	—	0.5–2	1.0	k
crocodile	<i>Chionodraco hamatus</i> *	—	1.6–3.4	0–2	0.29–0.47	l
Cod ice fish	<i>Pagothenia borkgrevinki</i>	1.05	2.00	0	0.06	m

*Note:* Where ventricle mass is not known values of 0.2 g/kg body and 0.08 g/kg body were assumed for elasmobranch and teleost species. Plus (+) denotes maximum value for postadrenaline infusion. Asterisk (\*) denotes maximum value in a perfused heart preparation.

# Blood pressures

- Systemic dorsal aortic pressure is 1/3 lower than that of ventral aortic circulation
- Primary = ventral and dorsal aortas
- secondary systems drain the veins and have accessory hearts arise from gills and arterial vessels =

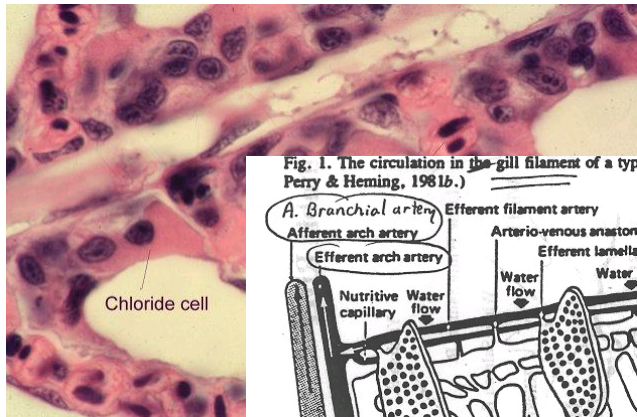
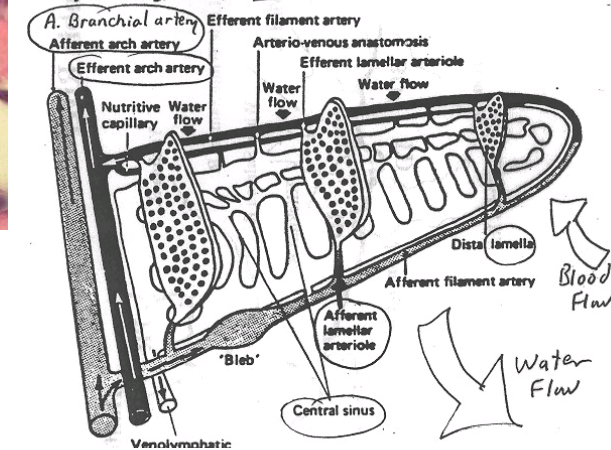


Fig. 1. The circulation in the gill filament of a typical teleost. (From Randall, Perry & Heming, 1961b.)



## Blood pressures of rainbow trout mm Hg

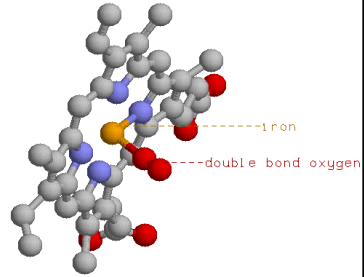
	<b>Resting</b>	<b>exercise</b>
• Ventral aorta	40/30	55/43
• Dorsal aorta	30/25	33/29
• Large veins	0-10	0-18

## Regulation of Cardiac Activity

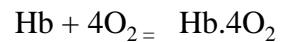
- Pacemaker cells – temperature response
- Autonomic neural control – neurotransmitters and Vagus activity
- Humoral control – circulating catecholamines e.g. Adrenaline and noradrenaline several types of receptors

# Hemoglobin, the Magic Molecule

- Tetrameric molecule in most fish
- Two alpha two beta chains



Four disks = where the oxygen molecules bind  
four sausage shapes represent polypeptide chains



- O<sub>2</sub> binds to the four heme groups
- H<sup>+</sup> and CO<sub>2</sub> bind to specific amino acid residues in globin chain



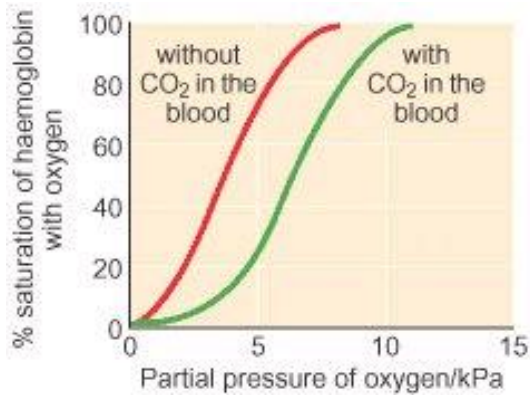
## Red Blood Cells of Fish

- Nucleated retain most organelles
- Long lived – rbt ~ 6 mo
- Mammalian red blood cells biconcave and extrude their organelles shortly after release into the circulation –short lived ~ 6 days

