

## DEATHS FROM THIAMINE DEFICIENCY IN HONEYEATERS FED IN A SUBURBAN GARDEN

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### SUMMARY

An investigation into the deaths of 40 garden-fed honeyeaters, predominantly Red Wattlebirds *Anthochaera carunculata*, showed that their diet was mainly cane sugar. Autopsy and laboratory investigations revealed no obvious cause of death. The epizootic rapidly subsided after the addition of thiamine to the garden feeds. It was concluded that death was due to thiamine deficiency, resulting from feeding refined sugar, which contains no thiamine, to honeyeaters.

### INTRODUCTION

On 24 September 1968, Mrs W. K. Walker reported to the author that approximately 40 honeyeaters had died in her garden at St Georges in suburban Adelaide, South Australia, during the preceding two weeks. The birds were two Eastern Spinebills *Acanthorhynchus tenuirostris*, four New Holland Honeyeaters *Phylidonyris novaehollandiae*, three Little Wattlebirds *Anthochaera chrysoptera*, and some 30 Red Wattlebirds *Anthochaera carunculata*. Inspection of the garden on 29 September revealed approximately 40 Red Wattlebirds and 40 New Holland Honeyeaters in the feeding area. There were water points, pieces of suet and some 20 cup-shaped dishes containing cane sugar. The Red Wattlebirds were gorging themselves with sugar and then taking a sip of water before flying off. All birds appeared to be in good condition and normally active. Two pairs of Red Wattlebirds were nesting in a jasmine tree within 25 cm of each other.

The mode of death of all birds followed the same pattern. Apparently healthy birds would suddenly fall from their perches, some grasping it momentarily with one foot before falling to the ground. When suspended by one leg, falling, or when on the ground, a bird would be attacked by several conspecifics. It would recover rapidly and fly to a tree only to have a convulsion and fall to the ground again. If rescued from the others, it would suffer a series of fits and die within 12 hours.

The diet and mode of death suggested that deficiency of thiamine (Vitamin B1) was the cause of the problem. Mrs Walker had ascertained that no near neighbours had used poisons or insecticide sprays and that in two other nearby gardens where honeyeaters had been fed there had been no deaths. She had been feeding birds for six years without observing any mortality. Originally she had given them 2 kg of sugar and 2.8 litres of syrup containing 0.5 kg of brown sugar, 60 g of honey and 30 g of treacle, a syrup that contained thiamine. Because the birds seemed to prefer the sugar she had stopped giving the syrup a few weeks before the deaths occurred. At that time, they were eating 3.18 kg per day of solid refined sugar, which contains no thiamine, the containers being replenished several times a day.

### AUTOPSIES

Two Red Wattlebirds were examined shortly after death by the Institute of Medical and Veterinary Science, Adelaide.

Autopsy revealed no abnormality. The spleens were not enlarged and the brains were normal. At the microscopic level, the only abnormality was found in the livers, which showed large amounts of iron-containing material and bile pigmentation of liver cells, changes that suggested haemolysis. Bacterial cultures were all negative. Organs from one bird examined by the South Australian Government's State Chemistry Laboratories showed insignificant amounts of DDE (0.2 ppm) and no other organochlorides or organophosphates were detected.

One bird had blood withdrawn from it before death. The red blood corpuscle cholinesterase was 0.9 units/mL. A healthy bird trapped in a different location had a level of 1.7 units/mL. This difference when assessed in relation to the known variation in other vertebrates (Altman & Dittmer 1974:111) was not sufficient to indicate organic phosphate poisoning. The blood pyruvate

level in one bird was 36 mg/L. This is high when compared with levels in humans (Diem & Lentner 1970: 607) and in keeping with thiamine deficiency but a single reading without controls is of little value. Facilities for estimation of erythrocyte transketolase were not available at that time.

#### SYMPTOMS AND TREATMENT OF SICK BIRDS

In an attempt to save the lives of sick birds, two Red Wattlebirds were treated medically.

Bird One was seen to have had several convulsions during the hour before its capture. It was an immature bird in good plumage and condition. It made no attempt to fly when perched on the hand and once rotated its head in a circular movement. Breathing was normal and the feathers were not ruffled. It was given 10 mg of thiamine by injection into the pectoral muscle 90 minutes after the first fit.

