prepared by differential centrifugation of sucrose hemogenates. The incubation medium was similar to that used for rat liver mitochondria (Hird and Marginson, Arch. Biochem. Biophys. 115:247, 1966). Flasks were shaken in air at 25° C and ammonia production was measured by a microdiffusion-colorimetric technique. The rate of glutamate (10mM) deamination by eel liver mitochondria was approximately 1 μmole/g liver per 30 min at 25° C as compared to a rate of approximately 2 μmoles/g liver per 30 min for rat liver mitochondria at 38° C. In contrast to previous findings with rat liver mitochondria (Hird and Marginson), an ADP generating system was not essential for glutamate deamination by eel liver mitochondria. Deamination of alanine and aspartate by eel liver mitochondria was increased several fold by the addition of α-ketoglutarate, indicating that the pathway of ammonia production from these amino acids is transamination with α-ketoglutarate to produce glutamate and subsequent deamination of the latter amino acid. No transdeamination of glycine, phenylalanine, lysine, or leucine was observed.

This research was supported by NSF grant GB5613.

1967 #14

NITROGEN METABOLISM IN FISH: EFFECT OF SALINITY ON UREA BIOSYNTHESIS IN THE SKATE (Raja erinacea)

Leon Goldstein and Deborah Funkhouser, Harvard Medical School, Boston, Mass.

The early studies of Smith (Am. J. Physiol. 98:279, 1931) showed that blood urea concentration was significantly lower in fresh water elasmobranchs than in marine forms, a condition which aids in osmotic regulation. An increase in urine flow is known to contribute to the reduction in blood urea, but the role of urea biosynthesis in this phenomenon is unknown. We, therefore, investigated the effects of salinity on urea biosynthesis in the skate, Raja erinacea. Four skates weighing 380-900 g were maintained in a 12 foot swimming pool in running water at 12°-15° C After one week in full strength sea water the rates of urea and ammonia excretion (production) were 213 ± 44 (S.E.) and 123 ± 47 μmoles/kg x hr. Diluted sea water was then added to reduce the salinity to 78% over a period of three days. The skates were maintained at this salinity for an additional three days. The rates of urea and ammonia excretion and blood urea concentration were determined on the last day. Plasma urea concentration was 332 ± 12 μmoles/ml as compared to a value of 395 ± 11 μmoles/ml in a separate group of skates maintained in sea water ($p < .02$). The rate of urea excretion in skates in dilute sea water was 457 ± 55 μmoles/kg x hr which was significantly higher than the rate observed in sea water ($p < .05$) by "paired-data" analysis. The rate of ammonia excretion (181 ± 37 μmoles/kg x hr) was similar to that observed in sea water ($p < .1$).

If it is assumed that urea excretion equates to urea production under these conditions, the rise in urea production in skates maintained in diluted sea water is surprising since this would tend to oppose the mechanisms operating to lower blood urea concentration. It is possible that the predominant stimulus for urea biosynthesis is not osmotic pressure but rather blood urea concentration.

This research was supported by NSF grant GB5613.