## Binding and pH

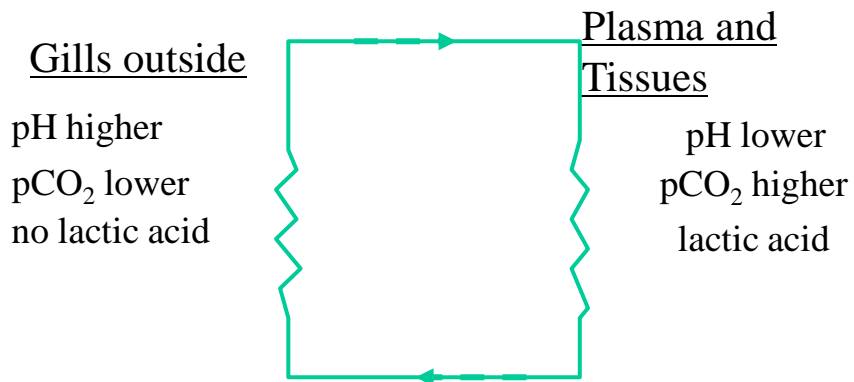
- Hb-O<sub>2</sub> binding affinity is decreased by reduction in pH (Bohr effect)
- Increase in Hb-O<sub>2</sub> saturation is associated with release of protons, **termed Bohr protons**, from hemoglobin (the Haldane effect)
- Acidification of rbc interior associated with CO<sub>2</sub> to blood in tissues will augment O<sub>2</sub> unloading in tissues via Bohr effect

CO<sub>2</sub> diffuses into the blood plasma and RBC (erythrocytes) in the presence of the catalyst carbonic anhydrase (CA) most CO<sub>2</sub> reacts with water in the erythrocytes and the dynamic equilibrium is established



## What the Bohr Effect Does

blood circulation



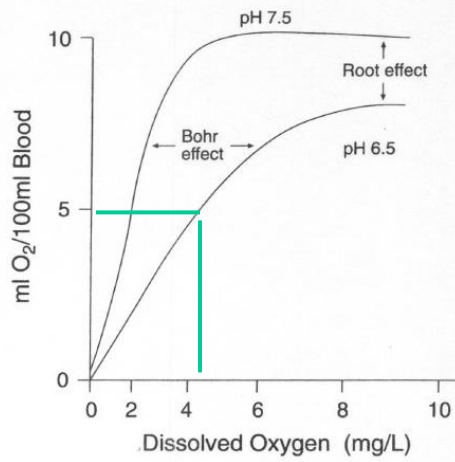
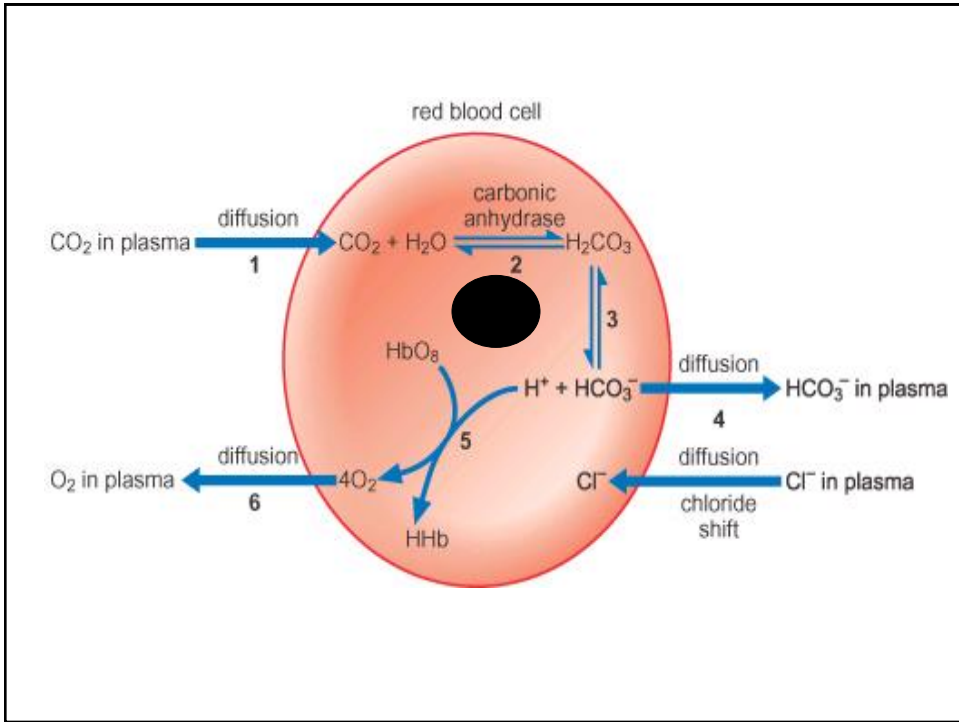


Figure 2.1. Effect of decreased blood pH due to hypercapnia or hyperlacticemia on the O<sub>2</sub> carrying capacity of salmonid hemoglobin.

## Binding of Hb-O<sub>2</sub>

- Nucleoside triphosphates for energy
  - ATP (adenoside triphosphate) & GTP (guanosine triphosphate)
- H ions
  - *Bohr effect*, reduction in pH decreases affinity
  - Increase in pH increases affinity



**Blood samples – which is from Arctic fish?**



## Summary - Quantity O<sub>2</sub> available is affected by

- Oxygen partial pressure
- Shape of oxygen equilibrium curve
- Blood oxygen carrying capacity
- Cardiac output

## Modification of Blood Oxygen Carrying Capacity

- Spleen can release rbc
  - Hypoxia; hypercapnia, exercise
- Chronic stress can also stimulate erythropoiesis (proliferation of rbc)

## Catecholamines affect RBC releases

- Chromaffin tissue areas that release
  - Post cardinal vein (head kidney)
  - Other regions of kidney

## Acid Base Regulation – Chemical/physical

- Normal blood pH 7.7 – 8
- Normal intracellular 7.2 – 7.5
- Maintenance via two tiered process of internal buffering, and transfer of acid base relevant molecules between animal and water

## pH of fish blood is temperature dependent

- $\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{HCO}_3^- + \text{H}^+$
- 8.0 at 10°C
- 7.7 at 30°C

## pH and temperature

- Neutral pH is only neutral at 24°C
- Below that the pH is higher
- Above that the pH is lower --- why?