During the next two hours, during which no fits occurred, it appeared alert and attempted to fly from the box, in which it had been held, when the lid was removed. When caught it struggled momentarily. On being held in various positions the head was always maintained horizontally suggesting an intact cerebellum. It refused food. It then became listless, shut its eyes and lay quietly on its side breathing regularly and deeply. Thirty minutes later it had assumed the upright position and appeared to be asleep. When awakened it was alert. Ten hours later, the following morning, it looked well but when picked up the head rolled around several times with the eyes closed and the feet clenched. It rapidly recovered and when the beak was dipped into a honey-water mixture the bird drank. Within five minutes of feeding a further fit occurred. The wings flapped rapidly, the legs moved spasmodically whilst the head was retracted and the tail brought up over the back to meet the head (*opisthotonus*). This state persisted for 30 seconds after which the bird was in coma with deep respirations. Eight hours later, when picked up the bird moved its head slowly in circles with the eyes closed and, as previously, this was followed by a fit. Three more fits occurred during the next two hours. It was then given 2 mg of phenytoin, an anti-convulsant drug, by intramuscular injection. There were no further fits that evening

and the following morning it was perched on the edge of a box in a cage and drank syrup and ate minced meat put into its bill. However, two hours later it suffered a spontaneous fit. It was then given 5 mg of thiamine and 2 mg of phenytoin intramuscularly. On regaining consciousness routine types of tests for sight and sensation were normal against human standards. Several further fits occurred during the morning and it died after a rapid series of fits (*status epilepticus*) 51 hours after its capture.

Bird Two was caught after the first observed fall. It was treated with thiamine (10 mg) and phenytoin (5.5 mg parenterally in three doses during the first day). On the second day it drank from a spoon voluntarily. Two fits occurred during this day, each followed by the administration of phenytoin. On the third day it was given 2 mg phenytoin prophylactically. It appeared to be well, drank well and ate minced meat. On the fourth day it ate and drank well and escaped from its cage and flew about the room. On the fifth day it behaved like a normal bird but at 1800 hours it suffered a minor fluttering of the wings. A further injection of 2 mg phenytoin was given. On the sixth day it weighed 91 gm and was banded in anticipation of release. On the eighth day it suffered a prolonged fit and slept all day after treatment but recovered to eat and drink that evening. On the ninth day it suffered a series of fits and died.

Because the mode of death was similar to that of untreated birds no autopsies were carried out.

#### MANAGEMENT OF THE EPIZOOTIC

Powdered thiamine hydrochloride (100 mg) was intimately mixed with each kg of cane sugar, and syrup made of one part of sugar, one of molasses (which contained 2.1 mg of thiamine/100 g) and eight parts of water, was supplied in addition to the enriched sugar. The following day no birds died. On the second day one Red Wattlebird died.

On the third day, the only Red Wattlebird with a leg band was seen to fall twice. It was seen on the following day and again two days later, apparently well and feeding. Nine days after modification of the diet one more bird had several fits and died. No further deaths were recorded over the following three months.

## DISCUSSION

Paton *et al.* (1983) investigated the cause of winter mortality of Red Wattlebirds in Melbourne. They concluded that death was probably due to thiamine deficiency, attributable in part to the scarcity of large insects, the main source of the vitamin, during the winter. Forty-seven Red Wattlebirds were found in convulsions before death but no other species were found.

In the present outbreak the number of dead Red Wattlebirds was at least seven times greater than the number of New Holland Honeyeaters, although the birds were present in approximately equal numbers. This suggests that Red Wattlebirds are more susceptible to thiamine deficiency or, perhaps, more likely, that the smaller, more active New Holland Honeyeaters foraged further afield for insects. One could assume that insects in the immediate vicinity were few because of the abnormally high density of birds. As each bird requires 200 kJ/day, 3.18 kg of sugar would feed 165 Red Wattlebirds/day (Paton *et al.* 1983).

An unexpected finding at autopsy was the iron overload and bile staining of liver cells. In thiamine-deficient animals, haemorrhages of the intestines and liver have been found (Marks 1968). This was also the case in some of the birds reported by Paton *et al.* (1983). In the cases reported here, haemorrhages were not found but no search for them was made and it is possible that multiple small submucosal haemorrhages may have occurred. Haemolysis of the red cells from such haemorrhage could lead to all the changes found in the livers.

The responses of two sick birds to treatment were disappointing, although life appeared to

have been prolonged. Paton *et al.* (1983) had limited success at rehabilitating sick birds but only after thiamine had been injected intramuscularly. Recovery had been reported by Brin *et al.* (1965) after administration of thiamine in symptomatic mammals and birds. The human equivalent, namely Wernicke's disease, responds to treatment but large doses of thiamine must be given early in the disease and intravenously (Victor & Adams 1983: 2115).

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