**Background:** Urolithiasis is common in developed countries with a significant recurrence rate. Hypocitraturia and hypercalciuria have been reported as the most prevalent risk factors. Citrate is a strong crystallization inhibitor and citrate supplementation has been introduced for metaphylaxis in recurrent kidney stone formers (rKSF) with hypocitraturia and normocitraturia. However, beyond its effects on urinary stone parameters only few studies have investigated the impact of citrate on other metabolic pathways that have been reported to be affected by citrate. Vitamin D has been shown to increase urinary citrate excretion by reducing mitochondrial citrate metabolism. Endogenous citrate has been described to have a positive regulatory effect on lipogenesis and a negative regulatory effect on glycolysis. Thus, the aim of this study was to evaluate the impact of oral citrate therapy on the urinary stone risk profile of recurrent calcium stone formers as well as on the glucose and lipid metabolism. Additionally, we wanted to test if 1,25-(OH)2-Vitamin D3 levels positively correlate with urinary citrate excretion.

**Methods:** This study is a retrospective analysis of prospectively collected data from the Swiss kidney stone cohort. 24h-urine parameters were measured at baseline, after 3 months and one year of therapy. The primary endpoint of this study is the change of urinary parameters after citrate supplementation.

**Results:** 445 participants (mean age 47±14 years, 70.6% male) were evaluated. 88% of stones were calcium-containing, 42% were pure calcium oxalate stones, followed by 2.5% pure calcium phosphate as well as 2.5% uric acid stones. Hypocitraturia was present in 18.4% of rKSF and potassium citrate was administered to 52 patients (11.7%) at a mean dosage of 2523±1173 mg citrate/d. Mean 24h-urine parameters at baseline were as follows: citrate 2.7±1.5 mmol/d, potassium 59.0±24.5 mmol/d, calcium 5.6±3.3 mmol/d, sodium 161.6±79.4 mmol/d, oxalate 0.2±0.2 mmol/d, ammonium 19.0±10.5 mmol/d, magnesium 3.9±1.8 mmol/d, pH 5.9±0.6, volume 1.8±0.8 l/d. Treatment with potassium citrate was associated with significant changes after 3 months in the following urinary parameters: pH (p=0.047), potassium (p=0.046), citrate (p=0.003), magnesium (p=0.015) and volume (p=0.039). Multiple linear regression analysis demonstrated no significant association of 1,25-(OH)2-Vitamin D3 levels with urinary citrate excretion. Exogenous citrate administration had no effect on cholesterol, fasting glucose and HbA1c after 3 months and 1 year.

**Conclusions**: Citrate supplementation in Swiss rKSF resulted in a significant increase of urinary citrate, magnesium, and potassium excretion as well as urinary pH resulting in a beneficial change of urinary risk profile parameters. There was a trend towards lower urinary calcium and phosphate excretion after citrate supplementation, potentially due to favorable effects of systemic alkalinization on bone turnover and binding of calcium to citrate in the gastrointestinal tract. 1,25-(OH)2-Vit. D3 levels were not associated with urinary citrate excretion. Cholesterol, fasting glucose and HbA1c were not affected by citrate therapy after 3 months and 1 year.