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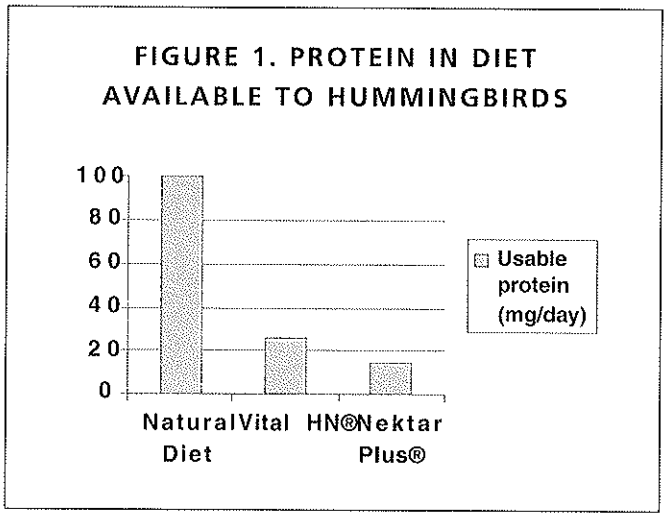
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Improving Detection of Pesticide Poisoning in Birds ... Meeting the Protein Requirements of Adult Hummingbirds in Captivity ... Psychological Aspects of Euthanasia in Wildlife Rehabilitation ... Downy Woodpecker Success Story ... Keeping Wildlife Wild ... Living with Canada Geese ... CD and Book Reviews ... and more

ABOUT THE JOURNAL . . .

THE JOURNAL OF WILDLIFE REHABILITATION is designed to provide useful information to wildlife rehabilitators and others involved in the care and treatment of native wild species, with the ultimate purpose of returning them to the wild. The Journal is published by the International Wildlife Rehabilitation Council (IWRC), which invites your comments on this issue. Through this quarterly publication, rehabilitation courses offered in numerous locations, and an annual conference, IWRC works to disseminate information and improve the quality of care provided to wildlife.

ON THE COVER: This skull of a Swainson's hawk was found in a field in Argentina, where approximately 5% of the worldwide population was killed in one season by the pesticide monocrotophos. [PHOTO: Scott Weidensaul. Printed with permission.]. BELOW: Comparison of hummingbird's natural diet to two commercial diets. [see "Meeting the Protein Requirements of Adult Hummingbirds in Captivity," page 14.]



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EDITORIAL

The Only Constant Is Change

From time to time, many people find themselves questioning whether their lives have become a comfortable, predictable routine, or if they are simply just stuck in a rut. Organizations also find themselves asking the same sort of questions. If an organization is trying to meet the needs of its members in a changing world, to stay the same is to fail.

IWRC is in the midst of a number of major changes, many requested by members, some planned by the board of directors, and a few big ones thrust upon the organization by circumstances beyond our control. The net result is an organization that has been jarred awake by world events, and an organization that is surging forward to meet new challenges.

One of the biggest changes at IWRC took place on June 28, when Executive Director Mary Reynolds left the staff after more than 13 years of service to the organization. Mary had been a rock of steadiness in our national office. Her departure presented the board with the need to make some big decisions about the future of IWRC, then find a new director who could implement our plans for the future and move us forward.

In trying to balance the immediate operational need for leadership in our office with the need to get member input and make thoughtful, long-term, strategic decisions, the board decided to hire an interim director to keep us on track and help us maintain the momentum we have been building over the last several years.

We are delighted to announce the appointment of **Jennifer Bennett** to this position. Jennifer has worked for nearly eight years in the field of wildlife care, holding positions that range from volunteer to executive director in various wildlife rehabilitation organizations around the San Francisco Bay Area. She is a longtime member of IWRC and has been both a participant and a presenter at many of our conferences. Perhaps more important are her exceptional skills in communication, organization management, and human relations. She brings new skills, new enthusiasm, and new energy to our national office. In short, she has it all.

Jennifer's appointment will run through the end of the year, during which time the board will evaluate IWRC's long-term needs and conduct a national search for a permanent director.

While the new face of IWRC is an obvious change, there is even more going on. Some of the changes are primarily internal ones that won't be obvious to members but that will result in far better service and support. The office recently installed a new computer system and is dramatically upgrading its membership and information management software. This has already increased the efficiency of the office, and will soon allow us to provide entirely new services.

One new service to our members is the *E-zine*, the online magazine, which can be found on IWRC's website, www.iwrc-online.org. The *E-zine* will replace the costly and inefficient newsletter that has outlived its usefulness. A small percentage of our members do not have access to the Internet, so some provision will be made to provide the new magazine in hard copy, at least for the time being.

In spite of rumors to the contrary, IWRC will continue to publish the *Journal of Wildlife Rehabilitation* in its current format.

In an exchange of correspondence I recently had with a member in Florida, I pointed out that your IWRC board is aware that we will never *please* everyone, but we are committed to *serving* everyone. In the aftermath of the terrible events of September 11, 2001, IWRC has faced many of the same challenges that have rocked, and occasionally crippled, other nonprofit organizations. We have had to make some hard choices to keep IWRC's programs and service alive and well.

I am pleased to report that, indeed, IWRC is alive and well. There are many changes taking place, but one thing will remain the same. IWRC is committed to the conservation of wildlife and service to those who care for them.

—Edward E. Clark Jr.
President, IWRC Board of Directors

More on *The Origin of Species*

In her recent review of Charles Darwin's classic book, *The Origin of Species ...* (*J. Wildlife Rehab.* [24]4: 34), Kieran Lindsey suggested that reading this almost-150-year-old book "would be well worth the effort." However, given the rapid pace at which the biological sciences have advanced, many people may see little value in reading a scientific book that was published so long ago. Because I share Ms. Lindsey's enthusiasm for this book, I wish to emphasize that even a perusal of this classic work, if not a thorough reading, would be a valuable experience, for several reasons. The process of evolution through natural selection laid out by Darwin in this book is arguably the most important concept in all of biology. But if this isn't enough justification to take a look, perhaps being made aware that natural selection has strong relevance to our daily lives almost a century and a half after the concept of this process was published will pique one's interest.

A brief scan through the book's pages demonstrates how thorough Darwin was in establishing the facts needed to strongly support his ideas. A bit more delving into the book will indicate that the process of evolution via natural selection actually is composed of three seemingly simple steps: (1) organisms produce more offspring than can survive in their habitats; (2) those individuals that are best adapted to their environment survive better and reproduce more than less well-adapted individuals; and (3) because offspring generally resemble their parents (through the mechanism of genetic heredity, which Darwin didn't know about at the time he wrote this book), better adapted individuals tend to produce better adapted offspring.

Thus, through natural selection, as one generation replaces another, organisms become better adapted to their environment, and they change as their environment changes or when they colonize new areas with different environments. How elegantly simple—three basic, indisputable steps to explain how Nature "works." The process of natural selection helps us understand why there is such a grand diversity of species exhibiting a truly amazing variety of lifestyles and inhabiting virtually every potential habitat on earth, from the tubeworms and crabs at the thermal vents deep in the oceans to the flies and spiders high up on the cold, barren slopes of the Himalayan mountains, and even the tiny mites that live in our eyelash pores without us being aware of them!

With even a rudimentary knowledge of the three simple steps of evolution via natural selection, one is left wondering why some people ("anti-evolutionists") want to prevent others from learning about this process. Understanding natural selection is all the more important as we face key challenges of the 21st century—problems whose roots lie in our failure to apply our understanding of a pervasive, natural process elaborated by Darwin many years ago. Some examples:

(1) Through excessive use of antibiotics in our domestic animals and ourselves, several bacteria that cause human diseases inadvertently have been selected by us to become resistant, such that former "miracle drugs" are now ineffective at controlling these pathogens. Similarly, through excessive use of pesticides, many species of insects and other pests inadvertently have been selected by us to become resistant to the once-toxic effects of these chemicals.

(2) Through habitat destruction and other causes, we are reducing populations of organisms (especially wildlife) to the point that they are too small to maintain the genetic variability necessary to produce the "better adapted" individuals that might survive in their changing environments, and thus these species are facing extinction.

(3) Through emissions of carbon dioxide and other "greenhouse" gases, humans appear to be warming the earth at such a rapid rate that many organisms may not be able to adapt rapidly enough to cope with the associated environmental changes.

(4) We are now tampering with the very process of evolution, transferring genes from one species to another, for example to create genetically modified crop plants. Adherents claim that the risks of this approach are minimal relative to the benefits, but the wisdom of such unnatural genetic manipulations has been questioned by others. Apparently, only time will tell the outcome of this grand "experiment" in circumventing natural selection and manipulating evolution.

The process of natural selection underlies each of these and some of the other "challenges" we face as we enter a new millennium, and it behooves us to look back to the 19th century to get some insight into how our purposeful ignorance of such a simple process as natural selection during the last century has brought us so many problems.

Although it is a long read, Darwin's book is certainly worth at least a brief perusal by anyone interested in biology, as well as by parents who want their children to receive an education that includes exposure to the process of evolution and its relevance to modern-day life. After all, the information and ideas provided by Darwin in this book have laid the foundation for our current understanding of evolution that has been attacked by anti-evolutionists. Check out *The Origin of Species ...* and make your own informed decision on this issue.

—Frank Slansky, Ph.D.
Professor of Entomology
University of Florida

Dr. Slansky writes: "I'm not a member of the IWRC, but my wife, Lou Rea Kenyon, is, and although I'm a 'bug man', I assist Lou Rea with wildlife rehab to a considerable degreee. We also work together on projects where insects and mammals overlap, such as bot fly parasites of squirrels.

The Editors encourage readers to respond to the articles, columns, and other items featured in the Journal of Wildlife Rehabilitation. *Please direct general questions and comments to the Editor at runmuki@earthlink.net (USPS address on the journal's inside back cover). To respond to a specific article or column, please contact the author directly. (NOTE: Letters may be edited for clarity and length.)*

Improving Detection of Pesticide Poisoning in Birds

by Pierre Mineau and Kelley R. Tucker

ABSTRACT: Pesticide effects on birds are multiple yet remain poorly understood. We know that birds are important sentinels of ecological health in every environment. Our knowledge of pesticide effects on birds and other wildlife is greatly improved when avian pesticide incidents are properly identified, reported, investigated, diagnosed, and recorded in accessible standardized formats. Properly informed and with resource support, the wildlife rehabilitation community can become a valuable source of information.

KEY WORDS: avian, pesticides, poisoning, organophosphorous, carbamate, anticoagulant, cholinesterase, neuro-toxicity, synapse, 2-PAM, atropine, clinical signs, measurement, monitoring, reporting

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Birds and Pesticides

Birds are an important and visible part of our environment and serve as sentinels of general environmental health. In North America, and in many other developed countries, most bird species are protected from unlicensed taking or kill of individuals. Yet one estimate of pesticide effects on U.S. farmland—considered conservative by many avian biologists and advocates—states that as many as 72 million birds die each year as a result of pesticide exposure (Pimentel 2001). Our ability to refine this estimate or to predict pesticide impacts more accurately is hampered by the difficulty of gathering data, identifying, and assessing pesticide effects on birds in real-life scenarios. Birds are extremely mobile, and it is difficult to exclude them from areas that are treated with pesticides. Whether in agriculture—where bird species attracted to agricultural pests can be economically important for the control of such pests (Kirk et al. 1996, Tucker 2002)—or in forestry, parks, or backyards, birds and pesticides intersect. Birds suffering from lethal or sublethal pesticide effects often escape the notice of humans. When they are noticed, some are brought in to rehabilitation centers. The wildlife rehabilitation community can become a valuable source of information. Research scientists and cooperators from government and industry are working with academic and nonprofit organizations to provide rehabilitators and wildlife professionals with the tools and resources to improve their knowledge of, and ability to report, pesticide incidents.

This paper will discuss the science behind the effects of pesticides on birds, with special emphasis on cholinesterase inhibition, and provide practical information for rehabilitators and others interested in responding to suspected avian pesticide poisonings. While the focus here is birds, much of the information can easily be applied to other taxonomic groups. Mammals, fish, amphibians, and reptiles have all been reported as casualties following pesticide field trials, and biologists around the world are sounding an alarm about the state of reptile and amphibian diversity and population numbers. Birds clearly are not the only taxon under fire.

Cholinesterase-inhibiting Pesticides

According to the Federal Insecticide, Fungicide, and Rodenticide Act—the governing legislation in the United States for pesticide use and regulation—"pesticide" is an umbrella term for a range of "substances or mixture of substances intended for preventing, destroying, repelling, or mitigating" any organism deemed to be a pest. Pesticides are most often categorized by target pest (e.g., herbicide, insecticide, fungicide, rodenticide, avicide) or by chemical class, a somewhat arbitrary system that groups chemicals according to their known biochemical mode of action and common chemical features (e.g., organochlorine, organophosphorous, or carbamate compounds, synthetic pyrethroids, triazine herbicides, etc).

Pesticides are designed to disrupt vital biological processes of a target organism—for example, photosynthesis in plants or blood clotting in mammals. These processes are often shared by nontarget organisms, thus creating a realm of risk. In the most general terms, risk to any specific nontarget organism is assessed by looking at the toxicity of the substance and the probability and nature of exposure of the organism to the substance. In the end, any estimated risk is weighed against the benefits of that same pesticide's prescribed use. Pesticides can be valuable tools in food or fiber production, habitat restoration, disease control, and indoor pest control—more so when used in the context of a well-planned "integrated management" program that emphasizes monitoring and includes nonchemical alternatives.

The importance to agriculture of the two main classes of "cholinesterase-inhibiting insecticides," the organophosphorous (OP) and carbamate (CB) products, is undeniable. The use of these products, however, has not been without problems. Their toxicity to birds has recently been compiled (Mineau et al. 2001). In North

America, it is estimated that since 1965, about 3% of bald eagles (*Haliaeetus leucocephalus*) examined by the U.S. Fish and Wildlife Service were poisoned by cholinesterase inhibitors (Franson et al. 1995). This is a known underestimate because the measurement of cholinesterase levels, the primary diagnostic tool, did not become routine until the early 1980s. The 12% calculated for red-tailed hawks (*Buteo jamaicencis*) between 1975 and 1992 (Franson et al. 1996) is probably closer to what we can reasonably expect with either species. It has been suggested however, that these types of estimates are all underestimates of the true impact of pesticides (Porter 1993, Mineau et al. 1999). In all diagnostic centers involved in assessing wildlife incidents, the investigation seldom goes further than establishing the primary cause of death. The most frequent causes identified are invariably trauma, often from impacts with static objects or motor vehicles and electrocutions. The possible contribution of sublethal pesticide intoxication to this type of mortality is very real and will be discussed later.

On a regional level, the importance of poisonings caused by cholinesterase inhibitors can be substantial and may account for a large proportion of all raptor intakes at select rehabilitation centers (reviewed in Mineau et al. 1999). Therefore, the diagnosis of intoxication by a cholinesterase-inhibiting agent (OP or CB) is currently one of the most important skills needed for the investigation of pesticide effects on wildlife. This may change in the future as a number of OP and CB products and use patterns are phased out. There are special problems associated with replacement chemicals that will be discussed briefly below. Although the discussion so far has emphasized birds of prey, intoxications with OPs and CBs are clearly not restricted to this group. Large numbers of songbirds are routinely killed when ingesting treated seed, granular insecticides, or contaminated seeds or insects (e.g., Mineau 1993, Friend and Franson 1999), but their small body size means that these incidents may easily be overlooked and/or may rarely involve rehabilitation. Waterfowl are also likely to be poisoned, especially when they graze on treated crops; their larger size means they are more likely to come into rehabilitation centers.

Clinical Signs Associated with OP and CB Poisoning

Although there are well-described clinical signs that are typical of poisoning by a cholinesterase (ChE) inhibitor, clinical signs can be so variable as to obscure diagnosis. A basic understanding of the mode of action of anticholinesterases helps us to understand this variation.

