

***Oxalobacter formigenes*: A Potential New Therapy for Primary Hyperoxaluria**

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Primary hyperoxaluria (PH), a rare genetic disease, results in marked endogenous oxalate overproduction by the liver. Resulting hyperoxaluria causes recurrent urolithiasis or progressive nephrocalcinosis, as well as calcium oxalate crystal deposition in the renal parenchyma, which leads to end-stage renal failure. Dialysis is unable to prevent systemic oxalate deposition. Combined liver/kidney transplantation is required in the majority of patients. Mammalian intestine has been shown to play an important role in oxalate homeostasis and is believed to both absorb and secrete oxalate. Enteric elimination of oxalate can take place through passive diffusion or active secretion involving anion transporters of the SLC26 family.

Oxalobacter formigenes, an anaerobic bacterium that degrades oxalate, naturally colonizes the colon of most humans. A pilot study showed that *O. formigenes* orally administered as a frozen paste or an enteric-coated capsule (Oxabact™, OxThera AB, Uppsala, Sweden) for 4 weeks significantly reduced urinary and plasma oxalate levels in patients with PH and varying degrees of renal function. Thus, *O. formigenes* is a potential new therapy for PH. A double blind, placebo-controlled trial of Oxabact has recently been conducted. Forty-three patients with primary hyperoxaluria from nine clinical sites in Europe and the United States, who met inclusion criteria, were studied. Outcomes of the clinical trial will be presented.

*On behalf of Bernd Hoppe and investigators of the Oxalobacter Study Group.
