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Renal ammonia handling in cirrhosis

Background The kidney plays a dual role in the ammonia metabolism by producing ammonia and controlling the amount of ammonia reabsorbed into the renal vein or excreted into the urine. In advanced stages of liver cirrhosis, renal reabsorption of ammonia seems to diminish in favour of urinary excretion ([1]). The underlying mechanisms are not fully understood, but it is likely that the decrease is triggered by an elevated arterial concentration of ammonia and by functional alteration of the ammonia transporter system along the renal tubule. We developed a mathematical model of renal ammonia handling to explore the parameters associated with an increased urinary excretion.
Methods The model is an adaptation of a model by Hervy and Thomas ([2]) and was initially designed to study the formation of the osmotic gradient in the medullary interstitium. It simulates the reabsorption and secretion of solutes (NaCl, KCl, urea, ammonia) and water along the renal tubules. Each idealized tubule is composed of a loop of Henle and a collecting duct, and is supplied by a vasa recta. The tubes are bathed and exchange solutes with in an interstitium, which is lumped with the ascending portion of the vasa recta. The equations describe the transmural fluxes between the tubes and interstitium due to osmosis, convection, diffusion and active transport. Baseline parameters values were taken from the rat literature.
Results We compare the outputs of the model obtained with parameters mimicking the healthy and diseased states.

References

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