The synapse

Nerve impulses rely on chemical messengers that travel from cell to cell. No chemical messenger is more important than acetylcholine. Acetylcholine-mediated pathways (or cholinergic tracts) occur in the central nervous system (CNS), in the peripheral nervous system, and at neuro-muscular junctions. There are few physiological processes that are not affected by acetylcholine at one point or another. Acetylcholine is released in the synapse, or gap, between the two communicating cells and stimulates the "receiver" cell. The proper functioning of the system requires acetylcholine to be removed quickly from the synapse once it has performed its function. This is accomplished by the enzyme acetylcholinesterase (AChE). If acetylcholine is not removed but allowed to pool in the synapse, the receiver cell, whether a nerve cell or muscle fiber, will be overstimulated. As it tires (because it is unable to repolarize), it may shut down altogether, preventing further impulses to pass. It is in part because of this variation of effect at the synapse (from initial overstimulation to eventual blockage) that clinical signs of poisoning can be variable and at times contradictory.

OP and CB pesticides have a high affinity for AChE and prevent it from acting on acetylcholine. The main difference between OPs and CBs is that, in the case of the former, the bond between the pesticide and the enzyme is very strong and quasi permanent. Recovery of enzyme activity is generally accomplished by synthesis of new AChE, although release of the enzyme from the OP can be hastened with drugs during rehabilitation (see below). The bond between CBs and AChE is weaker and can be broken by a simple reaction with water (hydrolysis). Upon exposure, a CB is every bit as dangerous to a bird as an OP of equivalent toxicity. However, birds surviving an intoxication with a CB make a much faster recovery than those exposed to an OP. The rapid hydrolysis of the CB-AChE bond means that such intoxications are more difficult to diagnose unless samples are taken early and preserved properly (see below).

Clinical signs and why they are so variable

In the somatic nervous system that controls voluntary muscle movement, overstimulation resulting from pooled acetylcholine typically gives rise to tremors, muscle twitches and piloerection, as well as paralysis resulting in ataxia. More rarely, the animal may convulse. Cholinergic tracts are also important to both the parasympathetic and sympathetic autonomic nervous systems but especially to the former, where they conduct impulses from the neural ganglia to a multitude of effector organs such as the heart, endocrine glands, and digestive system. Because the autonomic nervous system is subject to constant adjustment through feedback mechanisms, intoxication with a cholinesterase inhibitor sends the poisoned organism into a veritable roller-coaster ride. For example, individuals may show alternating constriction or dilation of the pupils, speeding up or slowing down of the heartbeat, etc. Also, because the somatic and autonomous systems react to different levels of cholinergic stimulation, some doses of an anticholinesterase may produce apparently opposite signs (e.g., contraction of the striated muscles involved in locomotion and simultaneous relaxation of the smooth musculature leading to a flaccid gut and food impaction). The rate at which the individual was exposed to the pesticide is as important as the dose itself. Typically, gradual exposure allows the individual to compensate and tolerate a higher dose than if the exposure was a single large dose.

Finally, different cholinesterase inhibitors have different properties that may dictate which clinical signs are expressed. Some pesticides are directly active on synapses, others need to be metabolized to the active molecule; some pass readily into the brain (with obvious effects on the CNS), others have difficulty crossing the blood-brain barrier and therefore show more "peripheral" effects. Porter (1993) cautions that many of the "classic signs" of parasympathetic stimulation reported from

standard toxicology texts may not be seen in poisoned raptors—and certainly not with any consistency. Shimmel and Snell (1999) similarly comment on the difficulty of arriving at a conclusive diagnosis without chemical or biochemical laboratory backup. Nevertheless, the following list modified from Grue et al. (1991) summarizes the signs noted by Hudson and colleagues (1984) following dosing of various bird species with a variety of cholinesterase-inhibiting agents:

- ataraxia (induced tranquility), lethargy
- ataxia (incoordination of muscular action)
- blindness
- convulsions (particularly just prior to death)
- defecation, diarrhea
- dyspnea (difficult breathing)
- epistaxis (bleeding from the nares)
- exophthalmia (protruding eyes)
- hyperexcitability
- lacrimation
- miosis (contraction of pupils)
- myasthenia (muscular weakness)
- mydriasis (dilation of pupils)
- opisthotonos (heads and limbs arched back)
- paresis (slight paralysis)
- piloerection (erection of contour feathers)
- polydipsia (excessive thirst)
- ptosis (drooping of eyelids)
- slurred vocalizations
- tachypnea (rapid breathing)
- tenesmus (spasmodic contraction of anal sphincter)
- tremors
- vomiting

As emphasized by Franson and Smith (1999), the presence of recently ingested food material in the upper gastrointestinal tract is suggestive of acute poisoning by a cholinesterase inhibitor. Certainly, identifying the gastrointestinal content may provide the best indication that the bird has been poisoned by an agricultural chemical (e.g., the presence of undigested grasshoppers or other insect pests, treated seed, granules of pesticides, etc.). A number of diseases produce signs that could be mistaken for poisoning. These have been thoroughly reviewed in Friend and Franson (1999).

Usual cause of death

Typically, but not always, poisoned birds die of anoxia because of respiratory failure. This is the result of one or a combination of factors: excessive secretion in the respiratory tract, bronchoconstriction, failure of the muscles required for respiration, and/or failure of the respiration center (see Gallo and Lawryk 1991 for a review).

Delayed mortality: Secondary causes

Because of the far-reaching importance of cholinergic tracts, poisoned individuals may be seriously compromised even if they survive the initial exposure—hence the importance of lengthy follow-up of animals in rehabilitation. Some of the most important delayed effects may be as follows (see Grue et al. 1991, 1997; Mineau 1991; and Parsons et al. 2001 for a more complete coverage of "sublethal" effects):

Trauma or other mishaps in the course of intoxication. It has been shown that birds sublethally exposed to cholinesterase inhibitors are more susceptible to predation (Galindo et al. 1985 Buerger et al. 1991, Hunt et al. 1992). There is also reason to believe that sublethal exposure to cholinesterase inhibitors makes animals more vulnerable to collision with objects, both moving (e.g., vehicles) or stationary (e.g., powerlines, fences, buildings). Upon arriving at rehabilitation centers, raptors, for example, are often diagnosed as victims of collision. The evidence of pesticide involvement is two-fold: (1) Anecdotal evidence from rehabilitation centers where cholinesterase measurements have been made on a routine basis (e.g., Porter 1993); and (2) The wealth of human evidence about the various visual and motor effects that affect the safety of workers (see Gallo and Lawryk 1991 for review) following exposure to OPs and CBs. Blurred vision is a common complaint; unequal miosis also can lead to a phenomenon called the Pulfrich Stereo Effect where depth perception and the ability to compute trajectories are affected. Any of these effects in a flying bird would be expected to lead to higher rates of collision.

Poor condition resulting from a reduced ability to feed, as well as a disruption in normal circadian patterns and thermoregulation. Energy deficits are thought to be especially important in explaining mass mortality of exposed wildlife under inclement conditions or when body reserves are low, such as on migration, especially in the case of small birds that cannot endure long fasts because of their high metabolic demands.

Muscular necrosis as a result of transient anoxia. In humans, this results in what has been termed "type II toxicity" or "intermediate syndrome," which typically results in cardiac failure several days after return to normal cholinesterase status. The authors are not aware of any descriptions of this syndrome in birds.

Delayed neurotoxicity. This syndrome is the irreversible dying back of neurons as a result of an enzyme inhibition effect other than the usual anticholinesterase effect. Only a few OPs are reputed to cause this. It is noteworthy that the chicken is the usual test organism for this syndrome, but effects are often seen at dosing levels that would be lethal were the animal not antidoted for cholinesterase inhibition effects, as such lab chickens commonly are. This syndrome has not been reported in wild birds, although we might expect to see it first in animals subject to intensive rehabilitation efforts. Woods and Plumlee (1999) briefly mention possible cases in cage birds from California but provide no further detail.

Treatment of Poisoning Cases

Treatment of wildlife generally follows a four-pronged approach. Atropine is considered to be the most effective antidote for both OP and CB intoxication. By effectively competing with acetylcholine for the muscarinic cellular recep-

tors, it prevents overstimulation of the autonomic parasympathetic system. Most importantly, it helps prevent asphyxia, the main cause of death. In human subjects, it is customary to infuse atropine constantly in order to maintain optimal concentration throughout recovery from the "cholinergic crisis." In wildlife rehabilitation this is impractical, and subjects need to be repeatedly injected as indicated by the reappearance of clinical signs.

The second prong consists of the administration of chemicals that hasten the release of bound OP from the acetylcholinesterase enzyme. This strategy is effective only where intoxication results from an OP pesticide and is recent. (Porter 1993 gives 24 hours as a guideline, but this will vary from pesticide to pesticide.) The most frequently used chemical for this purpose is 2-pralidoxime chloride (2-PAM).

The third prong in the approach is the provision of supportive symptomatic care, especially positive ventilation in case of respiratory arrest. Other supportive measures include a quiet and warm environment, rehydration, prophylactic use of fungicides to prevent aspergillosis, and the use of antiseizure medication such as diazepam.

Finally, it is important to eliminate the source of the exposure. Gastric lavage may be performed where there is evidence of a large food bolus, which may continue to release pesticide over time. Some have found surgical evacuation of the crop to be more effective and less stressful than forced regurgitation, such as in the case of bald eagles having scavenged contaminated waterfowl (K. Langelier, pers. comm.). Others prefer to remove crop contents in such larger species with forceps when circumstances allow. Frazier (2000) advocates the use of activated charcoal. Pesticides absorbed into the bird's subepidermal tissues can be slowly released over time and result in prolonged re-exposure (Henderson et al. 1994). Where dermal exposure is suspected, a vigorous rinsing of the feet with warm soapy water may help limit pesticide entry. Alcohol is used routinely to collect foot rinses from birds thought to have been exposed dermally, and these can be kept frozen for chemical analysis where feasible and warranted. However, there is a concern that applying alcohol to the feet may enhance pesticide penetration.

The exact dosages of atropine and 2-PAM to be administered are currently a matter of debate. The "traditional" approach is based on levels found to be effective in humans. For example, Porter (1993) recommends an injection of 0.5 mg/kg atropine IM (or one-fourth of the total dose given IV) repeated after 15 min if no decline in signs is observed. According to the same source, the recommended dosage of 2-PAM is 20 mg/kg IM. Frazier (2000) recommends 0.1–0.2 mg/kg atropine every 3–4 hours and suggests that 10–20 mg/kg 2-PAM be given concurrently. A far more "aggressive" approach was recently recommended by Shlosberg and colleagues (1997). Their experiments with chickens led them to recommend 25 mg/kg atropine and 50 mg/kg 2-PAM as the best treatment for an unknown cholinesterase inhibitor. These dosages were established empirically; the highest doses not causing obvious toxicity in normal chickens were retained. According to Schlosberg et al., the ease with which each species breaks down atropine varies. Future research should focus on establishing maximum tolerated dosages for atropine or, better still, an atropine + 2-PAM cocktail for those species commonly treated at rehabilitation centers. The best and final judge as to the dose needed, however, is the person performing the treatment. It is much better to "titrate" the clinical signs rather than use a fixed-dose approach.

Other possibilities for treatment exist, but they have not been systematically investigated in wildlife. Injections of glucose and of vitamin C have afforded some protection to small mammals experimentally dosed with various OPs (see Gallo and Lawryk 1991 for review). The ready availability of both make them obvious candidates for further experimentation. Frazier (2000) also notes that diphenhydramine has been used in mammalian intoxications to counter muscle fasciculations and other nicotinic symptoms not resolved by atropine.

When the time for release of recovered birds approaches, rehabilitators might consider the potential for re-exposure on the capture site. When initial capture sites are known to be contaminated or are regularly associated with suspected poisonings, it may be prudent to identify a separate release site. As a check for an individual bird's readiness for release, subjecting the animal to increased stress—for example, by "flushing" them from perch to perch in a large enclosed flight cage, thus forcing sustained, involuntary activity—may allow observation of possible lapse into cholinergic crisis. Many birds that appear fully recovered may again exhibit clinical signs of poisoning when exposed to brief periods of sustained stress, suggesting their normal activity in the wild would be compromised (M. Hooper and the authors, pers. obs.).

Measurement of Cholinesterase Levels

The best diagnosis of intoxication with a ChE inhibitor is the measurement of enzyme levels. The level of acetylcholinesterase in the brain is best correlated with morbidity and mortality, and is often used to diagnose mortality. A rule of thumb is that a 20% decrease of brain acetylcholinesterase is indicative of exposure. Lethal intoxication is usually associated with a 50% decline, although this figure can be lower or higher depending on the situation (see Grue et al. 1991 for a review). If a brain is to be subsampled (a good idea because it allows for repeat testing should anything go wrong and keeps some tissue for other possible tests, such as the determination of organochlorine residues), it is essential to do a sagittal section and test half of the brain. This is because different portions of the brain register a different cholinesterase activity. Where a live bird is to be tested, the test is carried out on a blood sample. In mammals, red blood cells also carry acetylcholinesterase. In birds, all the activity is associated with the plasma. In addition, all vertebrates have a related enzyme in plasma: butyrylcholinesterase. The function of this enzyme is a matter of debate; most likely it offers a first line of defense against cholinesterase-inhibiting substances of which there are a few in nature. This is important because the tests routinely used to monitor acetylcholinesterase levels are, in fact, unspecific and also measure the butyrylcholinesterase titer. Adding a little more complexity is the fact that the ratio of acetylcholinesterase:butyrylcholinesterase in avian plasma is extremely variable among different bird species. Inhibition of blood cholinesterases *per se* may not compromise an individual; however, it is indicative of the presence of a systemic cholinest-

erase inhibitor. In mammals, whole blood is usually tested because red blood cells have a high level of AChE activity. In birds, typically the blood is centrifuged and the plasma only is retained for analysis. Both brain and plasma samples can be frozen for future analysis (a minimum temperature of -30°C is recommended). Repeated freezing and thawing (as can occur in domestic self-defrosting freezers) must be avoided. Recently, the Canadian Wildlife Service Biomarker Laboratory has demonstrated the feasibility of collecting blood as dry spots on filter papers. This avoids the need to freeze the samples and allows them to be sent through the postal system. (Trudeau et al. 1995, Sans Cartier and Trudeau 2000). The laboratory analysis is modified to avoid interference problems with whole blood, although recent work shows that this may not be necessary.

A cholinesterase assay is economical and relatively easy to carry out. It provides useful information in many cases but is not infallible, and the results need to be interpreted carefully. It is important for wildlife rehabilitators to have a basic understanding of the assay. Even if they do not carry it out themselves, this understanding will allow them to ask the right questions and/or correctly interpret test results. For more detailed information on the cholinesterase assay itself, refer to Fairbrother et al. 1991 and Sans Cartier and Trudeau 2000. It is advisable to submit samples to the same laboratory over time and insist that the assay be rigorously standardized by the laboratory in question. Marden et al. (1994) showed how small interlaboratory differences in procedure could give rise to very different results. Repeat testing of the same species by the same laboratory will more easily paint a picture of what normal and abnormal activity values should look like.

