Summary

Background
Previous studies have shown that reduced renal plasma flow (RPF) may play a role in progression of renal disease in autosomal dominant polycystic kidney disease (ADPKD). Tolvaptan, a vasopressin 2 antagonist, reduces growth of total kidney volume and slows the decrease in estimated glomerular filtration rate (eGFR) in ADPKD. The purpose of this randomized, cross-over, double-blind, placebo-controlled study was to investigate if acute tolvaptan treatment increases RPF in ADPKD patients.

Methods
Eighteen ADPKD patients (chronic kidney disease stages I-III) were investigated twice (min. 10 days apart) after acute treatment with either tolvaptan 60 mg or placebo. Two hours after treatment RPF and GFR were estimated by Technetium-99m diethylenetriamine penta-acetic acid (99-mTc-DTPA) renography. During the examination day, central and brachial blood pressures (BP) were measured using Mobil-O-Graph□ PWA. We also measured plasma concentrations of vasopressin (p-AVP), renin (PRC), angiotensin II (p-AngII) and aldosterone (p-Aldo), urine excretion of aquaporin 2 (u-AQP2), urine output (OU), urine osmolality (u-Osm) and fractional excretion of sodium (FENa).

Results
99-mTc-DTPA renography showed a similar RPF (673 ± 262 ml/min after tolvaptan vs. 650 ± 209 ml/min after placebo, p = 0.571) and GFR (78 ± 26 ml/min after tolvaptan vs. 79 ± 21 ml/min after placebo p = 0.774) after tolvaptan and placebo treatment. P-AVP and UO increased and u-Osm decreased after tolvaptan and remained unchanged during placebo. Systolic BP tended to decrease during renography during tolvaptan. Very small or insignificant changes were seen in PRC, p-AngII and p-Aldo.

Conclusions
Acute tolvaptan treatment did not change renal hemodynamics in ADPKD.