

**HYPOXIA-INDUCED CATECHOLAMINE
SECRETION IN JEJU, TRAIRA AND PACU**

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EXTENDED ABSTRACT ONLY – DO NOT CITE

In hypoxia-intolerant temperate fish such as the rainbow trout, exposure to severe environmental hypoxia results in a large elevation of circulating catecholamines (Reid *et al.*, 1998). In general, these hormones serve to increase the transfer of oxygen across the gills and the transport of oxygen, in the blood, to actively metabolising tissues. During environmental hypoxia, catecholamines are mobilised into the blood when the arterial oxygen content decreases by approximately 50% (Perry and Reid, 1994). Furthermore, the concentration of plasma catecholamines can reach over 500 nmol l⁻¹ in this species.

This study examined whether hypoxia-tolerant neotropical fish also release catecholamines during hypoxia and whether the levels are comparable to those observed in the rainbow trout. Three species were studied: 1. Jeju,

Hoplerythrinus unitaeniatus (a facultative air breather). 2. Traira, *Hoplias malabaricus* (an obligate air breather) and 3. Pacu, *Piaractus mesopotamicus* (a species that performs aquatic surface respiration).

Individual fish were acutely exposed (15 minutes) to 3 levels of environmental hypoxia ranging between 140 and 10 mmHg water PO₂. Blood samples were taken at the end of the 15 minute period and arterial PO₂, O₂ content and haemoglobin levels were measured. The plasma was separated from the red cells and stored at -80°C prior to analysis for catecholamines. *In vivo* oxygen dissociation curves were constructed to determine p50 values and plasma catecholamine levels were expressed as a function of both water and arterial PO₂ to determine thresholds for release.

During hypoxia, Jeju breathed water exclusively until the water PO₂ reached 30-40 mmHg at which point they began to breathe air. At this point, the frequency of air breathing increased progressively as the water PO₂ was lowered. Air breathing defended arterial PO₂ at the severe levels of hypoxia such that PaO₂ was approximately 10 mmHg higher in Jeju with access to air compared to Jeju that were denied access to air. *In vivo* p50 values for Jeju were 14.3 mmHg in fish with access to air and 7.7 mmHg in fish denied access to air. Jeju denied access to air mobilised catecholamines into the circulation when the arterial PO₂ fell to approximately 15 mmHg. Maximum levels (adrenaline plus noradrenaline) were approximately 175 nmol l⁻¹. Jeju with access to air did not mobilise catecholamines into the circulation regardless of the severity of hypoxia. At all levels of water PO₂, plasma cortisol levels were higher in Jeju with access to air compared to those without access to air.

In Traira and Pacu, arterial PO₂ decreased progressively and linearly as water PO₂ was lowered. *In vivo* p50 values were 8.6 mmHg and 11.3 mmHg for Traira and Pacu, respectively. In Traira, plasma catecholamines were elevated at arterial PO₂ values below 10 mmHg and reached levels of approximately 75 nmol l⁻¹ (adrenaline plus noradrenaline). Plasma catecholamines in Pacu were only elevated in several animals at very severe levels of hypoxia.

The results indicate that Jeju denied access to air and Traira release substantial quantities of catecholamines into their blood during exposure to severe environmental hypoxia. However, these levels are significantly lower than the levels observed in the hypoxia-intolerant rainbow trout. The very low levels of plasma catecholamines observed in Pacu suggest that these animals are extremely hypoxia-tolerant. However, it was also possible that the capacity of

the Pacu chromaffin cells to release catecholamines was reduced compared to Traira and Jeju. To address this possibility, fish were given intra-arterial injections of nicotine which would be expected to cause a large release of catecholamines via direct stimulation of nicotinic cholinergic receptors on the chromaffin cell membrane (Reid, 1999). Plasma catecholamines were elevated in both Traira and Jeju in response to intra-arterial nicotine while in Pacu, nicotine failed to cause catecholamine release into the blood. This suggests that the lack of catecholamine release during hypoxia in this species may be due to different mechanisms of catecholamine secretion, at the level of the chromaffin cell.

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