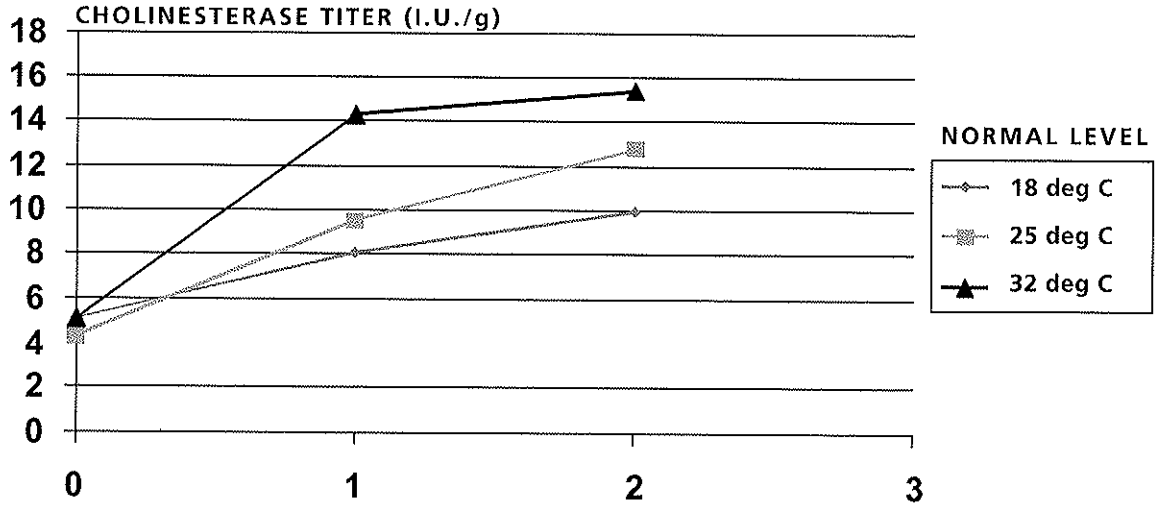
It is clear that CB poisonings are often harder to diagnose than OP poisonings. This is obviously a concern, given the overwhelming importance of some CB insecticides, such as carbofuran, in the avian kill record (e.g., Mineau et al. 1999). The difficulty of diagnosis results from the strength of the pesticide-acetylcholinesterase bond discussed earlier. Figure 1, derived from the data of Hill (1989), shows the effect of temperature on the spontaneous reactivation of brain tissue (hydrolysis of the AChE-pesticide bond as explained earlier) from quail lethally dosed with the CB carbofuran.

This could be a serious problem when carcasses are not immediately sampled. For purposes of a pathological examination, carcasses must not be frozen. Yet, waiting for the necropsy to be completed before taking brain samples for measuring acetylcholinesterase levels means that evidence of a carbamate exposure may be lost. When several specimens are available, some (or at least their heads) should be frozen as soon as possible without waiting for necropsy results. The reader is referred to Hunt and Hooper (1993) and Padilla and Hooper (1992) for a detailed discussion of cholinesterase measurements following a CB intoxication.

The capacity for CB-inhibited tissue samples to spontaneously recover cholinesterase activity differentiates between OP and CB intoxication (e.g., see Smith et al. 1995). In an analogous fashion, 2-PAM can be used to attempt reactivation of a sample and show involvement by an OP. Because the 2-PAM is added to samples in an aqueous solution, this may also foster recovery of a sample inhibited by a CB. However, a measurable recovery in enzyme activity with 2-PAM and concomitant absence of a recovery with water alone is diagnostic proof that an OP was present in the sample.

Another problem complicating diagnoses is that certain pesticides do not cross easily into the brain, especially when death is rapid, as is often the case with carbamates. Some OPs also exhibit this same characteristic (e.g., terbufos in Hooper et al. 1990), although in some intoxicated birds, brain AChE

FIGURE 1. POSTMORTEM REACTIVATION OF BRAIN TISSUE FROM JAPANESE QUAIL *(COTURNIX JAPONICA)* DOSED WITH THE CARBAMATE INSECTICIDE CARBOFURAN AND EUTHANIZED 1 HR POST DOSE (after the data of Hill 1989)



depression can be extreme with those compounds as well (see carbofuran and terbufos data below).

Figure 2 shows brain cholinesterase analyses performed on five bald eagles from five different poisoning incidents investigated by the Canadian Wildlife Service. These were chosen to illustrate some aspects of cholinesterase analyses.

The first eagle was a carbofuran victim and shows that spontaneous reactivation of the sample is not always successful following intoxication by a carbamate. In this case, the failure of the sample to reactivate was due to the presence of leftover unbound insecticide. When the sample was filtered in a special chromatographic column (which strips small molecules such as insecticides but allows large ones such as the cholinesterase enzymes to pass through—as described in Hunt and Hooper 1993)—the sample's cholinesterase activity was easily reactivated, showing that a carbamate was indeed present. The second sample—also carbofuran intoxication—was already reactivated or never showed any inhibition. The cholinesterase measurement was of no diagnostic help here. The third eagle was killed by the OP terbufos. The initial analysis showed a marked depression relative to controls. However, the sample could not be reactivated with the 2-PAM. The ability of 2-PAM to free the acetylcholinesterase molecule declines markedly over time. The fourth eagle shows the result of a positive reactivation following phorate poisoning (another OP). However, the last bird, also killed by phorate, failed the 2-PAM reactivation test. Why the difference? Perhaps it is explained by the presence of the insecticide (in which case a column should have been used to clean the sample) or a longer period elapsed since intoxication in the latter bird.

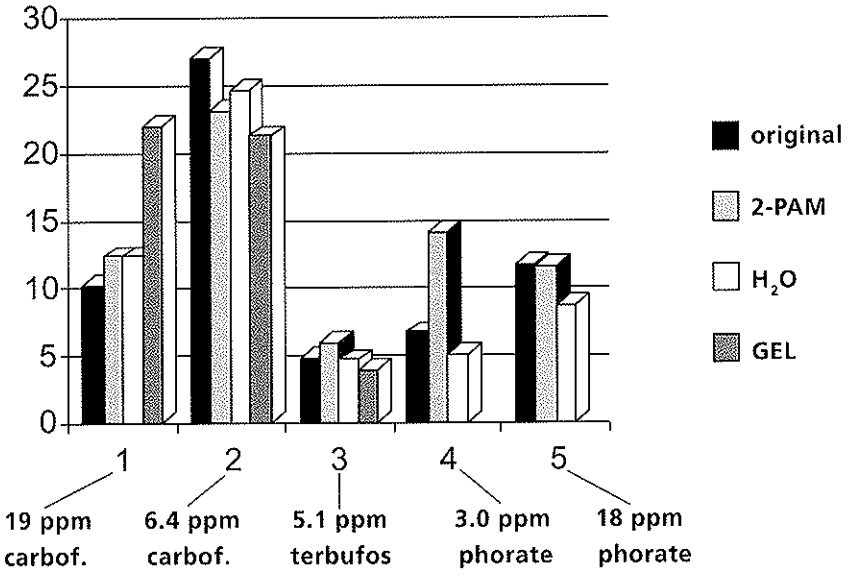
As noted earlier, cholinesterase activity in avian blood is entirely in the plasma fraction and results from a mixture of acetyl- and butyrylcholinesterase, the proportion of which varies between species. Most diagnostic centers associated with hospitals or veterinary clinics are well equipped and aware of the procedures associated with mammalian blood, but they generally are not familiar with analyzing bird samples. Avian plasma can be analyzed by the same techniques commonly used for mammalian whole blood samples. Blood cholinesterase levels are more variable than those of the brain and tend to indicate exposure rather than a life-threatening intoxication. For a live animal under care, repeated measurement of blood cholinesterase offers the most reliable indication of exposure to a cholinesterase inhibitor when serial samples show a recovery to normal levels over time (e.g., see Elliott et al. 1997 for an example of eagles poisoned by phorate).

In the end, while cholinesterase measurements are useful, they are not infallible and must be interpreted carefully, considering all the ancillary evidence and chemical analyses when available.

Beyond Cholinesterase: The Residue Angle

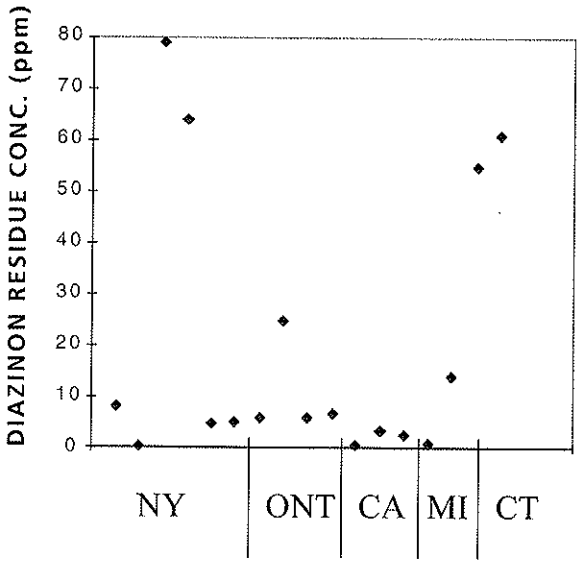
When there is evidence of cholinesterase depression, or even where there is no clear evidence but where circumstances are highly suspect, one should proceed with the chemical analysis of the gastrointestinal tract contents. This is usually—but not always—where the highest pesticide residues are found. Before gut contents are sent off for analysis, it is extremely important that they be well described. Often, gut contents offer the best clue to explain a pesticide kill. In some cases, it is

FIGURE 2. BRAIN AChE ANALYSES FOR FIVE BALD EAGLES (*HALIAEETUS LEUCOCEPHALUS*) RECEIVED AT BRITISH COLUMBIA REHABILITATION CENTERS MATCHED WITH RESIDUE ANALYSIS OF GASTROINTESTINAL TRACT CONTENT



Data from cases reviewed by Mineau et al. 1999; analyses provided by S. Trudeau, Canadian Wildlife Service Biomarker Laboratory. See text for details of sample treatment.

FIGURE 3. DIAZINON RESIDUES IN GRASS SAMPLES RETRIEVED FROM THE UPPER GASTROINTESTINAL TRACTS OF POISONED DUCKS OR GEESE



Data from Stone and Gradoni 1985, Stone 1987, and Frank et al. 1991. Reported cases are from New York State (NY), Ontario, Canada (ONT), California (CA), Michigan (MI), and Connecticut (CT).

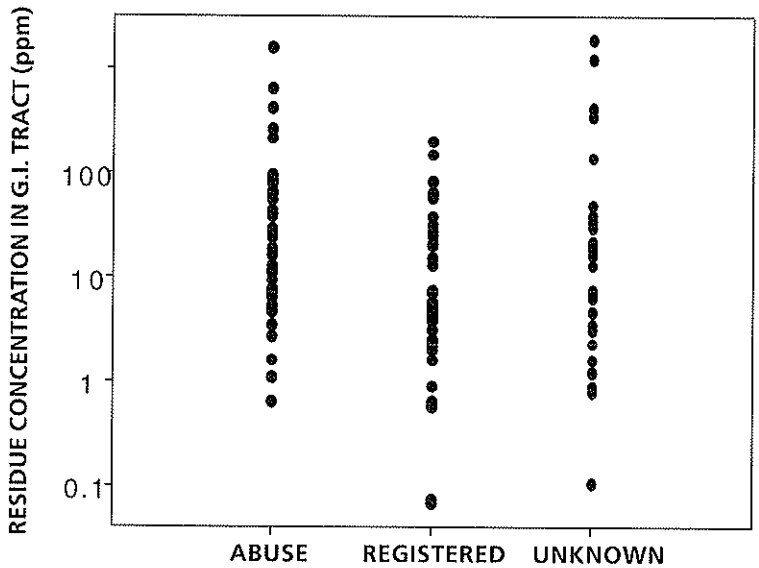
recommended to also rinse the feet and feathers of birds suspected of having been dermally exposed (e.g., Hooper et al. 1989), although rehabilitators are reminded that this may enhance pesticide absorption in a live bird. Residue analysis is a costly proposition, so sending a sample to be analyzed cannot be done lightly. It has been customary in several diagnostic centers to run a cholinesterase assay first and then a residue analysis if the former is positive. As the pesticide arsenal moves away from cholinesterase inhibitors to embrace new chemistry, this strategy will cease to be sufficient, although it will still be useful to direct the residue analysis. Chemical analyses are most expensive and least likely to succeed when the chemist has no idea of the nature of the pesticide being sought.

The level of a pesticide measured in a carcass is extremely variable. For example, Figure 3 shows levels of diazinon residues recovered in grass samples taken from the upper gastrointestinal tracts of poisoned ducks or geese.

Occasionally, criminal abuse cases are characterized by very high residue levels in gut contents because concentrated baits are used. In practice, however, the extensive overlap in residue levels between abuse cases and poisonings resulting from labeled pesticide use means that residue levels cannot reliably be used to separate those two situations. Figure 4 shows the extensive overlap in the concentration of carbofuran in the gut contents of birds of prey killed through abuse, through labeled pesticide use, or under unknown circumstances.

For reasons outlined earlier, the quantity of pesticide found in poisoned wildlife is often poorly correlated with measured levels of cholinesterase depression. Therefore, when the presence of a cholinesterase inhibitor is confirmed through a cholinesterase assay, any finding of a cholinesterase-inhibiting

FIGURE 4. CARBOFURAN CONTENT OF GASTROINTESTINAL TRACTS OF POISONED BIRDS OF PREY SEPARATED BETWEEN KNOWN ABUSE CASES, NORMAL USES, OR WHERE CIRCUMSTANCES WERE UNKNOWN.



Data from U.S., Canada, and U.K. cases reviewed by Mineau et al. 1999.

pesticide, regardless of its concentration, should be considered significant. Also, failure to find a pesticide agent through chemical analysis does not disprove that intoxication occurred.

Anticoagulants

Wildlife rehabilitators should be aware of at least one other class of pesticides: the anticoagulants. This is because the frequency of poisonings has increased dramatically in recent years, coinciding with the increased use of a new generation of single-dose products that are much more toxic and much more likely to lead to secondary poisoning in nontarget species. Anticoagulants in this group include difenacoum, brodifacoum, bromadialone, flocoumafen, and difethialone. Birds admitted to rehabilitation centers and diagnosed as nonpesticide cases often carry anticoagulant residues (Canadian Wildlife Service, unpublished), but the significance of these residues is not known. The second generation anticoagulants have a high affinity for the liver, where they bind to specific receptors. Once bound, residues can be extremely persistent—e.g., several years for brodifacoum, at least in rat livers (http://www.epa.gov/REDs/2100red.pdf). A pressing question is whether the presence of residues makes an animal more sensitive to this class of chemicals if re-exposed. In two recent surveys of cases in New York and California (Stone et al. 1999 and Hosea 2000, respectively) it has been established that birds of prey in proximity to urban centers have a high risk of exposure. This is in keeping with regulations and labeling that restrict use of these products to Norway and black (roof) rats or house mice in and around habitations or in sewers.

As with any anticoagulant intoxication, vitamin K is antidotal. Frazier (2000) recommends 2.5–5.0 mg/kg intramuscularly or orally every 24 hours (or 0.2–2.2 mg/kg every 4–8 hours), to be continued for at least 10–14 days in the case of warfarin or at least for 1 month in the case of the newer, single-dose products.

New pesticides

A few recent introductions are worth mentioning. Possibly the most interesting from the point of view of birds is chlorfenapyr, the first of a new class of pyrrole insecticides. In response to advocacy by the American Bird Conservancy and subsequent scientific criticism, this insecticide was the first pesticide to be denied a registration in the U.S. based solely on its toxicity to birds. It has very high acute toxicity (on par with some of the more toxic OPs and CBs), although mortality is typically delayed for a few days after exposure. It also causes notable effects on reproduction at extremely low levels of exposure (http://www.epa.gov/opprd001/chlorfenapyr/toc.htm). The high persistence and high aquatic toxicity of chlorfenapyr were undoubtedly factors that contributed to registration denial for cotton applications in the United States. Concerns continue because chlorfenapyr is registered in a number of countries, including areas comprising the wintering ranges of many North American bird species. New registration proposals are also being considered in the U.S.

Kills of pigeons and game birds have been reported in France with two other new insecticides used as seed dressings: imidacloprid and fipronil (Annual reports from l'Office National de la Chasse). It is not clear how extensive and/or important these problems will be, and whether kills may also result from other formulations of these same products.

A number of other insecticides from totally new chemical classes are gaining in popularity in the U.S. and elsewhere. On the positive side, some of these products may represent improvements over the OP and CB pesticides they are replacing. For example, some require a lesser amount of a more selective, or target-specific substance that can be applied with equal efficacy. On the negative side, we are ill prepared to ascertain their presence in wildlife tissue and there are no biochemical diagnostic techniques to aid in the investigation of a mortality incident. In the authors' opinion, development of the latter should be a requirement of any new pesticide registration; unfortunately, this is not currently the case.