## Conditions that disturb Acid Base Balance

1. Metabolic acidosis – lactic acid
2. Respiratory acidosis – high CO<sub>2</sub>
3. Environmental hypercapnia – high CO<sub>2</sub>
4. Environmental acidosis – mine drainages, acid rain

## What is pH

- Negative log of H ion concentration!



## Defense Systems/Compensations

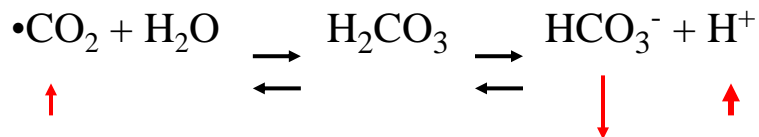
- Buffering in blood, intercellular
  - Instantaneous
- Respiratory Tissues, intercellular and extracellular
  - Within minutes
- Kidney Excretion (marginal factor in fish)
  - Slow, hours.
  - Conserving bicarbonates

## Buffering in Intracellular and Extracellular Systems

- Intracellular: Proteins and Phosphates = non bicarbonate buffers – Nbbs
- = 2/3 of body fluids
  
- Extracellular: Bicarbonates and Ammonias
  - Major anion Na

## Metabolic Acidosis

- Lactic acid produced in tissues as consequence of extreme exercise or hypoxia. H<sup>+</sup> ions and lactate ion diffuse into blood
- Effect on buffering system is shift to left



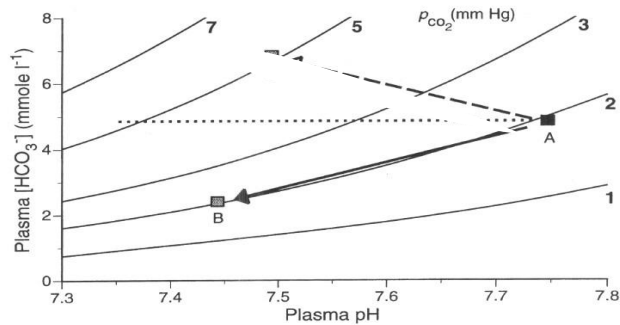
## Compensation – metabolic acidosis

- H<sup>+</sup> excretion and / or HCO<sub>3</sub><sup>-</sup> uptake
- The bicarbonate buffer shifts this to release excess CO<sub>2</sub>

# Metabolic Acidosis

180

*The Physiology of Fishes*



**FIGURE 1.** A pH-HCO<sub>3</sub><sup>-</sup> diagram for a hypothetical acid-base experiment. Point "A" represents a control plasma pH of 7.75, a [HCO<sub>3</sub><sup>-</sup>] of ~5.0 mmol l<sup>-1</sup>, and a p<sub>CO<sub>2</sub></sub> of 2.0 mmHg. The change to point "B" (solid arrow) demonstrates the "typical" pattern of a metabolic acidosis as plasma [HCO<sub>3</sub><sup>-</sup>] decreased to 2.5 mmol l<sup>-1</sup> and pH fell to 7.45. [an

## Compensation

- H<sup>+</sup> must be excreted to environment by direct or indirect exchange for environmental Na<sup>+</sup> and/ or HCO<sub>3</sub><sup>-</sup> – must be retained (baseline rate of exchange for environmental Cl<sup>-</sup> must be decreased)

# Respiratory Acidosis

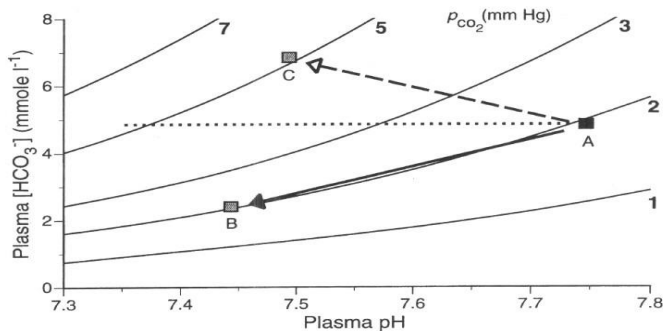
- Respiratory gas CO<sub>2</sub> is produced in large enough quantities so that it cannot be immediately eliminated by diffusion across gills



