P3–56.
Identification and functional analysis of choline transporter in the human immortalized hepatic cell line Fa2N-4

Choline is an essential nutrient and necessary for the synthesis of the major membrane phospholipid phosphatidylcholine and the neurotransmitter acetylcholine. Specifically, past studies revealed that the liver plays a major role regarding series of choline metabolism. However, the uptake system for choline and the functional expression of choline transporters in hepatic cells are poorly understood. In this study, we examined the functional characterization of choline uptake and sought to identify the transporters in human immortalized hepatic cell line Fa2N-4. Choline uptake was saturable and mediated by a single transport system, which are both Na⁺-independent and pH-dependent. RT-PCR and western blot analysis revealed that choline transporter-like protein 1 (CTL1) and CTL2 were mainly expressed. Immunocytochemistry revealed that the plasma membrane showed a considerable level of immunoreactivity with CTL1. In contrast, CTL2 immunoreactivity was recognized in mitochondria.

We conclude that extracellular choline is mainly transported by CTL1 that relies on a directed H⁺ gradient as a driving force. Furthermore, CTL2 may be the major site for the control of choline oxidation in mitochondria.

P3–57.
Blood urea nitrogen is a predictor of adverse outcomes in overweight or obese patients with acute decompensated heart failure

Background: Elevated blood urea nitrogen (BUN) has been shown to be closely related not only to renal dysfunction but also neurohumoral activation in heart failure (HF), and also reported to predict poor in-hospital and longterm outcomes in HF patients. In obese HF patients who have relatively lower BNP levels and higher neurohumoral activation, BUN may be greater significance.

Methods and Results: We enrolled 53 overweight or obese patients with acute decompensated HF. They were divided into 2 groups according to BUN at discharge (group L; BUN <25 mg/dL n=37, group H; BUN >25 mg/dL n=16). The composite endpoints were all cause death and re-hospitalization for HF were compared between the groups. Group H had significantly higher older age, lower hemoglobin levels at discharge. During a median follow-up period of 438 ± 408 days after discharge, the Kaplan–Meier curve showed group H had worse prognosis compared with group L (Log–rank test p<0.001). Multivariate analysis showed that BUN at discharge was a predictor of the composite endpoints (hazard ratio, 1.25; 95% confidence interval, 1.03 to 1.52; p<0.03) independent of other parameter of renal function.

Conclusion: In overweight or obese patients with acute decompensated HF, BUN at discharge may be a useful predictor for adverse outcomes.