Note

Part II of this paper will appear in the next issue of the *Journal of Wildlife Rehabilitation*.

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Meeting the Protein Requirements Of Adult Hummingbirds in Captivity

by Elizabeth Penn Elliston and Janine Perlman

Introduction

Hummingbirds can live for relatively long periods on a diet of sugar water and insects, which are the basic elements of their natural diet. Sugar water provides calories; insects supply protein and other essential nutrients. However, many hummingbirds in rehabilitation have partially or completely lost the ability to fly, and hence are unable to capture insects.

One of the most challenging aspects of hummingbird rehabilitation is supplying captive birds with the nutrition they require. In part, this is because their requirements are not well characterized. Gartrell (2000) concludes, in a review article, that "the specific nutritional requirements of nectarivores for maintenance, growth, and reproduction are not known."

When presented with an unlimited supply, hummingbirds catch and consume about 600–700 insects per day (Scheithauer 1967, p. 132; authors' obs.; pers. comm. with other observers). This number of fruit flies supplies 100 mg/day of high-quality protein (calculated from: G. Maroni, pers. comm.; Barker et al. 1998). On the other hand, if the number of insects available to hummingbirds preparing for release is inadequate, the birds fail to thrive (Elliston, pers. obs.). Brice (1992), in her study of the need for nectar and arthropods in the diet of captive Anna's hummingbirds (*Calypte anna*), found that two of her three subject birds lost weight after 10 days on an insect-free diet. Optimal foraging models (described in Ricklefs and Miller 2000) suggest that the birds would not expend the energy necessary to catch insects unless they required the nutrients provided by them.

Unless a bird in captivity is fully flighted and has free access to unlimited insects (and is actually consuming 100 mg/day of insect protein), it seems prudent to ensure that it receives that much protein from its nectar. Absolute protein requirements are independent of caloric requirements (Klasing 1998). In summarizing his work and that of others, Klasing says:

> ...[D]ietary protein requirements decrease with increasing activity level. A bird in the wild requires a lower % dietary protein than it does in captivity, because high energy needs associated with foraging and thermoregulation drive consumption of greater amounts of food. Although birds in captivity must consume foods with relatively high levels of protein, their daily protein requirement on a mg day^{-1} basis has not been shown to differ between captive and wild environments (p.142).

Therefore, whether a hummingbird is very active or inactive, its need for protein is relatively constant.

There appear to be two major challenges in formulating diets used for captive hummingbirds. One is that the protein must be soluble. That is, it must be dissolved in the sugar-water base of the nectar so that it is ingested, rather than precipitating out of solution, where it is unavailable to the bird. Most proteins are insoluble. In order to make them soluble, they must be chemically broken into pieces, or hydrolyzed. Foods containing hydrolyzed, soluble proteins are uncommon and expensive.

The second factor, which often goes unrecognized by caregivers, is that the diet must be acceptable to the bird in order to be ingested. Hummingbirds routinely starve when offered food that tastes bad to them. In addition to rejecting spoiled food, hummingbirds may display individual food preferences in captivity, seemingly dependent on age and on the history of their taste experiences. For example, subadult birds seem more accepting of a variety of diets than adults. Occasionally, birds that come into captivity as adults reject all types of complete soluble captive diets

ABSTRACT: Morbidity and mortality in captive hummingbirds are frequently a result of poor diet. It is not commonly understood that, for disabled birds, required nutrients must be dissolved in a sugar-water solution, or "nectar." If the nutrients are particulate, they cannot be ingested. Additionally, if the food is not palatable it will not be consumed.

The protein requirements of hummingbirds are traditionally believed to be very low. However, this paper considers evidence that the amount of protein hummingbirds routinely obtain in nature, and presumably their requirement for optimal functioning, is approximately three times the "minimum requirement."

The most successful captive diets in common use were analyzed for their available protein and found to be deficient compared to the "natural" amounts of protein eaten by hummingbirds. A source of high-quality, soluble protein is suggested, to increase captive dietary protein levels to approximate those of the wild diet.

KEY WORDS: nectar, protein requirements, soluble protein, hummingbirds, Vital HN®, tryptone, nutritional requirements, diet

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offered. Whether this is due to a rejection of artificial flavor or some other characteristic of the diet is unknown.

The hummingbirds of most concern here are those that are unable to hunt insects because their flight is impaired. In such cases, all of their protein must come from their nectar until they have healed and regained the ability to fly. Even if they are unflighted for a relatively short time, birds, like other animals (Ludwig 2001), require extra protein while healing. Thus the diets of unflighted birds must be paid particular attention.

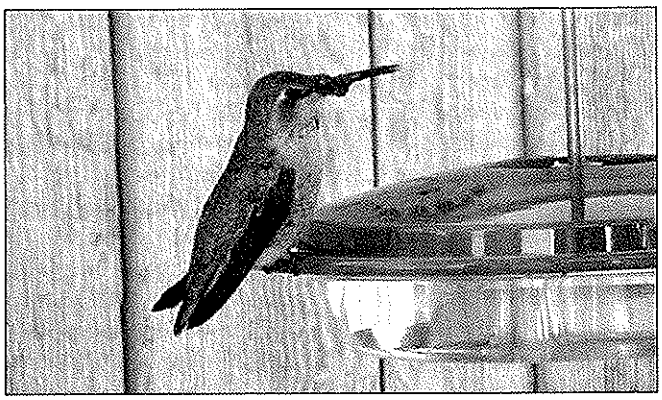
The authors were unable to find published studies of either the relationship of commonly used captive diets to the actual requirements of hummingbirds, or studies comparing diets.

It can be reasonably assumed that wild hummingbirds optimize their capture and intake of insects, as the birds in Scheithauer's (1967) experiments presumably did. His count of 677 fruit flies (average of 6 days observations) eaten daily by adult hummingbirds contains about 100 mg of high-quality protein.

Nestling hummingbirds are fed, by their mother, diets that are very high in insects (e.g., Sargent 1999). In 1992 author E. P. E. received two sibling nestlings that were younger than 1 week old. One bird had died from dehydration, the other was still alive. Both birds' crops were packed with insects. The crop contents of the dead bird weighed 200 mg and contained 20 large green leaf hoppers, ~40 gnat-sized insects, two tiny flies with striped abdomens, and one tiny rock. The large intestine was full of insect waste, and the bird had visible fat deposits. (The other bird fledged 17 days later and was eventually released.) On another occasion more than 60 midges and two tiny green leaf hoppers were counted from the crop of a dead nestling. The number of calories consumed by a particular bird in the wild depends on season, ambient temperature, size of bird, feather condition, and probably a number of other factors. Baltosser and Russell (2000) report the average field metabolic rate of four black-chinned hummingbirds (*Archilochus alexandri*) as 29.1±6.3 kJ/d (range 23.4–37.9, n=4). This translates into 7.0 Kcal/24 hours (1 Kcal=4.18 kJ). Nagy (1987) and Powers and Conley (1994) conducted studies in the Chiricahua Mountains of Arizona, where post-breeding-season black-chinned hummingbirds were found to use an average of 6.9 Kcal/day. This was a value 17% higher than that predicted using allometry (Baltosser and Russell 2000). Tyrrell and Tyrrell (1986, p. 138) describe a study of a flighted male Anna's hummingbird in which the caloric expenditure for various activities over a day were measured in detail, giving a total expenditure of 6.6 Kcal. In addition, Elliston and Perlman have separately measured food intake of known caloric concentration in captive birds (Elliston and Gillett 1994; unpublished data). Measurements were made to 0.2 cc in a 3-cc syringe feeding tube. There was insignificant evaporation and wastage. All measurements are consistent with 6 Kcal as a reasonable estimate of 24 hours' intake (and requirement) for a relatively inactive captive hummingbird at "room temperature." If such a bird cannot capture insects, it must obtain all needed nutrients, including 100 mg protein/day, in those 6 Kcal of nectar.

Methods

The authors chose to examine only diets in widespread use that appear to give consistently good results (i.e., they have been popularly used to maintain adult hummingbirds in captivity over a period of years). Diets that are not in extensive use or that have not been generally acceptable to those who use them were not included in this analysis.



SANDRA BROWN

Two diets currently appear to meet both criteria. One is Nektar Plus® (Nekton); the other is the diet formulated by Elliston and Gillett (1994) that includes Vital HN®. To the authors' knowledge, most flighted birds maintained long term on these two diets also have some insect supplementation. Dietary analyses were conducted using data supplied by the manufacturers, including personal communications with company nutritionists.

Energetics reports from field studies were used with theoretical and allometric values, as well as previously unpublished reports from hummingbird rehabilitators, to determine reasonable estimates of the birds' nutritional needs.

Dissected gizzards were bathed in isotonic saline and laid on their sides, approximating the way they lie in the body. The external dimensions of the gizzards, with contents, were measured from the anterior end of the beginning of the small intestine (pylorus) diagonally across the widest expanse of the organ. Another measurement was taken perpendicular to the first; the thickness of the organ at its widest point was also taken. The instrument used for these measurements was a Mituyoyo Corporation digimatic caliper, 0.01–150 mm.

Multiplying these measurements results in the volume of a rectilinear prism of approximately the right size. Dividing that number by 0.52 approximates the volume of the actual pancake-shaped organ.

Results and Discussion

Although some reports state that hummingbirds and other nectarivores have low protein requirements, it is critical to consider the way in which those requirements have been measured.

Generally, the minimum protein requirement is considered to be the dietary level at which nitrogen (protein) intake equals nitrogen loss. Brice and Grau (1991) determined that level to be about 30 mg/day of protein for Costa's hummingbirds (*Calypte costae*). However, van Tets and Nicolson (2000) note in their own and in other studies, such as those of Brice and Grau, that nectarivores continue to retain nitrogen at dietary protein levels far above those considered "minimally required," and that this phenomenon has not been addressed. It appears that nectarivores can be very thrifty with nitrogen when it is present in low levels in the diet, but that for optimum func-

tioning, they need—and retain—more. It seems, then, that the "empirically derived" values for protein requirements are, in fact, much lower than those actually needed for normal or optimal physiological function (I. van Tets, pers. comm.).

Numerous field observations have shown that adult hummingbirds can subsist on diets that are mostly insects. For example, ruby-throated hummingbirds (*Archilochus colubris*) and rufous hummingbirds (*Selasphorus rufus*) that overwinter in areas outside of their normal winter range where there are few or no flowers available rely mostly or completely on small insects to survive (R. Sargent, pers. comm.). Blue-throated hummingbirds (*Lampornis clemenciae*) and black-chinned hummingbirds have both been observed to eat solely insects when there was no nectar available (Baltosser and Russell 2000; Tyrrell and Tyrrell 1986, p. 135; Sargent 2000).

The original search by Elliston and Gillett (1994) for a completely defined and palatable food for captive hummingbirds was stimulated by the frequent mysterious mortality of healthy juvenile birds. The diets in use at the time were a complex combination of pureed insects and nectar. Warm summer weather produced fermentation and spoilage evidenced by a dripping syringe feeder. The birds, it turned out, refused to drink fermenting food.

The commercially available hummingbird diet (Roudybush®) marketed for raising orphaned hummingbird nestlings comes in three protein concentrations for various ages (0–18 days; 3–7 weeks; >7 weeks). The protein of this product is isolated soy protein powder. Not only is this material of plant origin, lacking the essential amino acid profile of an insect diet, but soy protein is insoluble. High morbidity and mortality in birds fed this product have led one of the original formulators (Heinemann 2000) to withdraw recommendation for its use.

In 1986 Elliston was alerted to the dangers of particulate solids in the diet when several formerly vigorous nestlings over the age of 2 weeks, that were fed Roudybush®, suddenly showed signs of slowed emptying of the crop, after which they died (Elliston 1999). On postmortem, the gizzards of these birds were found to be full of powder and enlarged over the size of normal hummingbird gizzards, as recorded for birds euthanized as a result of trauma.

TABLE 1. EXTERNAL DIMENSIONS OF GIZZARDS (NOT EMPTIED OF THEIR CONTENTS) OF BIRDS FED SOY-BASED FOOD VS. WILD DIET

Group	Volume	n
Soy-fed gizzards	28.8 mm^3	11
"Wild" gizzards	10.2 mm^3	18

A t test for independent groups was applied to these data; t = 7.66 (df = 27, p < 0.001).

On examination, one deceased bird in particularly acute condition proved to have a swollen, red pylorus. Attempts at perfusing some of these gizzards from the intestine through the pylorus (backward) demonstrated that they were very firmly packed, such that no fluids could normally pass into the lower digestive tract. Birds with more chronic conditions contained small and large intestinal walls that were thin and friable. The lower gut in controls (birds euthanized due to trauma) were thick walled and well vascularized.

Two birds fed a soy protein diet from naked nestling stage with poor feathering were kept for a year on the juvenile/adult Vital HN® diet (Elliston and Gillett 1994) in hopes that they would be able to molt into adequate feathers. They were unable to do so and were euthanized. The gizzards of these birds measured 10.2 mm^3 and 7.0 mm^3, well within the range of the gizzards of normal "wild" birds.

During this period, Wildlife Rescue, Inc. of New Mexico (with which author E. P. E. is associated) received a vigorous nestling about 4–5 days old. It was fed the Roudybush® diet for 1 week, after which gaping became less frequent and crop clearance began to slow. Fortunately, it was at this time that some older birds were experiencing acute problems, so the rehabilitators (including E. P. E.) were alerted to closely examine the protocol. The bird's feces were checked and occult blood (identified by guaiac) was present. Prozyme™ and steroids (dexamethasone at 125 mg/kg) were added to a completely soluble diet, and in 10 days the bird had regained its former crop capacity, blood was gone from the stool, and the bird fledged. This was one of the experiences that led to the author's adoption of Vital HN® as the protein source for hummingbird food. Nestlings are fed by gavage and, unlike adult self-feeding birds, are unable to filter out, or reject, unnatural particulates (see also Powers and Perlman 2001).

As discussed above, the natural diet of hummingbird nestlings is highly insoluble: it is insects. Hummingbirds possess the capability, both physical and enzymatic, to digest insects. The residuum of chitinous plates of tiny insects passes through the digestive system, resulting in surprisingly bulky droppings, as can be confirmed by examination of the area around any wild hummingbird nest. The specific characteristics of solids ingested by hummingbirds determine their digestibility.

There are very few commonly available sources of soluble protein. One source is milk. Scheithauer (1967, p. 125) describes milk as an ingredient in 8 of 10 zoo diets for which he was able to obtain recipes. All of those recipes also contained honey, a practice that we now know to be inadvisable due to the presence of viable spores. Both condensed milk and powdered milk were included in these diets, and Scheithauer observed that powdered milk caused the birds he tested to have digestive problems in the form of constipation. Milk contains lactose. Lactase, the enzyme required to digest lactose, is not endogenous to avian digestive systems (although bacteria which do possess limited lactase are often present in the gut of a wide array of birds; see, e.g., Shetty et al. 1990 and Lombardo and Thorpe 2000). On a weight basis, milk contains more lactose than protein. So, it appears, milk is an unsuitable source of protein for hummingbird food.

Egg also contains soluble proteins, if they are not denatured by heat. Uncooked egg white, however, contains avidin, a glycoprotein that binds the vitamin biotin and makes it unavailable to the bird (Groff and Gropper 2000, p. 284).

Egg yolk contains 33% protein and 60% fat. Experience has shown that excess fat in hummingbird diets, either from the use of egg yolk or crushed mealworms, has led to lethargy, abnormal droppings, and sickly behavior (Scheithauer 1967, p. 146; authors' pers. obs.).

