The Consortium for Radiologic Imaging Studies of Polycystic Kidney Disease (CRISP) followed 241 subjects (15-46 y.o.; GFR at beginning >70 ml/min) done between Jan 5, 2001 and Aug 26, 2005 with baseline and three yearly visits taking measurements of total kidney volume and GFR. A subset (131) of these patients had Renal Blood Flow measurements by MRI. Analysis of the baseline predictors of disease progression in this subset of 131 showed that total kidney volume, Renal Blood Flow, and urinary sodium excretion independently predicted increases in Total Kidney Volume during 3 years of follow-up.

The association between urinary sodium excretion and structural progression of the ADPKD suggests that sodium intake affects the progression of ADPKD. To further evaluate this hypothesis we extended the analysis to include the entire CRISP I patients for an extended study called CRISP II. Variables included age, gender, body mass index, hypertension status, mean arterial pressure, estimated protein intake, ln total kidney volume, GFR, serum uric acid, HDL and LDL cholesterol, urine volume, ln urine albumin excretion, urinary sodium excretion, and excretions of urea nitrogen and citrate.

Only those variables with p-value <0.1 were further considered in the subsequent model building. Stepwise selection was used to obtain a final main effect model. Baseline ln total kidney volume and urinary sodium excretion were positively and HDL cholesterol negatively associated with ln total kidney volume slopes during baseline-YR3 in CRISP I (n=201, P values <0.001, 0.003 and 0.004, respectively) and during baseline-YR6 in CRISP II (n=140, P values 0.001, 0.020 and 0.004, respectively). Interestingly, urinary sodium excretion has remained mostly constant for individual patients indicating that it is a relatively fixed trait.

Conclusion: Dietary sodium intake and HDL cholesterol are potentially modifiable phenotypic traits that likely contribute to the progression of ADPKD. The underlying mechanisms deserve investigation.