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Dietary Citrate Treatment of Polycystic Kidney Disease in Rats

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Key Words

- Autosomal-dominant polycystic kidney disease
- Calcium citrate
- Sodium citrate
- Transforming growth factor- β
- Han:SPRD rat

Abstract

Progression of autosomal-dominant polycystic kidney disease (ADPKD) in the heterozygous male Han:SPRD rat is dramatically slowed by ingestion of potassium or sodium citrate. This study examined the efficacy of delayed therapy with sodium citrate, the effect of sodium citrate therapy on kidney cortex levels of transforming growth factor- β (TGF- β), and the response to calcium citrate ingestion. Rats were provided with citrate salts in their food, and renal clearance, blood pressure, blood chemistry, and survival determinations were made. Sodium citrate therapy was most effective when started at age 1 month, and delay of therapy until age 3 months produced no benefit. Kidney cortex TGF- β levels were elevated in 3- and 8-month-old rats with ADPKD, but not in 6-week-old rats. Sodium citrate treatment, started at age 1 month, lowered TGF- β levels to normal in 3-month-old rats, but this is probably not the primary mechanism of citrate's beneficial effect. Calcium citrate had only a modest effect in preserving glomerular filtration rate. Effective treatment of ADPKD in this rat model requires early administration of a readily absorbed alkalinizing citrate salt. Existing data on ADPKD patients on vegetarian diets or with kidney stones should be studied in light of these findings.

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