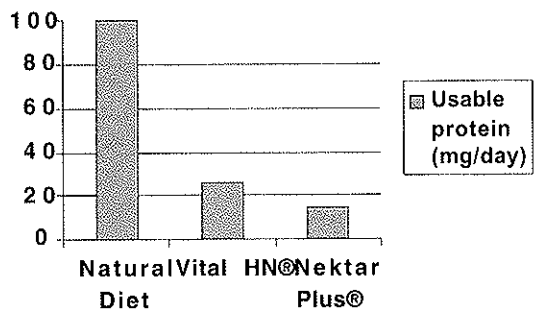
A third liquid containing soluble proteins is blood. Whole blood, however, is far too rich in iron to be a desirable ingredient, and although blood serum (bovine) was also an ingredient in some of the recipes Scheithauer examined (p. 134), it is often not easy to obtain.

Nektar Plus® contains both soy protein (pers. comm. with Nekton nutritionist) and free amino acids. The soy protein is insoluble. Hummingbirds' tongues collect nectar by capillary action rather than by suction (Sargent 1999), and they typically capture insects by flying at them and catching them with open bill. They have no means to "slurp up" solids sitting in the bottom of a container. Although Nekton sells feeders that purport to allow hummingbirds to consume the insolubles, many rehabilitators do not use feeders of that design. In addition, to reiterate, soy protein does not have an optimal amino acid profile for birds that, in nature, rely on insects for their protein. Commercial diets based on soy protein and fed to wild birds, both passerines and hummingbirds, have poor outcomes (Heineman 2000; pers. comm. with numerous rehabilitators). Possible reasons for this are emerging in the literature (MacLeod and Perlman, in prep.). In addition, quality control in animal foods is less rigorous than in products for human consumption. While the authors believe that the information on the label of a product designed for humans with digestive incapacity is likely to be reliable, they are less sanguine about the actual contents of commercial animal foods (e.g., Garrecht Gassen 2002).

Vital HN® is a complete diet for humans with digestive disease. It contains, among other ingredients, partially hydrolyzed proteins from whey, meat, and soy. The percent of the hydrolyzed protein that is, in fact, soluble, is not known precisely (D. Cockram, pers. comm.). Vital HN® also contains free amino acids that are soluble. The amount of truly soluble protein is, maximally, the total present, although it may be somewhat lower. Even so, in contrast to particulates, relatively large peptides (pieces of proteins), while not actually soluble, remain suspended in solution for roughly 10 hours. Thus they are available to the birds through the normal drinking process.

These two diets were examined (Figure 1 and Table 2) for the protein that is soluble/suspended and therefore available to hummingbirds. The Vital HN® diet is as described by Elliston and Gillett (1994), made with 4 g of Vital HN® per 180 cc of 20% sucrose solution. The Nektar Plus® diet is constituted as directed by the manufacturer.

FIGURE 1. PROTEIN IN DIET AVAILABLE TO HUMMINGBIRDS



It is clear that the amount of soluble protein in Nektar Plus® does not approach the amount of protein that hummingbirds provide for themselves in the wild, and presumably need (Figure 1 and Table 2). The amount of available protein in Vital HN® is higher than that in Nektar Plus®, but its level, though shown by Elliston and Gillett (1994) to be adequate for long-term "maintenance" use, is significantly less than what the birds consume in nature. As noted above, most birds that have been on these diets long-term have also had access to insects. However, in acute care, many birds cannot capture insects at all. Further, such birds have additional protein requirements for healing.

This cannot be remedied, with either of these products, by the use of "more" of them. Nektar Plus® is a mixture of sugars and other nutrients to which only water is added to make the diet, so it is not possible to increase the amount of protein relative to sugar. It would be theoretically possible to add more Vital HN® to the sugar-water solution, but adult birds tend to reject concentrations high enough to supply 100 mg/day of protein (authors' obs.).

However, there is a relatively simple way to address this problem. There exists a product for microbiological use called tryptone. Tryptone is an enzymatically digested form (pancreatic hydrolysate) of casein, a milk protein that is of high quality (i.e., has an amino acid profile that is a good match for animals that obtain their protein from animal sources). It is very soluble. In the authors' experience, it is also apparently palatable (or perhaps tasteless) to hummingbirds.

TABLE 2. CHARACTERISTICS OF CAPTIVE DIETS

Diet	Kcal/cc	Total protein (mg) per 6 Kcal	Soluble protein (mg) per 6 Kcal	Soluble protein as % of required protein
Nektar Plus®	0.53	43.5	17.4	17.4%
Vital HN®	0.9	25.3	[up to] 25.3	[up to] 25.3%

VITAL HN® DIET (ELLISTON AND GILLETTE 1994) WITH TRYPTONE

Nectar

60 cc (0.25 cup [C]) granulated sugar

Fill cup with warm distilled water (to a volume of 240 cc [1 C]) and stir to dissolve. Yields a concentration of about 24% sugar.

Vital HN® Mix (Whole Food)

180 ml (6 fl oz., or .75 C) nectar
4 g (6.7 cc or approx. 1/2 tablespoon) Vital High Nitrogen®
2 g (approx. 6 cc or one slightly rounded teaspoon [t]) tryptone powder
0.63 cc (1/8 t) Super Preen™
0.63 cc (1/8 t) Nekton Tonic-I®

Shake or stir until all Vital HN® and tryptone dissolve.

2 g of tryptone powder can be added to the 4 g of Vital powder, and the mixture dissolved as usual. This amount of tryptone plus 0.7 g of protein from the Vital will yield 2.7 g protein/180 cc, or 100 mg of protein in 6.7 cc of nectar, which is the expected intake of an inactive 3 g bird. If the bird chooses to eat more, it will take in more calories and more protein.

If Nektar Plus® is chosen as the base food, a tryptone solution will have to be made, the final concentration of which (after boiling; see below) is 50 mg/ml.

TRYPTONE SOLUTION

Tryptone solution is an exceedingly good medium for bacterial growth, so all possible precautions should be taken to prevent contamination and spoilage.

Weigh 5 g tryptone powder. The final desired volume of the solution is 100 ml, so add the tryptone to approximately 95 ml water. Stir or shake to dissolve and boil the mixture for 5–10 minutes to sterilize. Add sterile water to make 100 ml. Cap loosely while the solution cools so that bacteria cannot enter. Apportion it into several small, very clean or sterile containers, in portions of 5–10 ml. Freeze these immediately and keep them frozen until they are thawed briefly for use.

Nektar Plus® with Tryptone

Every 1 ml of the tryptone solution described above, when added to a day's food, makes available 50 mg of additional protein. If 6 Kcal of Nektar Plus® is to be supplemented to the level of 100 mg of protein per day, 80 mg of tryptone must be added, which requires 1.6 ml of a 50 mg/ml solution. This should be substituted in place of 1.6 ml of water in which the Nektar Plus® powder is dissolved. E.g., if 11 ml of water is used to make the day's food (5.8 Kcal), use instead 9.4 ml of water and 1.6 ml of tryptone solution.

(***Nutritional Note:*** One author [J. P.] recommends adding very small amounts of B-complex vitamins; vitamin C; and vitamins A, D, and E, along with a small amount of live-culture yogurt, to the food daily. The rationale for this is described, in part, in MacLeod and Perlman 2001. The yogurt adds only a small amount of lactose, and the *Lactobacillus* bacteria supply lactase that aids in its digestion. The *Lactobacillus* mainly benefits the bird by competing against pathogens [Reid and Howard 2000, p. 12] both in the food and in the bird's gut. In 4 years (cumulative) work with hummingbirds this author has never observed diarrhea in a hummingbird fed yogurt.)

The authors consider it essential to offer a choice of foods to self-feeding individuals. Three choices—water; a defined, nutritionally complete food source; and sugar water alone (with no additional nutrients)—provide two choices of food that are unlikely to spoil simultaneously.

Water, ad lib, can be chosen if needed by the bird. The plain sugar water provides a back-up to sustain the bird if the highly nutritious material becomes unpalatable. Again, hummingbirds will starve to death before they will eat something that tastes bad to them. (Some birds may choose sugar water to the near-exclusion of the complete diet, even if they will eat the complete diet in the absence of other choices. The rehabilitator must monitor intake of various foods and adjust their availability accordingly.)

To ensure that the broad spectrum of needed nutrients is offered the unflighted captive bird, it may also be advisable to provide as much natural food as possible in addition to the "complete diet" supplemented with tryptone. One possibility, reported by Van Epps (1999) in her work with nestlings, is to add pureed insects to the nectar. Such mixtures should be offered for very short periods (2 hours or fewer) to prevent spoilage, and another sugar source, unlikely to sour quickly, should be made available, as mentioned above.

Conclusion

As shown in Figure 1 and Table 2, even the most successful hummingbird diets in common use are probably deficient in protein for birds that are impaired in their ability to capture insects.

It is possible—and likely—that there are interspecific differences in protein requirements. There are surely variations in the requirements of a single bird over its lifetime and within a year, due to the demands of growth, molting, and breeding, for example. When there appear to be only the nutritional demands of maintenance on adult birds, they ingest about 100 mg/day of protein (about 3% of their body weight). Growth, molting, breeding, and healing require significantly higher levels of protein (Klasing 1998). The amount of tryptone suggested here only brings the total protein to 100 mg/day, and it is possible that injured, unflighted birds might benefit from more protein than that. The authors have not conducted studies to address that possibility, but more tryptone could, in theory, be added to the food to increase its protein content. More research should be made in this area.

While high-insect diets are eaten under at least some circumstances, there is no evidence that, conversely, hummingbirds in nature ever forego insects. Studies show that nectarivores retain much more protein than they nominally

"require," and it would seem prudent to provide them with the amount of protein in captivity that they ordinarily consume in the wild.

"Complete" dietary bases such as Vital HN® supply necessary nutrients that the highly purified protein is lacking, while the addition of tryptone to the diet of captive hummingbirds assures that their protein requirements can be met in the absence of their ability to capture insects.

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Product Information

Dexamethasone Elixir USP, a palatable pediatric formulation of dexamethasone, Barre National Inc. Baltimore, Md.

Nektar Plus® and Nekton Tonic-I® are available from Guenter Enderle Enterprises, 727/938-1544.

Prozyme™, a digestive enzyme, is available from Prozyme Products, Ltd. 800/522-5537; http://www.prozymeproducts.com/main.html

Super Preen™ powder, an avian nutritional supplement, is available from Arcata Pet. 800/822-9085; http://www.arcatapet.com

Tryptone is available as product WW-79-4420 from Carolina Biologicals, 800/334-5551; http://www.carolina.com/

Vital HN® is available through Ross Laboratories, a division of Abbott, 800/986-8510; http://www.ross.com/

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Downy Woodpecker Success

by Marjorie Gibson

ABSTRACT: The life of most wildlife rehabilitation patients remains a mystery following release back into the wild. U.S. Fish and Wildlife Service bird banding and the use of radiotelemetry have improved the odds of receiving post-release information on wildlife patients. In 1994 the Raptor Education Group, Inc., in Antigo, Wisconsin, received a baby downy woodpecker (*Picoides pubescens*). Following successful rehabilitation the bird was banded and released. Five years later, the bird was trapped at the site of its release.

This case provides information on successful rearing of downy woodpecker chicks, longevity, and suggests site fidelity.

KEY WORDS: wildlife rehabilitation, wildlife markings, bird banding, downy woodpecker, *Picoides pubescens*

MARJORIE GIBSON is director of Raptor Education Group, Inc, a nonprofit avian rehabilitation center located in Antigo, Wisconsin. She has been rehabilitating birds for forty years and is a former president of the IWRC.

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Natural History

Downy woodpeckers (*Picoides pubescens*) are the smallest woodpeckers in the Northern Hemisphere. The demure black and white bird measures only 15–18 cm (6–7 in) long. It is just one of two woodpeckers to have an all-white back. The other, the hairy woodpecker (*Picoides villosus*), is somewhat larger at 20–23 cm (8–9 in) (Sibley 2000, p. 312; Terres 1996, pp. 1018–19).

Like most birds of its species, the downy woodpecker's diet consists of a variety of insects, including many that cause damage to trees and therefore affect the forest-products industry. These insects include woodborers, grubs, weevils, gypsy moths, and many others. According to Martin, Zim, and Nelson (1951), berries, seeds, and nuts make up 14–29% of the bird's diet, depending on the season. This woodpecker's tiny size allows it to feed on shrubs and weed stalks as well as the more typical tree trunk or large branch (Kaufman 1996). Downy woodpeckers are most often observed in the winter. It is during this time when they congregate at backyard bird feeders with chickadees, nuthatches, juncos, and other birds (Terres 1996).

Rehabilitation History

In the summer of 1994 the Raptor Education Group, Inc. (REGI) in Antigo, Wisconsin, received a call from a woman stating that she had a baby downy woodpecker in her possession. She had the bird for a little more than a month. She did not have a rehabilitation permit. The history of how she came to have the bird was sketchy. The woman said that she was going on vacation and needed someone to look after the bird while she was out of town. She added that while the little bird was amusing, it did not seem to be faring as well as she had hoped. She expressed concern about how to determine whether the bird was ready for release.

A few hours later the woman appeared at REGI's receiving room, rusty cage in tow. The bird was indeed a young female downy woodpecker. The bird was nearly fully feathered; however, the poor housing situation had damaged many of its feathers. The decrepit old cage was extremely small, measuring about 25 x 31x 31 mm (10 x 12 x 12 in). It had likely been intended for a finch in the years before it was understood that even small species require adequate space to exercise. The woodpecker had no vertical perch and was forced to cling to the wire sides of the cage. Its chest and head feathers appeared to have been rubbed off. A commercial suet block—the only nutrition provided during the bird's confinement—was attached to one side of the cage, where it occupied most of the available space. The bird's body condition was poor, most likely due to a combination of inadequate nutrition and lack of exercise.

The first order of business was to provide the woodpecker with proper housing and nutrition. The little bird responded eagerly to its new vertical rough bark perches. The rough log surfaces enabled it to climb "woodpecker style" for the first time since it was fledged. After a few crashes to the floor of the flight, the bird quickly learned to how to hang on to the bark and move forward and back. Gut-loaded mealworms and grubs were provided in the crevices of the bark. The woodpecker probed them curiously at first, and then ate them with great relish. Within a few days, the young woodpecker was negotiating its new captive habitat skillfully. Its new diet included a good avian vitamin to augment the fresh food.

The woodpecker's feather condition was addressed next. The bird was covered with rancid suet grease; the greasy feathers resulted in part from physically contacting the suet and in part from ingesting so much of it. The bird was bathed three times the first week in Dawn® liquid detergent and then given access to a shallow water bath of fresh water, where it was able to continue bathing on its own.

Upon arrival at REGI, the woodpecker was placed in a communal shared flight with various nonaggressive passerines, a few chipping and song sparrows, and—during

the final week of captivity—an adult downy woodpecker. The adult downy was recovering from a wing fracture and was content to continue its rehabilitation without confronting the young woodpecker. (Woodpeckers can be aggressive to each other, so care must be used when introducing them into a shared flight.)

Release

Finally, in late September 1994, the young bird had completed its juvenile-to-adult body molt, replacing the damaged feathers. On the day of release, the bird was banded with a U.S. Fish and Wildlife Services band.

As is REGI's standard protocol with young birds with no identified breeding territory, the bird was released using the soft hack method. This type of release allows the bird to leave on its own when it feels comfortable to do so. The hack hatch was closed in the evening to prevent predators from entering, and opened again in early morning. The woodpecker came and went from the flight for a few weeks. In October, the young bird no longer returned to the site.

Opportunity Knocks

Years passed. A pigeon cage to house the center's flock of homing pigeons had been built in subsequent years near the former hack flight. Occasionally downy woodpeckers would appear at the bird feeder. My thoughts frequently turned to the pitiful little female that had been rehabilitated in 1994. One day in December 1999, my husband saw a female downy on the roof of the pigeon building. The bird stayed long enough to get a spotting scope on it: it was banded. This was noteworthy, but there are many woodpeckers banded during migration, so this was not necessarily a former REGI patient. The banded bird came and went for most of the year. One day I noticed a woodpecker inside the pigeon yard. As I ran out to free the bird from the yard, I saw a silver band on its leg. I could hear opportunity knocking loudly, and grabbed a net and quickly captured the little woodpecker. I raced for my banding log and paged through...1999, 1998, 1997, 1996, 1995. There, in the log for September 1994, I found the identical number of the little bird I held in my hand. The comments read: "Downy woodpecker, female, suet-covered, immature feather damage," and the corresponding rehab number. Our little grease baby had come back.