# Respiratory Acidosis

180

*The Physiology of Fishes*

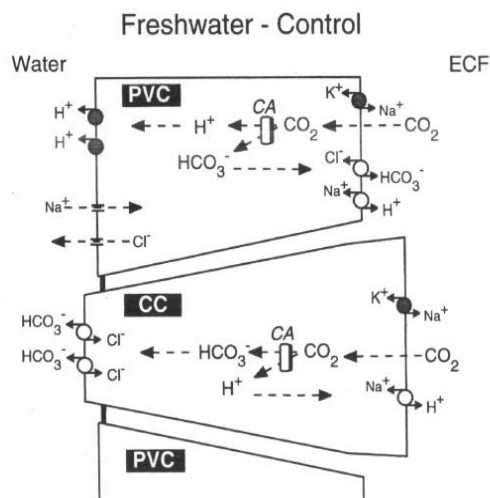


**FIGURE 1.** A pH-HCO<sub>3</sub><sup>-</sup> diagram for a hypothetical acid-base experiment. Point "A" represents a control plasma pH of 7.75, a [HCO<sub>3</sub><sup>-</sup>] of ~5.0 mmol l<sup>-1</sup>, and a P<sub>CO<sub>2</sub></sub> of 2.0 mmHg. The change to point "B" (solid arrow) demonstrates the "typical" pattern of a metabolic acidosis as plasma [HCO<sub>3</sub><sup>-</sup>] decreased to 2.5 mmol l<sup>-1</sup> and pH fell to 7.45. The respiratory acidosis shown in the change from A to C (dashed arrow) is due to an elevation of P<sub>CO<sub>2</sub></sub> to ~5 mmHg as pH decreased to 7.50. The increase in [HCO<sub>3</sub><sup>-</sup>] (also represented by the slope of the dashed arrow) is due to the nonbicarbonate buffering of a portion of the H<sup>+</sup> formed as P<sub>CO<sub>2</sub></sub> increased. The horizontal dashed line represents the theoretical change in pH and [HCO<sub>3</sub><sup>-</sup>] projected for this same P<sub>CO<sub>2</sub></sub> increase had no Nbbs been present (see text).

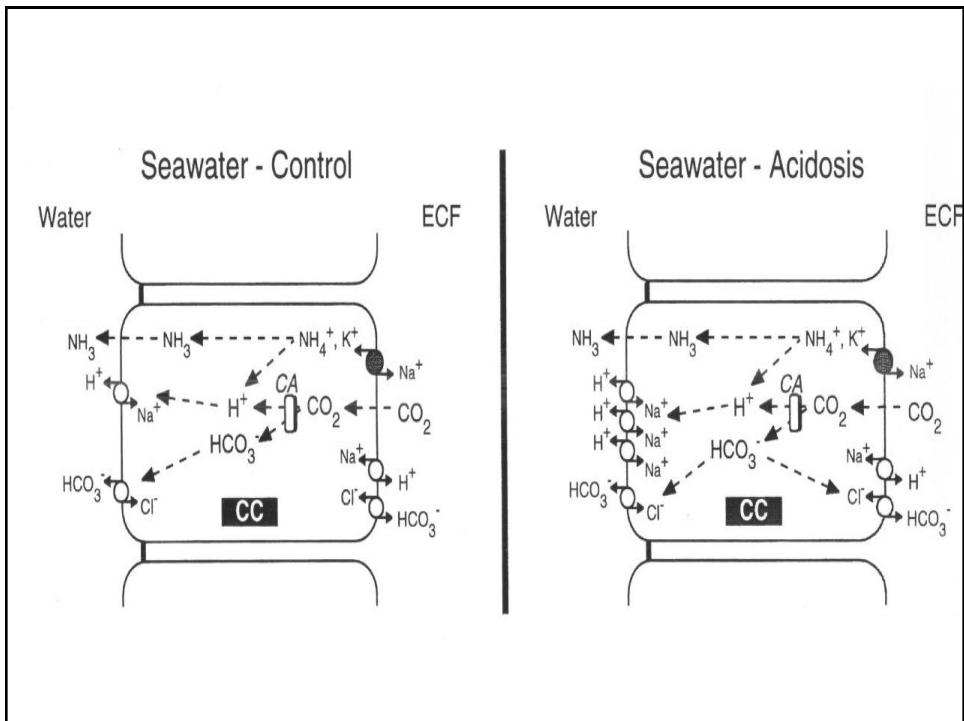
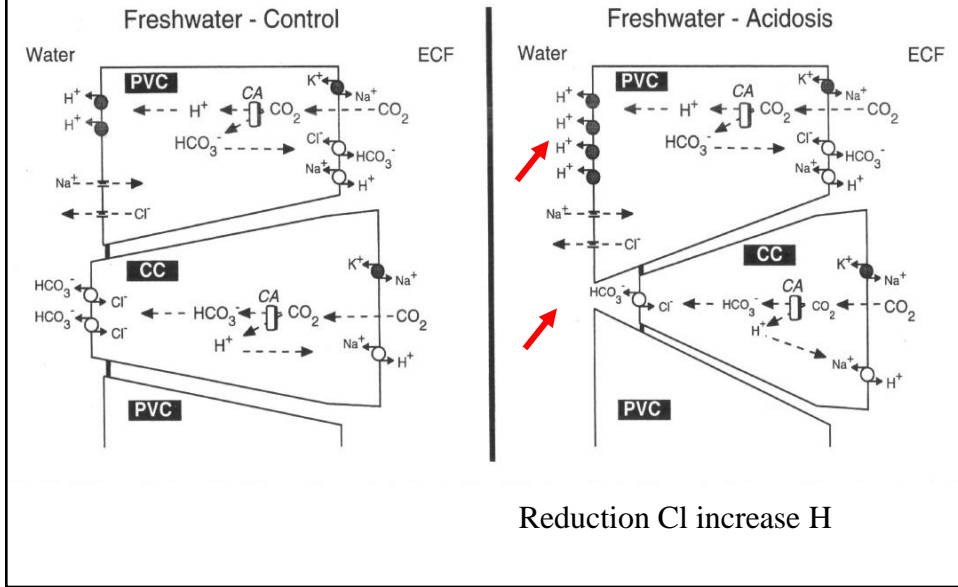
# Compensation for Respiratory Acidosis

- CO<sub>2</sub> diffusion from blood to water will occur with bicarbonate buffering.

