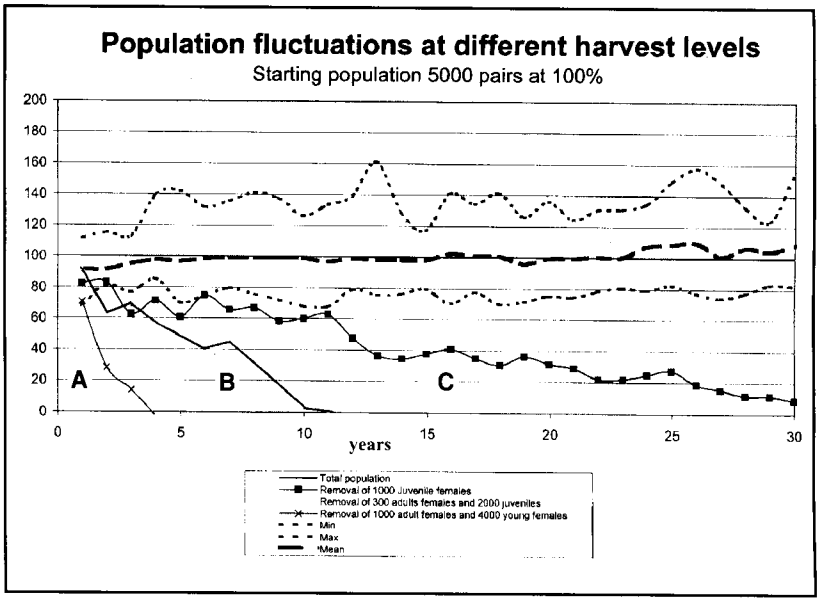
A) 1000 adult females and 4000 juveniles removed from world population annually

B) 300 adult females and 2000 juveniles removed from wild population annually

C) 1000 juvenile females and no adults removed from wild population annually



Poisoning by acetylcholinesterase inhibiting pesticides in free-ranging raptors: a case reported from Saudi Arabia

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Poisoning of raptors by acetylcholinesterase (ChE) inhibitors, particularly organophosphorous and carbamate compounds are frequently encountered in Europe (Keymer *et al.* 1981; Lumeij *et al.* 1993). Although secondary poisoning with ChE inhibitors of scavenging birds of prey feeding on poisoned birds and rodents has been documented in Israel (Mendelssohn & Paz 1977), reports of pesticide poisoning of free-ranging birds in the Middle East remain rare, being most probably overlooked. Recently we have reported a case of pesticide poisoning in a free-ranging lappet-faced vulture (*Torgos tracheliotus*) which to our knowledge was the first documented case of pesticide poisoning of a bird of prey in Saudi Arabia (Ostrowski & Shobrak 2001). This article summarises a number of points that may help veterinarians in the Middle East recognize and treat this intoxication in birds of prey.

Diagnosis of ChE inhibiting pesticide poisoning in the live bird is usually based on history, clinical signs and depressed plasma cholinesterase levels (Meerdink 1989).

History
In the Middle East a history of pest control pesticide spraying over crop fields or over entire portions of territories for locust control must draw the attention in case of abnormal mortality with ataxic symptoms in free-ranging raptors. In the case we described in a free-ranging lappet-faced vulture two organophosphorus pesticides, fenitrothion and chlorpyrifos, had been used to control outbreak of locusts. The recommended rate of fenitrothion application (250 to 300 g/ha) was near that shown to cause mortality in birds (Steedman 1988) and chlorpyrifos, at a rate of 250 g/ha, might also pose a hazard to migrating birds (Smith 1987).

Clinical signs
ChE inhibiting pesticide poisoning in raptors appears clinically different than is typically described for mammals (Porter 1987). Clinical signs are not pathognomonic. They include ataxia, spastic nictitans, a detached attitude, inability to fly and occasionally convulsions (Dumonceaux & Harrison 1994). Birds are 10 to 20 times more susceptible to ChE inhibitors than mammals, and young animals appear to be even more susceptible (Humphreys 1988).

Laboratory diagnosis
Organophosphate and carbamate compounds owe their toxicity to their ability to inhibit ChE, the enzyme that hydrolyses acetylcholine. Theoretically organophosphate and carbamate poisoning can be diagnosed by measuring plasma cholinesterase activity. In practice however definitive diagnosis can be difficult to perform because very little has been published on normal plasma cholinesterase values in raptors (Porter 1987; Hill 1988), analytical method used could markedly affect the results (Fairbrother & Bennett 1988) and other factors (end-stage liver disease, heavy metal poisoning) can also depress plasma ChE activity (Hill & Fleming 1982).

It is therefore important to duplicate analyses in the same laboratory and test concomitantly a negative control bird

(long-term captive) preferably of the same species. In the case we described ChE activity was depressed by 245% compared to the value 20 days after treatment, and by 290% compared to the value measured in a captive griffon vulture (*Gyps fulvus*) tested the same day.

Treatment

Specific treatment relies on the administration of atropine sulfate that blocks the muscarinic effects at the nerve synapsis. Dosage of 1% atropine sulfate administered intramuscularly is 0.5 mg/kg (Porter 1993). A higher dosage can be used according to the severity of clinical signs. We used about 1 mg/kg upon arrival as an initial dose in the lappet-faced vulture we treated, and then 0.5 mg/kg at day 3. Improvement was immediate and spectacular. Other symptomatic and supportive treatment should also be provided.

Conclusion

Populations of India's commonest *Gyps* vultures have recently dramatically declined due to a mysterious disease (Prakash 1999). Sick birds appeared lethargic with drooping heads and wings, all symptoms compatible with a ChE poisoning in bird. However, recent investigations seem to have ruled out anti-ChE pesticides as the cause of the disease and instead point towards an infectious cause (Prakash et al. 2002). People have conceived that the disease that seem to affect all Gyps vultures could spread from South Asia throughout the Middle East and the Old World (Prakash *et al.* 2002). It is important therefore that veterinarians throughout the Middle East play a role of epidemiological sentinels and investigate to the best of their capacities any sick vulture with a lethargy syndrome. Should any case arise, anti-ChE poisoning will have to be ruled out.

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