Conclusion

It didn't look at all like the young woodpecker REGI had admitted five years earlier. The bird's feathers were now bright and perfect. Its formerly marred face and beak from its time contacting the wire cage was now flawless. There was no sign of the rancid smell of tallow the bird had carried during its months at REGI.

We took photos and then released the five-year-old downy woodpecker within minutes of capture. The bird stayed most of that winter eating at the feeder, but did not repeat the mistake of getting inside the pigeon yard.

It is always exciting to see or hear from former patients. We like to think the birds are well and propagating their species as nature intended. It makes the joy all the greater when you have evidence they are not only making it, but have longevity under their wings as well.



MARJORIE GIBSON

The downy woodpecker, just prior to release in 1994.

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{ NEO-REHABILISM... **savian** *bird rescue* }

Dealing [with] Death: Psychological Aspects of Euthanasia in Wildlife Rehabilitation

by Diane Winn

ABSTRACT: Because of our exposure to injury and death, wildlife rehabilitators may be vulnerable to the same stress syndromes that have been documented in trauma workers. Euthanasia can be a particularly difficult aspect of wildlife rehabilitation. This paper discusses both physical and psychological strategies for administering euthanasia. It also suggests some ways to minimize or resolve uncomfortable reactions to euthanasia: allowing ourselves to grieve, talking to other rehabilitators about our experiences, and conceptualizing death, in this situation, as another type of release.

KEY WORDS: wildlife rehabilitation, stress, death, euthanasia, grief, release

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Introduction

As a rehabilitator who is also a psychologist, I read with dual interest the literature on "compassion fatigue" and "burn-out," syndromes that can arise in people who have close relationships with victims of trauma (see Figley 1995, for a discussion). Caregivers such as physicians, nurses, and psychotherapists are vulnerable, as are people such as paramedics, police, and firefighters, who work at disaster scenes. Although wildlife rehabilitators have yet to be recognized in the psychological literature as vulnerable to compassion fatigue, it is obvious to anyone in this field that we have a lot in common with the professionals mentioned above; our roles are similar to most of them *combined*. And burn-out in rehabilitation is something we have all heard about, if not experienced personally.

One reason trauma work is so stressful is that it can remind caregivers of their own traumas. Perhaps not many wildlife rehabilitators have been orphaned, kidnapped, shot, hit by cars, or attacked by cats, but death touches all our lives, and often in difficult ways. So the experiences we have with death in rehabilitation can be stressful not only because of what happens at that moment in our practice, but because of what it reminds us of in our personal lives. Several years ago I sobbed almost uncontrollably over a swift that had died while I was away from the clinic. It was not (just) the swift I was crying for though—it was also for my mother, from whose side I had also been absent when she died.

Having exaggerated feelings like these isn't necessarily a bad thing, but it is something we should pay attention to. If we find ourselves overly preoccupied with an animal's death (e.g., unable to forget it, having repeated images of it, feeling much more upset about it than seems warranted, etc.), we should take these feelings as a signal to consider getting some help with resolving the situation. Getting help for ourselves should not be seen as a failure, but as an intelligent part of good rehabilitation work. Additionally, if we don't get resolution on a particular experience with an animal, then our judgment might be clouded in dealing with the next traumatic event in our practice, and we may tend to under- or overcompensate with a new experience because of unresolved feelings from a past situation.

But spillovers between our personal and our rehab lives don't always come with warning flags; they are often much more subtle. Some years ago, my first baby of the season was a grackle with cat bites. It survived, but developed multiple problems in succession: a pox lesion, misaligned mandibles, a jaw tumor, recurring coccidia, and a coracoid fracture it probably got while in my care. In retrospect, it's obvious that the bird was nonreleasable. And although some part of me probably knew at the time that I was only delaying the inevitable, my father had died that summer, and I just couldn't bring myself to euthanize the bird; I'd had all the death I could handle right then. So I convinced myself that the grackle had a chance, got permission to overwinter it, and made an appointment in the fall with a homeopathic veterinarian for a constitutional workup. But the evening before the appointment, I found the grackle drowned in its water bowl, and have regretted ever since that I did not make its death an easier one.

We are not really trained to cope with all the different faces death wears in rehabilitation. And whether we are dealing with the deaths in one large-scale disaster like an oil spill, or the accumulation of deaths in a season's practice, or the death of just one animal we were particularly close to, there are rarely any counseling services or trauma debriefing sessions designed specifically for wildlife rehabilitators. (Lainson [1998], however, does suggest borrowing that technique from fire service and emergency medical personnel.) In particular, we're rarely taught how to cope with the impact euthanasia has on us. Unlike other trauma workers and caregivers,

a routine part of our job is *taking* lives—not just trying to save them. That aspect of our work makes compassion fatigue and burn-out even more difficult and complex potential syndromes. We "deal" death . . . and then we're left trying to *deal with* having done so.

The literature does contain some helpful material. Klein (2000), Richards (1993), and particularly Beaver et al. (2001) provide information on *how* to euthanize. Miller (1993) and White (1993) offer guidance regarding *when* to euthanize. Richards (1993) and Lainson (1998) acknowledge that euthanasia does evoke emotions; they adapt the Kubler-Ross (1969) stages of emotions in reactions to terminal illness to wildlife euthanasia. Miller (1993) makes the point that, although we can't remove the emotions, we can develop guidelines that make the decision easier and remove some of the doubt. Her point is well taken, but considering the animal first and dealing with our emotions second is often easier said than done. Emotions are *very* powerful motivators. The euthanasia process might be less difficult if we could proactively engage our emotions in it in a positive way, rather than "just doing" it and cleaning up the emotional aftermath later. So, as a psychologist, I would like to take the above discussions further.

The Physical Process

The first step is a mechanical one: figure out how you are going to euthanize, in a way that will cause not only your animal *but also you* the least discomfort. The animal is not the only one experiencing a death—you are too. Many people, myself included, want to be able to euthanize on site, rather than drive the animal to the veterinarian's office. Carbon dioxide is probably the most commonly used method in home-based centers; plans for constructing a CO_2 chamber are available in Seitz and Nuzzo (1998). In our early experience with CO_2 euthanasias at Avian Haven, birds sometimes appeared to be struggling to breathe, regardless of whether or not we heated the gas or prefilled the chamber. We found the process easier on both the patient and the caregiver to have the animal "asleep" for the euthanasia process.

Options for preliminary sedation include any tranquilizer that your veterinarian is willing to dispense to you. If your veterinarian is comfortable with your having isoflurane or halothane on site, isoflurane on a cotton ball can be used as a pre-CO_2 sedative; birds and small mammals may be euthanized by placing them in a small closed container with a halothane-soaked cotton ball (Beaver et al. 2001). Rehabilitators using isoflurane or halothane in this manner should talk with their veterinarian about human exposure cautions.

Presently at Avian Haven, we use our general anesthesia apparatus to anesthetize the birds with isoflurane, then we switch the oxygen to carbon dioxide. The birds "go to sleep" gradually and with no apparent discomfort. We find this procedure less stressful—both for the caregiver and for the animal—than injectable euthanasia agents.

Creating a Ritual

Once you have the mechanics of the physical process in place, think about how you could elaborate on it in a way that is psychologically comforting—again, for you as well as for your animal. All societies have developed death rituals to help both dying humans and their survivors make the transition. Why not design one for rehabilitators and our dying animals? As children, we created ceremonies around the deaths of our pets almost instinctively, without self-consciousness or inhibition or fear of looking foolish to another person. We did then what we somehow knew was necessary in order for us to come to terms with death, and we can allow ourselves to become aware of whatever it takes for us now, as the adult rehabilitators we have become. As McElroy (1997) wrote in *Animals as Teachers and Healers*, ". . . there is nothing foolish or sentimental about pausing for a moment, with respect, when we see a life end. Neither should we be ashamed for feeling discomfort, sadness, or even amazement at the finality of death, whether it manifests itself in the death of a sparrow or a human being" (pp. 179–80). A euthanasia ritual can be as simple as "pausing for a moment, with respect," or it can be more elaborate. Each of us can develop a process that is comforting to us as the unique individuals we are. I can tell you what I do, but I intend it only as an example that might inspire others to design their own ceremonies.

I start by giving the bird a flower essence combination called "Transition" that is formulated to help animals in crossing from body into spirit. When the time comes, I speak softly, saying something like this:

> "Sweet little cousin, your body is damaged beyond the ability of a clumsy human to repair it. Never again can you live free in the wild, nor can I offer you a comfortable and quality life in captivity. It falls to me to make the choice I think you would make if you could—to free your spirit from its damaged shell, which serves you no longer. It will not be painful; it will be as if your body has fallen asleep. When you leave it, look for and go into the Light, where you will find brothers and sisters to greet you. I will go with you as far as I can on your journey. May you walk and fly in beauty, little one. May your soul find love and peace. Thank you for sharing the final part of your time on earth with me. I promise never to forget the lessons you have taught me."

While the bird is under anesthesia, I hold it for a final treatment of Reiki (a spiritual "energy" healing practice that originated in Japan) until life leaves the body. Then I remove any bandages, wash the body if necessary, wrap it in a soft cloth (I may add flowers or stones), and put it in a special place designed for this purpose. It remains undisturbed for at least an hour before we do a necropsy or dispose of the body. People who use CO_2 could also create a chamber that is more than just a garbage can or aquarium. For a while, we used a round, white plastic container that we thought resembled an egg viewed from within. I made a nest inside it, and added objects that were special and sacred to me. I do not wish to imply that everyone needs or should need such procedures. But I encourage people who are aware of such a need to develop ideas for a euthanasia ritual.

Afterwards . . .

So now that we have "dealt" death, it's time to start dealing with death—going through the mental and emotional process necessary to get closure on that death, so that it does not continue to gnaw at us. Some part of that processing will prob-

ably involve grieving. We often think of grief as more or less synonymous with sorrow, but grieving can involve many emotions, including guilt, anger, and a feeling of helplessness, to name a few. It is important to let these emotions surface, to feel them and to go with their flow; there is a wisdom to it. As Lainson (1998) noted, "If grief goes unresolved and becomes internalized in the individual, problems are bound to arise . . . Unresolved grief comes from trying to jump from knowledge of the loss to acceptance of that loss without acknowledging and experiencing the feelings that are arising over that loss" (p. 93).

Sometimes while grieving we get stuck in wondering over and over again what would have happened "if only" this or that had been done differently. One thing that helps processing along is to tell the story—to talk about the chain of events that led to the euthanasia (or other death), and also to talk about what you thought and how you felt about that death. McElroy (1998) believes that telling the death story is a crucial tool for self-forgiveness—but adds that it is essential to have the appropriate audience. Talking about pain and remorse to someone who can't understand or identify with it can derail the process. The appropriate audience may, for some of us, be just ourselves—writing the story in a private journal, for example. But telling our stories to others helps heal not only ourselves, but also those who listen. It is important to know that we are not alone in these feelings. And "confessing" what we might see as a failure may result in some feedback to the contrary from others who have been there. Telling our stories helps balance the things we think we might have done wrong with the things we might not realize we did right. And this helps us to forgive ourselves and to acknowledge that, sometimes, we are truly powerless to do anything that can save the animal. So one of the things I would suggest is to make sure we have opportunities to talk about the deaths that are especially hard for us. We could establish grief networks, for example: we could promise to allow one other rehabilitator to call and share difficult experiences anytime, day or night, that the person needs to talk. We could find someone who will make that promise to us.

The point, again, is this: the animal is not the only one who is experiencing death. We, the rehabbers, are too, and I think that, as a community, we need to acknowledge this aspect of euthanasia more fully than we often do. The model of a dispassionate scientist, which we sometimes emulate in our desire not to be regarded as "bunny huggers," is the wrong model for most of us when it comes to death in rehabilitation; our relationships with these animals have often been much too intimate for that. Many of us conceal our grief—perhaps because it seems unprofessional to show it—but it is not far below the surface and often erupts if the door is opened even a crack. At a euthanasia workshop I gave recently, virtually everyone in the audience was in tears, as was I. But a woman who spoke to me afterwards told me that, in all her years as a rehabilitator, she had never until that day seen another rehabber cry. This, to me, was as sad as the deaths over which we had wept that afternoon.

The "Other Release"

Allowing ourselves to cry and to grieve is an important step to take, but there might be one more. Perhaps we can also deal with death by understanding it as something other than simply the end of life. So often we think of our euthanasias as our failures, but the idea of euthanasia as a gift is mentioned here and there in the rehab literature (see, e.g., Moore and Joosten 1997), and I would like to see this idea become more fully integrated into our understanding of euthanasia. Sometimes we feel that a euthanasia is a *betrayal* of our animal's trust, but another point of view is that it is a *fulfillment* of that trust, the ultimate gift we can give.

A related conceptualization that influenced me was the title of Miller's (1993) article: "Euthanasia: The Other Release." For Miller, this "other release" is one from pain and suffering; Frink (1993) used the term a little differently, however, when she talked about euthansia as a way to "release these wild creatures from their tortured bodies so that their spirits, at least, could fly free again" (p. 6). This is how I came to understand the "other release" too, and, for me, it's more than a comforting metaphor.

One of my interests as an academic psychologist is death studies. In my seminar (at Colby College) on the Psychology of Death and Dying, my students and I explore literature pertinent to the question of what (if anything) happens to consciousness when people die. Western psychology is still dominated by the materialist viewpoint that mind depends on brain; despite not understanding how mind could emerge from brain, it is widely held that, without brain, there can be no mind. But more and more evidence now suggests that mind can function independently of brain; for example, people who are clinically dead as far as heart, lung, and brain activity are concerned may report, when subsequently revived, having had conscious awareness while "dead." Sometimes they can accurately recount events that, as far as science is concerned, they had no way of knowing, since they had no working brain at the time. This is not the place to present that evidence (but for a review and discussion of the most striking cases, see Cook, Greyson, and Stevenson 1998; and Kelly, Greyson, and Stevenson 1999–2000), but it puts a slant on euthanasia that helps me deal with it. If consciousness or spirit (or whatever one chooses to call it), can operate without a functioning brain, then perhaps it could operate without a living one, and perhaps the phenomenon could occur in animals other than humans.

I realize that the above ideas may be very different from the belief systems of some people. But belief is not required; I simply suggest that, if we accept *as a working hypothesis* the idea that euthanasia releases a spirit from a hopelessly damaged body, it may be easier for us to deal, and deal with, death. This conceptualization is not a substitute for grieving. I still cry over some of the birds I euthanize; I hope I always will. But my grief is modulated by the thought of the bird's spirit finally flying free. And some of my tears are tears of joy for that spirit, just as they are when I set birds free in those "other releases."

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Product Cited

Flower essence combinations called "Transition," and other essences for animals, are made at Green Hope Farm, Meriden, N.H.; 603/469-3662, green.hope.farm@valley.net and at AnaFlora, Mt. Shasta, Calif.; 530/926-6424, anaflora@mail.snowcrest.net.