Pavement cell, chloride cells

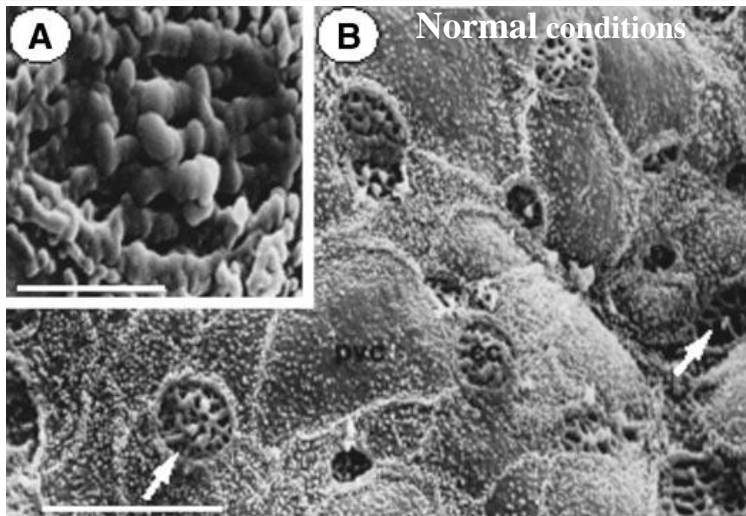
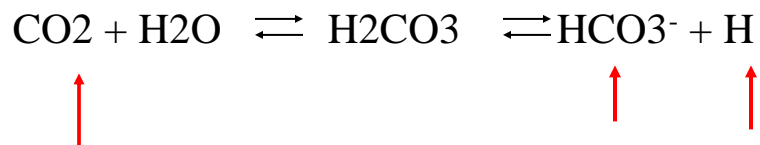


## Pavement cell, chloride cells



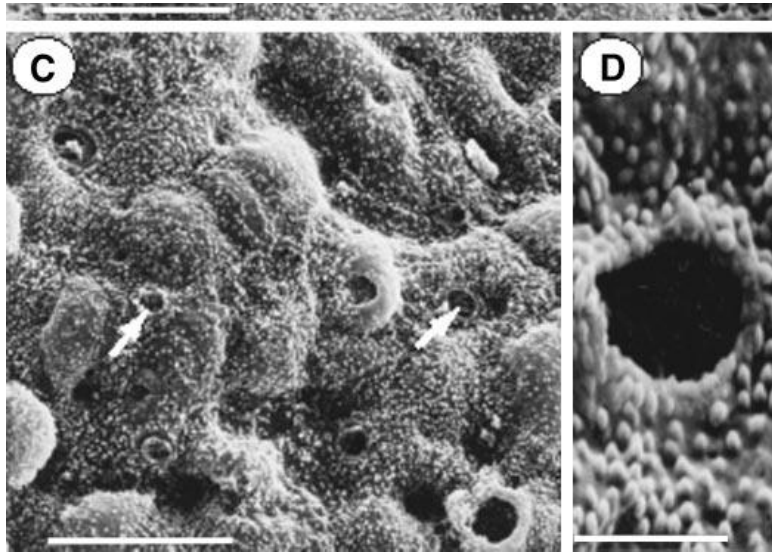
## Environmental Hypercapnia

- Environmental  $PCO_2$  is elevated.
- Often by ice cover, or metabolic of plants, animals or microorganisms or  $CO_2$  anesthesia

