## BACKGROUND:

Necrotizing enterocolitis (NEC) is a devastating condition affecting premature infants and leads to high mortality and chronic morbidity. Severe form of NEC is associated with acute renal failure, fluid imbalance, hyponatremia and acidosis. We investigated the effect of NEC on tight junction (TJ) proteins in kidneys using a NEC mouse model to investigate the basis for the observed renal dysfunction.

## METHODS:

NEC was induced in C57BL/6 mice by formula feeding and subjecting them to periods of hypoxia and cold stress. NEC was confirmed by gross and histological examination. We studied various markers of inflammation in kidneys and investigated changes in expression of several TJ proteins and AQP2 using immunofluorecent staining and Western blotting.

## RESULTS:

We found markedly increased expression of NF?B, TGF? and ERK1/2 along with claudin-1, -2, -3, -4, -8 and AQP-2 in NEC kidneys. The membrane localization of claudin-2 was altered in the NEC kidneys and its immunostaining signal at TJ was disrupted.

## CONCLUSION:

NEC led to a severe inflammatory response not only in the gut but also the kidneys. NEC increased expression of several TJ proteins and caused disruption of claudin-2 in renal tubules. These observed changes can help explain some of the clinical findings observed in NEC.


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