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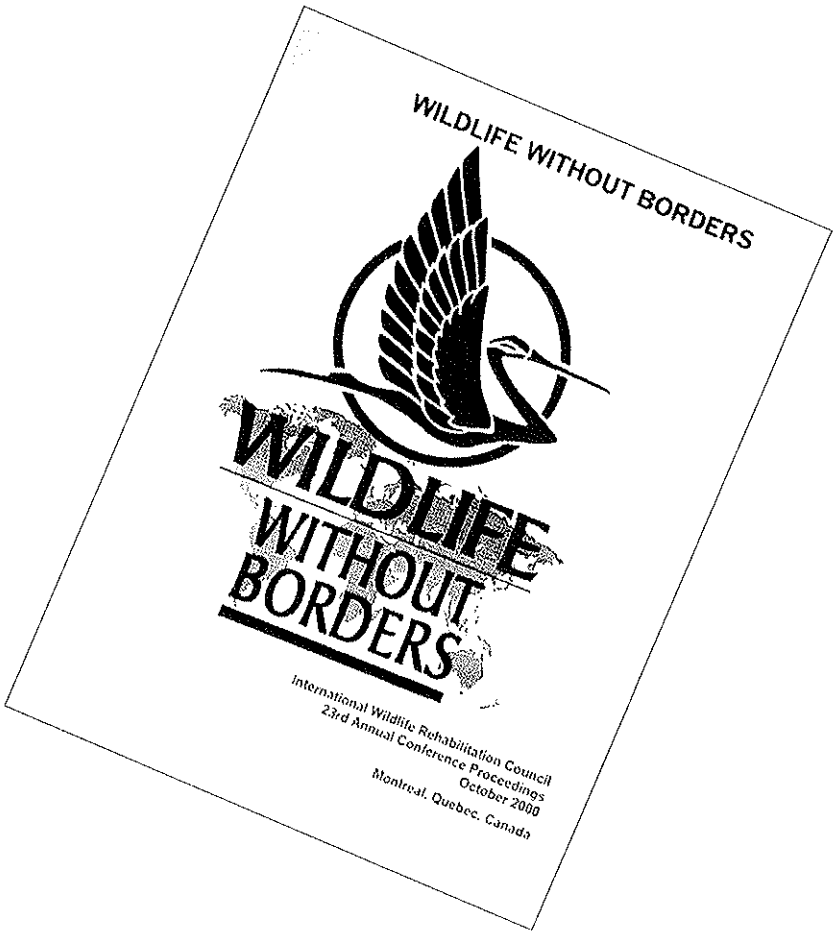
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Operating Instructions

URBAN CANADA GOOSE ISSUES

by Nathalie Karvonen

Canada geese, our feathered urban neighbors, are often a cause of frustration for people. With the proliferation of artificial urban landscapes across North America, we have inadvertently created paradise for these birds.

When dealing with members of the public who are angry at the prevalence of geese in an area, providing information about the natural history of this species will help the caller to understand that the geese are not behaving with malicious intent.

If you were a goose in an urbanized area, you would look for:

- Short grass, which is easier to walk on and hard for predators to hide in.
- New, growing shoots (e.g., recently cut grass) to feed on.
- Open expanses of green space (where it is easier to see predators coming).
- Easy access to water from land, and land from water.
- Hand-outs from human visitors.

Now, think about typical urban areas, such as parks, golf courses, and corporate and public green spaces. We continue to design and maintain areas with characteristics that make them the perfect habitat for Canada geese.

The good news is that habitat modification may provide an easy solution. Recommend that callers:

- **Let their grass grow** at least 4 inches high.
- **Plant trees, bushes, and gardens with tall plants,** or put up fences to create visual barriers (at least 30 inches high) for the geese. If geese think that a coyote may be lurking behind a bush, they won't be comfortable spending much time near it.
- **Put in dense, thick barrier plantings along the edges of ponds, streams, and other water bodies** in high-goose-traffic areas. Avoid gaps in vegetation that will allow geese access. If that coyote comes along, the geese want to be able to rush directly into the water, especially if they have young or are flightless because they are molting. If geese are in the water, they want an easily accessible land area for foraging.
- **Use a bird feeder that has a tray underneath** to prevent spillage. (Spilled seeds can make a tasty snack for the geese.)
- **Stop well-meaning people from feeding the geese** by distributing information pamphlets, putting up signs, or hosting a meeting. It is a bad idea to feed wildlife because bread is not a nutritious food for geese, the geese will lose some of their natural fears of people, and the handouts will encourage the birds to stay permanently.

Our local zoo had a serious problem with a walking path that cut through a grassy area and was close to a waterway: Canada geese loved the area and had moved in. The public complained frequently about having to step in and around the geese's feces. After some investigation, a landscape architect working for the zoo decided to plant native grasses, flowers, and other vegetation along the edge of the waterway. The zoo put educational signs among the plants to explain what they were doing. Soon native birds, as well as butterflies and other insects, were attracted to the plants. The geese no longer had easy access to the water, so they moved on. And for the people, the walking paths were free of poop—and the new plants were beautiful.

Canada geese are notorious for picking inconvenient spots to nest—in front of the doors of a workplace, in the middle of a parking lot, even on balconies of apartment buildings. At a wedding reception I attended recently, a goose had nested in a large planter on the outdoor patio. All night long, people chicken-danced and time-warped by her. She didn't budge, but rather nipped at the butts of passers-by who came too close for comfort. Later I was told that she nests there every year, and every year they have wedding receptions while she sits on her eggs. Often there is an aggressive gander defending the nest, and usually he is of greatest concern to people.

It is important to educate your callers about the breeding habits of Canada geese, especially the length of the incubation period and the fact that soon after hatching, the parents will take their young and leave the area. Often putting up a temporary barrier, such as construction fencing, will keep the birds safe in a very busy area (such as a parking lot) or may stop the gander from charging (if the geese and people are on opposite sides of the fence). Make sure that the caller does not feed the geese while they are nesting, as this may encourage them to stay once the eggs hatch. We do suggest that callers put out a bowl of water if the nest is in a hot area, as long as the gander won't beat them up!

Remind callers that Canada geese are federally protected birds (in Canada and the U.S.), so regardless of how annoyed they may be with the geese's activities, they will need a federal permit to interfere with adult birds, nests, or even eggs. Let them know, too (in case they decide to ignore the law), that if the nest is moved any significant distance at all, the adults likely won't recognize it as their own and will likely abandon the eggs.

For more information, refer callers to the Humane Society of the United States at www.hsus.org or Geesepeace at www.geesepeace.org. Hopefully by working with the public, we can help them to find a resolution to their concerns and a peaceful coexistence with these beautiful birds. JWB

Nathalie Karvonen is the executive director of the Toronto Wildlife Centre. TWC operates a wildlife hotline service available to the public. Last year the hotline received more than 16,000 calls.

COLUMN

Food for Thought

MACROMINERALS

by Astrid MacLeod and Janine Perlman

Minerals, in the context of nutrition, are inorganic substances required for life and health. *Inorganic* refers to the fact that they are not generally part of molecules containing carbon—i.e., they are not substances made by other living things. Macrominerals, somewhat arbitrarily defined by their relatively large amounts in the vertebrate body, are calcium, phosphorus, magnesium, sodium, potassium, and chloride. Sulfur technically qualifies as well, but as it is ordinarily obtained in the sulfur amino acids of protein, it is not generally considered separately from protein intake.

Phosphorus

Phosphorus (P) is a sort of counterpart to calcium (Ca), which was discussed in a previous "Food for Thought" column (*J. Wildlife Rehab.* 24[1]: 26–27). Phosphorus is needed as a "partner" to calcium in bone, in a weight ratio of about 1 P: 2 Ca. Together, they constitute the crystals (apatite) that form the hard bone matrix. Phosphorus is required in many other roles as well, including as a component of cell membranes and nucleic acids (DNA and RNA); as a storage depot for chemical energy; and as an acid-base buffer. Phosphorus is adequate to plentiful in most foods, and it rarely needs to be supplemented if nutritious and natural diets are supplied in captivity. However, much of the P in grain occurs in the form of phytates, which are not bioavailable.

An excess of phosphorus can cause metabolic bone disease. It should be present in the diet at about half the level of calcium, and at an absolute level (on a dry weight basis) of 0.1–1.0%, depending on age and species.

Magnesium

Magnesium (Mg) is a mineral about which little is heard relative to its very great importance. It is a significant component of bone and is present at relatively high concentrations within cells. It is a necessary cofactor for at least 300 enzymatic reactions, including many that harvest energy from foods and those that synthesize RNA and DNA.

Chlorophyll is a molecule that is complexed with magnesium. Plants store the mineral in their seeds as well, so it is widespread in whole foods of plant origin; animal-based foods are generally adequate sources. Magnesium probably does not receive a lot of attention because deficiencies are uncommon, and toxicities require intentional supplementation. There is growing evidence, however, that current American (human) diets may be deficient in this mineral.

Magnesium is one element of urinary tract struvite "stones," or calculi. This type of "stone" forms in alkaline urine, which is why, with the aim of acidifying the urine, phosphoric acid is added to cat food.

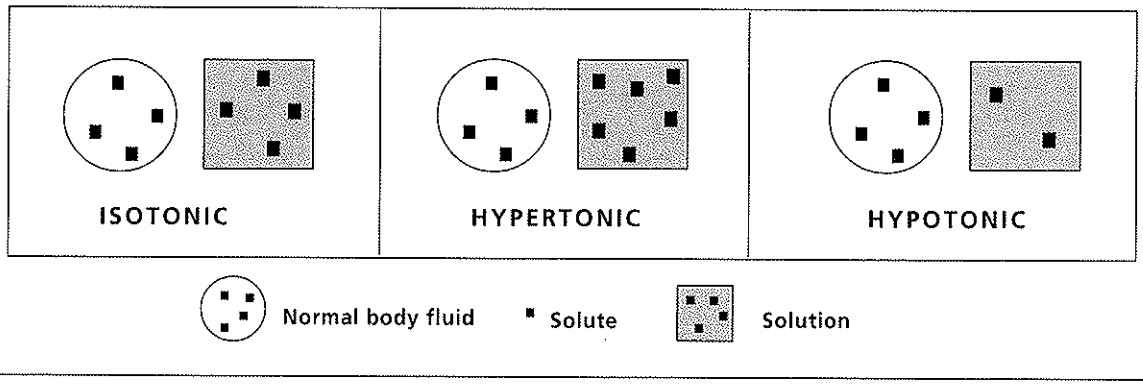
Electrolytes

Blood and other extracellular fluids bathe the cells of animals in salt solutions similar to that of the seas in which life evolved. Salts are composed of ions —charged atoms or molecules. For example, table salt is NaCl. Dissolved in water, it dissociates into Na+ and Cl-. Electrolytes are dissolved salts present in all body fluids.

Sodium (Na+), potassium (K+), and chloride (Cl-) are the major electrolytes. Positively charged ions are *cations*; those that are negatively charged are *anions*. Simplistically, cations tend to serve the most central functions, but with minor (yet crucial) exceptions, charged species cannot exist alone; they must be balanced (neutralized) by the presence of an equal number of ions of the opposite charge. Chloride often accompanies cations, in maintaining charge neutrality.

Electrolytes serve several life-sustaining purposes. First, they are *osmoles*—dissolved substances (solutes) that maintain the correct amount of water in blood, cells, and fluids around and between tissues (interstitial fluid). Cell membranes and the lining of capillaries form semipermeable barriers to bodily fluids. Water passes freely through these membranes, but many solutes do not. Physical/chemical laws dictate that solutions "want" to be uniform everywhere. Therefore, if the concentration of a salt is higher on one side of a mem-

FIGURE 1. ISOTONIC, HYPERTONIC, AND HYPOTONIC SOLUTIONS



brane than on the other side, and since the membrane is permeable to water but not salt, water will flow through the membrane to the side of higher salt concentration until the two sides are equal. This is the process of *osmosis*.

In healthy animals, blood, extracellular fluids, and cells are *isotonic*, meaning that the tonicity, or solute concentration, is "physiological," and allows metabolic processes to take place normally. *Hypertonic* solutions are those with solutes at higher concentrations than physiological; *hypotonic* solutions are more dilute (have more water and less solute) than normal body fluids (Figure 1).

An animal that is severely dehydrated for any reason has lost both water and electrolytes. Particular routes of electrolyte loss include vomiting, diarrhea, burns, and blood loss. Without the replenishment of electrolytes as well as water, there will be insufficient osmoles to "hold" water in cells, blood, and other fluids, and rehydration attempts will fail. In most cases, isotonic or hypotonic solutions should be used. Hypertonic solutions, on the other hand, are themselves dehydrating, because water will be pulled from the body generally, into the compartment where the concentrated solutes have been introduced.

The kidney is the main regulator of the body's overall electrolyte/water balance; its function is controlled by a series of hormones and negative feedback loops.

Another crucial role of electrolytes is the maintenance of an ***im***balance across cell membranes. The concentration of sodium in the extracellular fluids (interstitial and blood plasma) is high compared to that within cells. Intracellular fluids, on the other hand, are high in potassium. This difference creates an electrical charge difference between the interior and exterior of the cell, and it is that voltage across the membrane that allows the transport of important molecules in both directions. Without the constant flux of metabolites in and out of cells, life comes to a halt.

Because cell membranes are slightly leaky to some salts, an active and energy-requiring pump (actually, a protein of which there are many copies) sits in the membrane and continuously pumps Na+ out of, and K+ into, every cell. Toxins that interfere with the Na+/K+ pump are, as you might imagine, very deadly poisons.

The "counterpart" relationship of sodium and potassium is exemplified in the fact that dietary sodium increases blood pressure, while potassium appears to help lower it.

There are a number of ions that function as electrolytes in addition to the macrominerals. All the electrolytes influence, in complex ways, the pH (acidity/alkalinity) of bodily fluids.

Although cations are of obvious import, chloride plays crucial and specific roles as well. The stomach secretes HCl, or hydrochloric acid, which functions both as protection from pathogens and as a medium for chemical and enzymatic digestion of protein. The body-wide importance of chloride is demonstrated in cystic fibrosis (CF). CF is a genetic disease that results from a defect in the channel in cell membranes (a protein) through which chloride ions transit.

Animals acquire electrolytes in a variety of notable ways. As babies, altricial birds are fed animal-based foods almost without exception. In addition to meeting their needs for protein, these foods supply sodium, which is typically scarce in plant foods. Columbids (doves and pigeons) secrete crop milk, which contains appropriate sodium. Infant mammals, of course, drink milk, which is high in potassium as well as sodium.

For obvious reasons, sodium is adequate in whole foods of animal origin. However, Na+ can be difficult for plant-eating animals to obtain since, as mentioned above, many plants, fruits, and seeds are sodium-poor as a result of sodium-poor soils. New spring forage is particularly deficient, at a time when lactating animals require more sodium than at other times of year. In some areas, productivity and even survival of florivorous wildlife appear to be limited by dietary sodium. Salt licks can attract many kinds of plant-eating wildlife.

Marine animals fed freshwater fish in captivity can experience sodium deficiencies.

In nature, sodium can also be present in dietary excess, in the ocean and in inland bodies of water with high salinity. Seabirds possess salt glands that concentrate salt and excrete it through the nares. (Marine birds that have been deprived of saltwater in captivity must be given gradually more saline water before their release in order for their salt-excretion glands to regain function.)

Reproductive success of birds that harvest food from high-salt water depends on the availability of fresh water and/or freshwater prey for their nestlings.

Potassium is adequate in animal-based foods, and plant foods are typically rich in the mineral.

There are optimal dietary levels of sodium, potassium, and chloride, both individually and in relation to each other. Animals can generally tolerate electrolytes at levels considerably above their minimal requirement as long as fresh water is freely available. Of course, fish and reptiles and amphibians that live partly or completely in water have specific requirements for the salinity of their aquatic environments.

Macrominerals are so crucial to the structure and function of animals that we truly "embody" the sea and the earth from which we evolved. In our bones and blood and fluids, we and our fellow creatures carry the chemical elements that reflect our origins in the primeval oceans. In the veins of every living animal courses our 3.5-billion-year-old heritage. We are all living fossils. JWR

The authors welcome feedback and suggestions for future columns. Please feel free to contact them.

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Janine Perlman, Ph.D., *is an avian rehabilitator and has conducted laboratory research in biochemistry and molecular genetics for twenty years. She can be contacted at jpandjf@swbell.net.*

NEO-REHABILISM...

dehighdration
when fluid levels crash

COLUMN

Open Minds

KEEPING WILDLIFE WILD

by Beth Dillingham

Want a six-foot-long iguana that is used to having his own bedroom? How about a Bengal tiger in your backyard? Care for a macaw that recently chewed the corner off a dining room table? These are all calls or stories I have run across as a zoo educator.