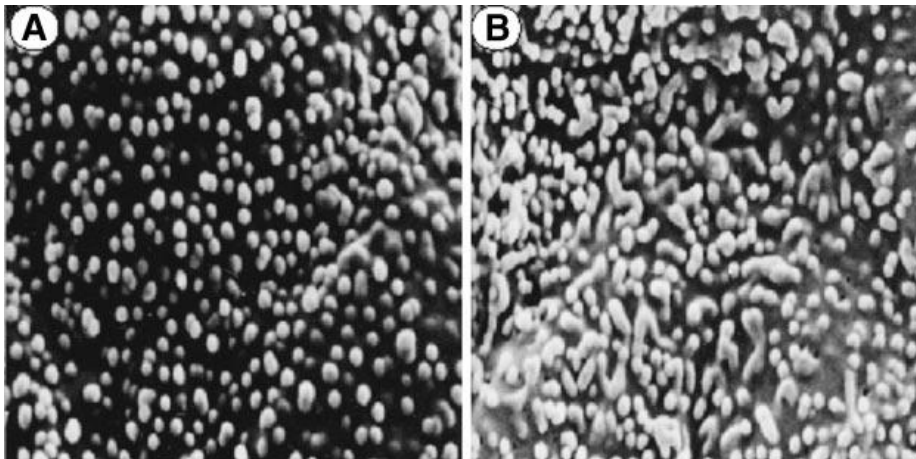


Mitochondrial rich cells (MRC- chloride cells)  
large area





**6 hr of hypercapnia. MRC are reduced, and look different with surface area reduced.**



**Before**

**After**

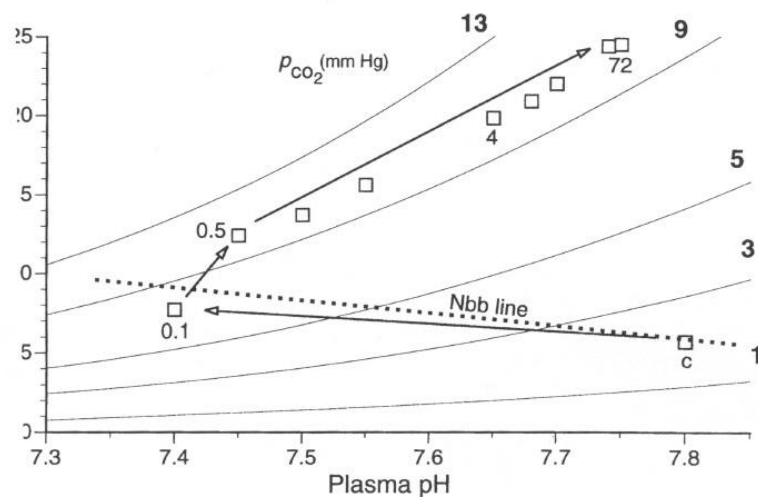
**Epithelial pavement cells (PVC) before and after 6 h hypercapnia. Note density in microvilli is increased**



# Compensation

- Because environment is elevated in CO<sub>2</sub>, equilibrium is shifted, bicarbonate reserves are limited (except in air breathers)
- The compensations can take a long time, depending on the environment

**Blood to water gradient is affected and excess CO<sub>2</sub> cannot be lost by diffusion. To restore H<sup>+</sup> back down, HCO<sub>3</sub> must be retained, and H<sup>+</sup> excretion increases**



## Environmental Acidosis

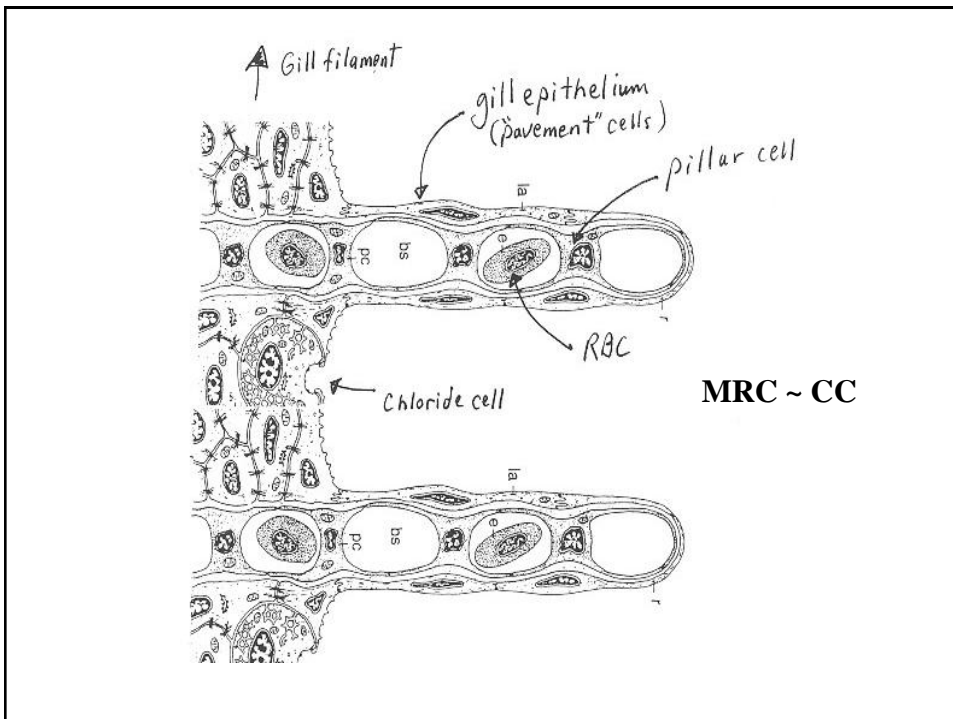
- Acidification of water due to mine drainage
- Acid rain, etc.
  
- Response similar to metabolic acidosis

## Longer term compensation for acid base disturbance

- Buffering is short term
- Active exchanges occur at respiratory epithelium
- Export of H ions through  $\text{Na}^+/\text{H}^+$  exchange
- Chloride cells  $\text{Cl}^-/\text{HCO}_3^-$  some through morphology changes

Relative contribution of bicarbonate and non bicarbonate for fixed or respiratory acid base changes for blood, and external and internal cell fractions

	<u>Fixed Acid or Base</u>		<u>Respiratory Acid Change</u>	
	<u>HCO<sub>3</sub></u>	<u>Nbbs</u>	<u>HCO<sub>3</sub></u>	<u>Nbbs</u>
Blood & Extra CF	some	some	none	all
Intra CF	little	most	none	all

