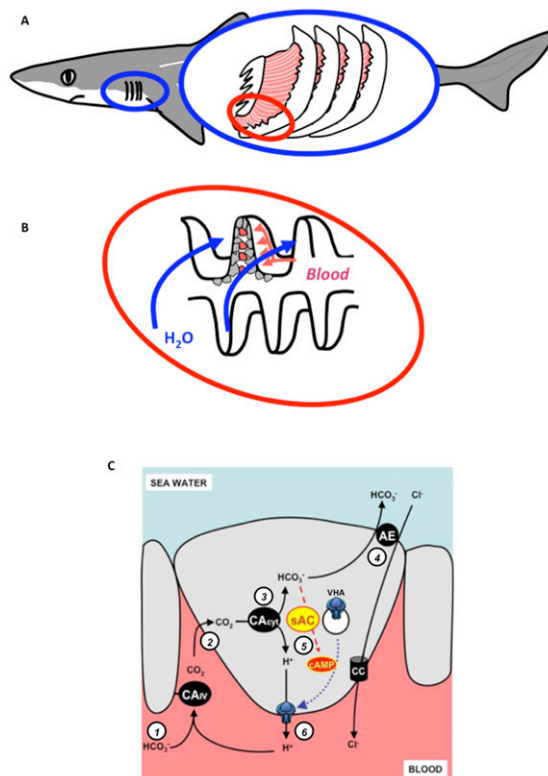


# Supporting Information

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**Fig. S1.** Dogfish gill and VHA translocation in response to blood alkalosis. (A) Dogfish have four branchial arches (plus one hemibranch, not drawn) at each side. (B) Each gill has several filaments with finger-like protrusions (lamellae). Blood and seawater flow in a countercurrent fashion. (C) Subset of cells in the gill epithelium is VHA-rich,  $H^+$ -absorbing, and  $HCO_3^-$ -secreting. (1) During increased blood  $HCO_3^-$  and pH,  $HCO_3^-$  in blood is dehydrated into  $CO_2$  by extracellular CA ( $CA_{IV}$ ) (1). (2)  $CO_2$  enters the VHA-rich cells. (3) Once inside,  $CO_2$  is hydrated back into  $H^+$  and  $HCO_3^-$  by intracellular CA ( $CA_{cyt}$ ) (2, 3). (4)  $HCO_3^-$  is secreted to seawater in exchange for chloride, probably via a Pendrin-like anion exchanger (4). (5) Elevated intracellular  $HCO_3^-$  activates sAC to generate cAMP, which triggers the microtubule-dependent translocation of VHA (blue icon) containing cytoplasmic vesicles to the basolateral membrane. (6) Basolateral VHA reabsorbs  $H^+$  into the blood to counteract the original alkalosis (5–7). The positive current is probably neutralized by a transepithelial chloride conductance (CC).

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**Table S1. Summary of the statistics used in Table 1**

Variable	Treatment effect	Time effect	Interaction effect	Additional test
pH	S	S	NS	Tukey–Kramer planned test
$[HCO_3^-]$	S	S	S	Bonferroni's posttest
$PCO_2$	NS	S	NS	None

All variables were initially analyzed using repeated-measures two-way ANOVA. NS, no statistical significant differences found ( $P \geq 0.05$ ); S, statistical significant differences found ( $P < 0.05$ ).