

**THE FRESHWATER STINGRAY *HIMANTURA SIGNIFIER*  
IS ABLE TO INCREASE UREA PRODUCTION  
IN RESPONSE TO BRACKISH WATER (UP TO 20 PPT),  
BUT APPARENTLY HAS A LIMITED CAPACITY  
TO RETAIN UREA**

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**EXTENDED ABSTRACT ONLY-DO NOT CITE**

The river Batang Hari originates from the Barisan Range, flows eastwards through the whole of Jambi, Indonesia, and drains into the South China Sea. *Himantura signifier* (Dasyatidae) is a stingray found in the Batang Hari basin in Jambi, Sumatra. It is believed to occur only in freshwater. In the laboratory, *H. signifier* could survive in freshwater (0.7 ppt) indefinitely or in brackish water (20 ppt) for at least two weeks. In freshwater, the blood plasma osmolality (416 mosmolal) was maintained hyperosmotic to that of the external medium (38 mosmolal). There was approximately 44 mM of urea in the plasma, with the rest of the osmolality made up mainly by Na<sup>+</sup> (167 mM) and Cl<sup>-</sup> (164 mM). In freshwater, it was not completely ureotelic, excreting at most 45% of its nitrogenous waste as urea. It had a functional ornithine-urea cycle in the liver. The hepatic carbamoylphosphate synthetase III and glutamine synthetase activities were similar to those of other marine elasmobranchs.

When it was exposed to a progressive increase in salinity (0.7 ppt→ 5 ppt→ 10 ppt→ 15 ppt→ 20 ppt→ 20ppt→ 20ppt→ 20ppt) through a 8-d period, there was a continuous decrease in the rate of ammonia excretion. After exposure to 20 ppt for 4 d, the ammonia excretion rate was only 1/5 that of the freshwater control. In 20 ppt water, there was no change in the ammonia content in the muscle and plasma, but a decrease was observed in the liver. Presumably, ammonia was used as a substrate for urea synthesis and storage for osmoregulation at higher salinities. Indeed, in 20 ppt water, urea levels in the muscle, brain and plasma increased significantly. In addition, certain free amino acids were used as intracellular osmolytes in the muscle ( $\beta$ -alanine, glycine and sarcosine) and the brain ( $\beta$ -alanine, glycine, glutamate and glutamine).

In the blood plasma, osmolality increased to 571 mosmolal, in which, urea,  $\text{Na}^+$  and  $\text{Cl}^-$  contributed 83, 231 and 220 mM, respectively. This was almost isoosmotic to the external medium (540 mosmolal). The total amount of urea accumulated in the tissues of the specimen exposed to 20 ppt water was equivalent to the deficit in ammonia excretion through the 8-d period, indirectly indicating an increase in the rate of urea synthesis at higher salinities would have occurred. However, no induction in the activity of carbamoylphosphate synthetase was observed. It is possible that the carbamoylphosphate synthetase activity was enhanced in vivo by an increase in the concentration of N-acetylglutamate which was not determined in this study. There was also a significance decrease in the rate of urea excretion during passage through 5, 10 and 15 ppt water. However, the rate of urea excretion increased back to the control value ( $3.5 \mu\text{mol day}^{-1} \text{g}^{-1}$ ) when the stingray reached 20 ppt water on the 5<sup>th</sup> day, presumably resulted from the steeper urea gradient built up between the plasma (83 mM) and the external medium (0 mM). In comparison, the local marine stingray, *Taeniura lymma*, maintained a urea excretion rate of  $4.7 \mu\text{mol day}^{-1} \text{g}^{-1}$  in full strength sea water (30 ppt), with a plasma urea concentration of 380 mM. Therefore, *H. signifer* appeared to have reduced its capacity to retain urea in order to survive in the freshwater environment. Consequently, it could not survive well in full strength sea water, although it was more euryhaline than the South American freshwater stingray, *Potamotrygon motoro*. Different from *P. motoro*, *H. signifer* retained the capacity to produce urea, as demonstrated by the capability of *H. signifer*, but not *P. motoro*, to detoxify ammonia to urea during ammonia loading.

In freshwater containing 10 mM  $\text{NH}_4\text{Cl}$  at pH 7, ammonia accumulated in the muscle, brain and plasma of *H. signifer*. The primary strategy adopted was to allow ammonia to build up internally, especially in the plasma, to slow down the

influx of exogenous ammonia. This was reflected by the unaltered urea excretion rate ( $3 \mu\text{mol day}^{-1} \text{g}^{-1}$ ) in specimens exposed to ammonia for the first day, during which ammonia excretion ( $7.3 \mu\text{mol day}^{-1} \text{g}^{-1}$ ) was presumably impeded totally. However, the urea excretion rate increased continuously to  $7.4 \mu\text{mol day}^{-1} \text{g}^{-1}$  by the 4<sup>th</sup> day of ammonia exposure, with no change to the muscle urea content, indicating that it was able to release the excess urea without creating a problem for osmoregulation.