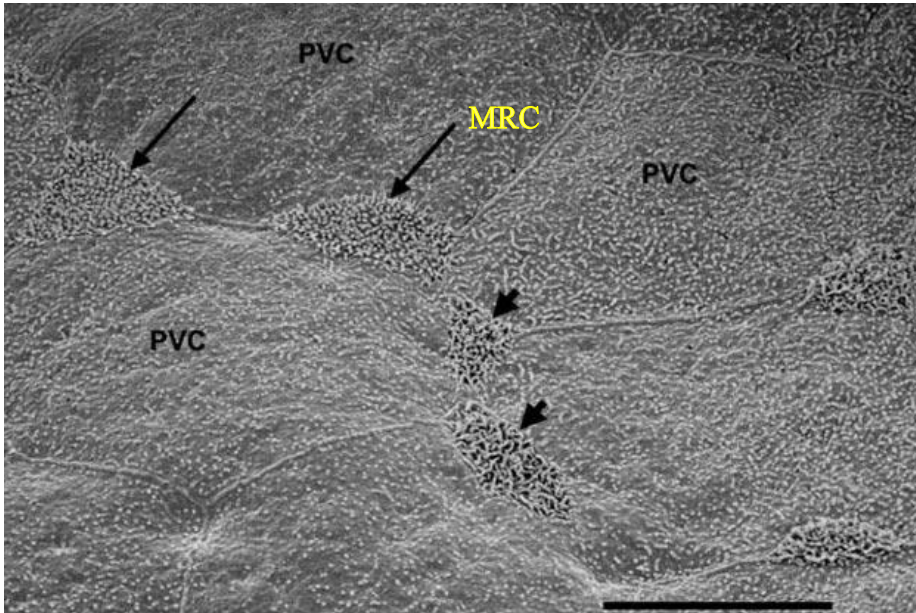


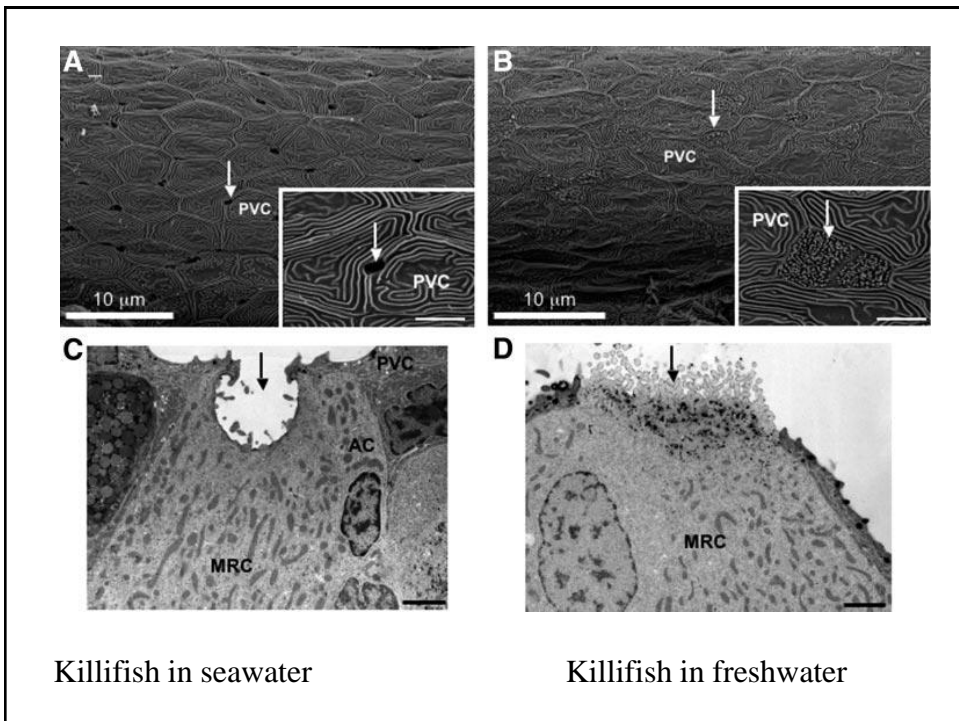
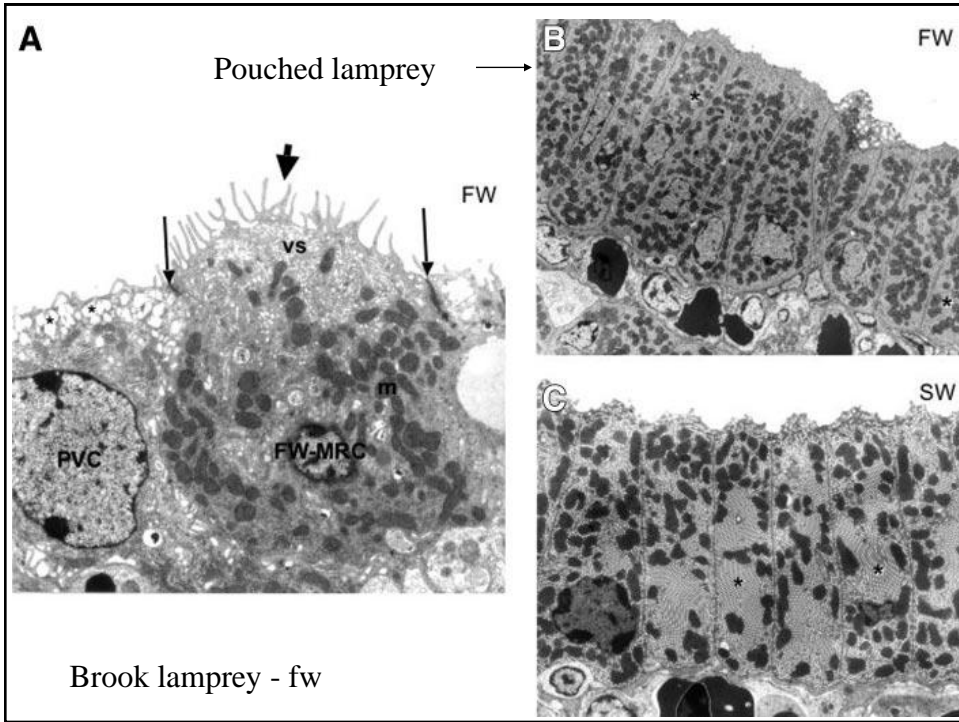
**TABLE 2**

