

# Ingested *Eucalyptus viminalis* implicated in oxalate nephropathy of marmoset monkeys

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The laboratory colony of Common Marmosets (*Callitrix jacchus*) was founded at the University of New England in 1992. Prior to the oxalate poisoning that we report here, only 5 deaths had occurred and at the beginning of 2002 the colony comprised 6 males and 11 females. Seven of these died between February and August 2002. Death was attributed to kidney failure from an oxalate-induced nephropathy. The source of oxalate was identified as *Eucalyptus viminalis*. Eucalypt branches, both dried and freshly cut from various sources had always been provided for climbing, and just before January 2002 the branches used were *E. radiata*. Branches from a new source, a recently pruned *E. viminalis*, were put in the marmosets' cages in early January 2002 and the marmosets were observed to chew on leaves and bark. The deaths commenced in February and the branches were removed in March. Urinalysis indicated that all the surviving marmosets had chronic renal damage, and as a result deaths continued until August 2002.

The marmosets were housed in rooms indoors and also had access to outdoor cages. They were fed a selection of foods once daily: orange, beans, pear, peach, banana, sultanas, peanuts, yoghurt, cheese, boiled egg, bread, dog biscuits, mealworms, Nutri-Grain®, Pentavite®, banana cake and meatloaf. Sufficient food was provided to allow the marmosets to feed *ad libitum*. Water was also available *ad libitum*. No dietary changes had been made prior to the deaths. The only management change was the introduction of the new eucalypt branches.

Post-mortem examination of the affected marmosets revealed enlarged pale kidneys. Histopathologically the kidneys were undergoing a

chronic degeneration with tubular and glomerular atrophy, chronic inflammatory cellular infiltrates and fibroplasia. Numerous crystals were observed within dilated tubules, and within macrophages and epithelial cells. These were confirmed as calcium oxalate through use of special stains (Von Kossa's and Pizzolato's) as well as the use of cross-polarised filters. High concentrations of crystals were observed in the 3 monkeys that died before the suspect branches were removed. Lower concentrations were observed in the kidneys of those that died later. Further, a kidney from a marmoset that died in May 2001 before the outbreak had no oxalate crystals.

Crystals were observed in high concentrations in the suspect leaves and bark. They were confirmed as calcium oxalate: birefringent under polarised filters, soluble in hydrochloric acid (50%) and insoluble in acetic acid (45%).

Urine samples were collected from the surviving marmosets 80 and 122 days after contact with the suspect branches. Urinary protein levels were elevated (mean 500mg/dl), and ketone levels were elevated in some. Three new marmosets were introduced into the colony in 2003. Analysis of their urine showed low protein (mean 10 mg/dl) and no ketones.

Insoluble calcium oxalate crystals can precipitate in the kidneys of animals and man. The resulting nephrosis can result in death, either acutely or from chronic renal failure. The source of soluble oxalate can be either endogenous or exogenous. The epidemiology of this outbreak suggests an exogenous source and there is strong evidence it was the bark and leaves of *Eucalyptus viminalis*.