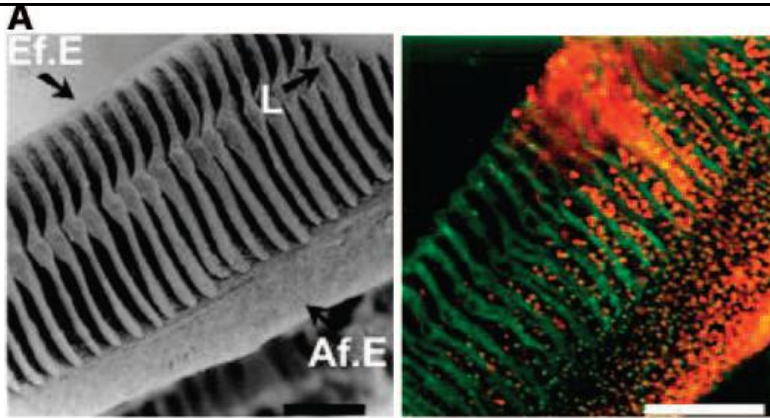
**Overview of General Acid-Base Responses in Fish to External and Internal Experimental Challenges**

Perturbation	Acid-Base Relevant Internal Change	Extracellular Acid-Base Effect	Compensatory Response in ECF	Degree of Compensation	Fish ↔ Water Compensatory Transfers
Water hypercapnia	Hypercapnia	pH decrease	Increase plasma [HCO <sub>3</sub> ]	Good	Net H <sup>+</sup> excretion
Water hyperoxia	Hypercapnia	pH decrease	Increase plasma [HCO <sub>3</sub> ]	Excellent	Net H <sup>+</sup> excretion
Water hypoxia	Hypercapnia, metabolic acidosis	pH decrease, plasma lactate increase	Reduce P <sub>CO<sub>2</sub></sub> , increase plasma [HCO <sub>3</sub> ], metabolize lactate	Excellent	Initial net H <sup>+</sup> excretion, later HCO <sub>3</sub> excretion
Water acidification	Metabolic acidosis, gill permeability increase	pH decrease, ion loss	Decreased gill permeability (some species)	Poor to excellent	Little
Water alkalinization	Hypocapnia, metabolic alkalosis	pH increase, plasma P <sub>NO<sub>3</sub></sub> /NH <sub>4</sub> <sup>+</sup> increase	Decrease plasma [HCO <sub>3</sub> ], increase lactic acid	Fair to excellent	Increased ammonia excretion, urea excretion, H <sup>+</sup> uptake
Switch from water to air breathing	Hypercapnia	pH decrease	Increase plasma [HCO <sub>3</sub> ]	Poor	—
Water temperature increase	Temperature increase	pH decrease, P <sub>CO<sub>2</sub></sub> increase, plasma [HCO <sub>3</sub> ] decrease	—	Little	Net HCO <sub>3</sub> excretion
Exercise	Hypercapnia, metabolic acidosis	pH decrease, plasma lactate increase	Reduce P <sub>CO<sub>2</sub></sub> , increase plasma HCO <sub>3</sub> , metabolize lactate	Excellent	Initial net H <sup>+</sup> excretion, later HCO <sub>3</sub> excretion
Acid infusion	Metabolic acidosis	pH decrease	Recovery of plasma [HCO <sub>3</sub> ]	Excellent	Net H <sup>+</sup> excretion
Base infusion	Metabolic alkalosis	pH increase	Reduction of plasma [HCO <sub>3</sub> ]	Excellent	Net HCO <sub>3</sub> excretion

**freshwater elasmobranch**







**B**

Gill filaments, A: scanning electron micrograph (*left*) and a corresponding confocal laser scanning micrograph (*right*) of a filament from a tilapia. MRCs are stained red; note their increased abundance between the lamellae, which are stained green, and on the Afferent A, compared with the Efferent A.

## Autonomic nervous system mediators

parasympathetic

sympathetic

Mediator	Acetylcholine	Epinephrine (Adrenaline and norpinephrine)
Sources	Vagus nerve	Spinal nerves Chromaffin tissue
Types of receptors	one	A receptor B receptor (dialator)

## Target tissues

- Heart: Decreased or increased heart rate and stroke
- Gills: Increased or decreased resistance
- Vasculature: Increased resistance