As wildlife rehabilitators and educators, we all encounter the confusion people have about wild animals as pets. In this column I'll offer some strategies to counter the public's fascination with owning a wild animal and to help people think more clearly about keeping these animals healthy and in the wild.

Our consumer culture seems to have no boundaries. People want to connect with nature; they also want to distinguish themselves as individuals. These desires frequently lead people to assume they can own and keep wild animals as pets. Often this results in terrible tragedies for the animals and sometimes for the people. There have been more than a few "pet" tigers whose instinctive behaviors have caused deaths or severe injuries. I have given many presentations about wild animals and their habitat and how we can help make sure they can continue to live in the wild, and am discouraged when both kids and adults ask me where they can get the animal for a pet. How lucky you are if you are working with a protected species and can say one just can't have them as pets. Even if your animals are protected, it is important to get people to understand and respect the difference between companion animals and wild animals.

Animals that have been domesticated have been selectively bred over generations to exhibit characteristics that are of use to humans or that help the animal adapt to human environments. There is a big difference between the behavior of a domestic dog and its ancestor, the wolf. The large number of wolf and wolf-hybrid rescue programs around the country are evidence of the problems involved with the confusion of these animals as "pets."

A wild animal lives in its natural and uncultivated state. It has evolved characteristics to help it survive in the natural environment. Recently the Rio Grande Zoo became home to three orphaned mountain lion cubs whose mother had been killed by a hunter. Even as babies, their play was all about developing the skills necessary to kill prey. (For instance, one of the cubs pounced on my neck in play.) It would never occur to me to want to have one of these wild animals at home. Similarly, I continue to be amazed when people ask me if the zoo's staff "all pet the polar bears." But, as the saying goes, these questions give me a once in a lifetime opportunity to educate.

How can we encourage people to join us in our fascination with wild creatures without wanting to own them? How can we teach them to respect and admire the animals' wildness for its own sake? We have an important role as the interface between these animals, the natural world, and human civilization. In this role, we need to represent these animals as best we can.

First, while the individual stories of the animal that you are working with are very powerful, **it is important to connect that individual's story to the whole species.** Give information about the role of that animal in the natural ecosystem. Tell your audience about the problems that species faces in the wild. Talk to them about how people can change their behavior to help that species to continue to exist and to fulfill its role in the web of life.

Names can be very powerful. Many people choose not to name the animals at all, in order to avoid the attribution of human characteristics to these creatures. If you do use names, however, pick names that can connect to your message. I have a three-year-old friend who decided to name our 300-pound anaconda snake "Fluffy." But I call her "Amazon," in honor of the river anacondas live in and for their powerful nature. We have a prairie dog named "Sage," for the sagebrush areas that prairie dogs often live near. We call our ferret "Hunter," because of the hunting ferrets do to find their prey. We call our alpacas and llamas "Lima," "Peru," and "Inca" to represent where they came from in the world. When you have only a short time to connect with people, the name you give your animal can help reinforce your message.

Avoid emphasizing your personal relationship with an education animal. I once saw a humane educator hold a blind great grey owl during her presentation. The owl was huddled up next to her because it felt safe, and she caressed it as she talked. I have to say, this made me think more about wanting to hold that owl myself that way, rather than seeing the owl as a wild being. When kids see us petting an animal, they want to do it, too. If possible, put the animal on a perch during your education programs. If you have to hold it, avoid petting the animal in front of the public. Wild creatures have incredible charisma, and that charismatic draw extends to the educator. This can be a powerful tool, but unless you are careful your message will get lost as the audience focuses more on wanting to be like you or have one of those animals and less on what you are actually saying.

Any up-close contact with a wild animal will provoke myriad responses from your audience, but with thought and care you can move them beyond a simple desire to own that wildness. We must strive to encourage a respect and an empathy for these animals that leads to a motivation to keep them wild. JWR

Beth Dillingham has taught in both classroom and informal learning settings for fourteen years. She ran an environmental education center in the Sandia Mountains in New Mexico, and is currently the education coordinator for the Rio Grande Zoo. She may be reached at thebnm@earthlink.net.

COLUMN

By the Book

DEFINITIONS OF "GAME BIRDS," POWER-LINE ELECTROCUTIONS, AND MORE

by Rob Lee, Special Agent, United States Fish & Wildlife Service (USFWS)

Q As far as I can tell from reading the lists of federally protected birds at the U.S. Fish and Wildlife Service (USFWS) website, game birds (quail, pheasants, etc.) are *not* federally protected. Is this true? Does one only require a state permit to rehab them.

A There are three regularly accepted definitions for "game birds":

(1) **Migratory Game Birds:** Those families of birds included in the terms of conventions (treaties) between the United States and any foreign country for the protection of migratory birds, for which open hunting seasons are prescribed and that belong to the following families: Anatidae (ducks, geese, and swans), Columbidae (doves and pigeons), Gruidae (cranes), Rallidae (rails, coots, and gallinules), and Scolopacidae (woodcock and snipe). This definition is found in your regulation package at Title 50 Code of Federal Regulations Part 20.11(a). While these species may be hunted, they are still all protected by both state and federal laws. Rehabilitating them always requires a federal permit and a state permit (if your state issues permits).

(2) **Resident Game Birds:** Those species of nonmigratory birds that are regulated by the states and for which there are state hunting seasons. Generally these are quail, turkeys, pheasants, chukars, and prairie chickens. They may be native or exotic. They are not protected by federal regulations and you do not need a federal permit to rehabilitate them. You would need a state rehabilitation permit (if your state issues permits).

(3) **Non-Game Birds:** Those species of birds that do not have an open hunting season. This is a very diverse group that includes everything from auks to woodpeckers, raptors, wading birds, songbirds, and just about every other kind of bird native to the U.S. not already mentioned. Generally, these are migratory birds that are protected by both state and federal law that would require a federal rehabilitation permit and a state permit (if your state issues permits).

The short answer to your question is: pheasants, quail, and related species are "Resident Game Birds," and a federal permit is not required to rehabilitate them.

Q Could you please expound on what law enforcement does when we report an electrocuted bird? It's my understanding (and local experience) that they *do* check it out, and sometimes do cite and fine the utility, and require that the pole in question be retrofitted.

A Bird, especially raptor, electrocutions are a significant problem throughout the world. It was not too long ago that we would lose 100–200 bald and golden eagles in the panhandles of Oklahoma and Texas every year. While this type of killing is unintentional, it is still a violation of the Eagle Act and the Migratory Bird Treaty Act (MBTA). It has been the policy of the USFWS to notify the "owner" of the pole and request that they modify the pole according to the latest edition of "Suggested Practices for Raptor Protection on Power Lines," published by the Edison Electric Institute and considered the state of the art for preventing raptor electrocutions. Provided the company makes the modifications, no charges are filed. Gradually, the poles that kill are being identified and corrected.

However, if a company fails to comply, charges will be filed, as was the case in the 1998 prosecution of Moon Lake Electric Association in Colorado. They were charged with killing (electrocuting) 12 golden eagles, four ferruginous hawks, and one great horned owl after they had been warned that their lines were killing birds and they failed to respond. Moon Lake filed a motion to have the charges dismissed, arguing that the MBTA and the Eagle Act "...do not apply to unintentional conduct that is not the sort of physical conduct normally exhibited by hunters and poachers." In a very important decision, U.S. District of Colorado Judge Lewis T. Babcock denied Moon Lake's motion. They subsequently were sentenced to pay $100,000 in fines and restitution, were placed on three years probation, and were ordered to retrofit their lines to prevent future electrocutions. This is truly a landmark decision that prompted considerable reaction by the utility industry to proactively identify and correct electrocution hazards to migratory birds.

Q Rehabilitators frequently have to deal with the disposal of deceased animals. The law provides disposition alternatives of burning, burying, or donating such material to an appropriate public or educational facility. Do permitted rehabilitators need special permits to prepare and transport carcasses to an approved educational institution (e.g., museum, university)?

A No. But transporters should carry a copy of your permit when transporting migratory birds, whether they are alive or dead.

I encourage permittees to find proper facilities to which to donate specimens. They are valuable for education and research if you provide locality and date data with the specimen. JWR

Rob Lee has been a special agent for the USFWS for fourteen years. Prior to that he served as a refuge manager with USFWS for eight years. He is a certified wildlife biologist and has a MS in biological science from California State University, Sacramento.

NEO-REHABILISMS...

dumbrella permit
enough said

mapillaria
what you wish the directionally challenged member of the public had, as you're 40 minutes into waiting for them to find your house

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FACT SHEET

Rehabilitation Notebook: AVIAN POX

Causative Agent

Avian pox, or fowl pox, is caused by a large virus belonging to the avipox virus group, a subgroup of poxviruses. This group contains several similar virus strains, some that are species-specific, and others that have the ability to infect similar groups of birds.

Clinical Signs and Lesions

- Rapidly enlarging, proliferative masses on the face, feet, and other featherless areas of the body.
- Lesions on the face and feet are most common; lesions on the face usually occur around the eyes and commissures of the beak.
- The number and size of the lesions is variable, from small, scabbed-over nodules to multiple, extensive masses that may interfere with vision and feeding.
- Affected birds may have difficulty breathing if the lesions interfere with air intake, and may be thin or underweight due to difficulty feeding.
- Lesions generally spread and enlarge over a 1–2 week period, and regression usually occurs within 4–6 weeks, resulting in rapid resolution of the disease.
- Death can occur if affected birds are unable to feed or breathe, or if vision is impaired.
- Secondary bacterial and fungal infections are common and contribute to the mortality seen with this disease
- A systemic form of this disease, called "wet pox," can occur in wild turkeys and some other birds; this form of avian pox causes severe digestive and respiratory lesions and is often fatal.

Common Hosts

- Raptors (hawks, falcons, and eagles; very rarely owls)
- Chickens and wild/domestic turkeys
- Waterfowl are not commonly affected.
- Songbirds
- Marine birds

Transmission

Avian pox is most commonly transmitted by mosquitoes, but direct contact with virus-contaminated surfaces (e.g., bird feeders and perches) can result in infection when the virus enters the body through abraded skin or mucous membranes. Transmission can also occur by inhalation of infected dander, ingestion of virus-containing scabs, and direct contact between birds.

Seasonality

Avian pox infections can occur year-round, but are more common during the mosquito season and during periods of increased temperature, humidity, and moisture. During the winter months, increased incidence of infection can occur in songbirds that congregate at bird feeders and feeding stations.

Diagnosis

- Diagnosis can be inferred based on the characteristic clinical signs and appearance of pox lesions.
- Microscopic examination of tissues from affected birds may reveal characteristic particles inside the cell called Boolinger bodies, and this can confirm diagnosis.

Treatment and Release of Rehabilitated Birds

- Because avian pox is a contagious disease, infected birds must be isolated indoors. Strict hygienic practices must be followed to prevent its spread to other birds.
- Many birds are able to mount an effective immune response without treatment and eliminate the disease 4–6 weeks after infection. These birds are believed to have lifelong immunity to the disease.
- Birds that are unable to eat or drink, or that have secondary infections, may need supportive care, antifungals, and antibiotics to help them recover.
- Birds that have recovered from this disease are not considered to be carriers of the disease, and can be safely released once they are rehabilitated.

How You Can Help Control the Spread of Avian Pox

- Eliminate sources of standing water in the yard that could be breeding grounds for mosquitoes (buckets, old tires, etc.).
- Regularly clean and disinfect bird feeders and bird baths with a 5% bleach solution.
- Consider eliminating tube-style bird feeders and using feeding trays or platforms instead. This will limit the amount of facial contact with the feeding surface, which can be a source of mechanical transmission of the disease.

This Fact Sheet is based on one prepared by the Wildlife Center of Virginia and is published with the center's approval.

Conservation Classics: *The Education of Little Tree*

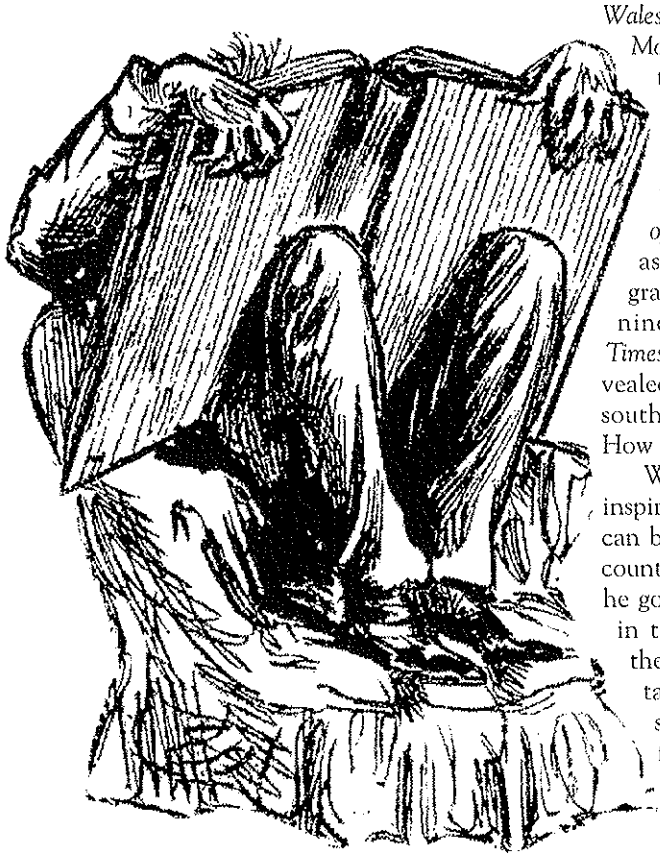
by Forrest Carter
Reviewed by Elsa C. Bumstead

Albuquerque: University of New Mexico Press, 1976
(reprinted 1986, fifteenth paperbound 1993)
216 pp.; U.S. $7.95 (paperback)
ISBN: 0-8263-0879-1

I wondered how this slender book, the winner of the very first ABBY (American Booksellers Book of the Year) Award, could be surrounded with controversy. The ABBY Award was created in 1991 by the American Booksellers Association "to honor the 'hidden treasures' that ABA bookstore members most enjoyed recommending to their customers during the previous year." My subsequent investigation uncovered conflicting information about the author. Who was this man who wrote such a poignant story about a boy growing up in the hills of the eastern Cherokees?

Forrest Carter (1925–79) was born Asa ("Ace") Earl Carter in Alabama. The eldest of four children, he and his siblings were raised by both parents in Oxford, Alabama. After finishing local schools, serving in the Navy, and attending the University of Colorado, Carter became a radio broadcaster and political writer. Too lengthy to discuss here, be advised that there is varying information about his association with violence and his years as a segregationist, KKK member, and speechwriter for George and Lurleen Wallace during the 1960s. Eventually, Carter ran as a gubernatorial candidate against Wallace in 1970, and finished at the bottom of the pack.

It was about that time that he began a remarkable turnaround and remade his image. He assumed the pen name "Bedford Forrest Carter" and the persona of a self-taught, part-Cherokee novelist, Gundi Usdi ("Little Tree"). He moved to Texas and wrote his first novel, *Gone to Texas* (1973). It was made into a successful movie, *The Outlaw Josey Wales*, with Clint Eastwood, in 1976. Carter then moved to Florida, where he wrote a sequel, *The Vengeance Trail of Josey Wales* (1976), and *Watch for Me on the Mountain* (1978) about Geronimo. At the time of his untimely death, Carter left an unfinished sequel to his third book—the one bound to be remembered—his boyhood tale about *The Education of Little Tree* (1976). First marketed as a Native American autobiographical account, it appeared for nineteen weeks on the *New York Times* bestseller list before it was revealed that it had been written by a southern-born "white supremacist." How could *that* be?



Written in the first person, it is an inspirational story of a Native American boy growing up in Cherokee hill country. Orphaned at the age of five, he goes to live with his grandparents in the Depression era 1930s. And there, through his eyes, begins the tale of what it means to have a sense of place: to be home, to be family, to be loved, and to be "educated."

It will leave you with much to ponder. What does it mean to "govern" and be "government?" Why is it so important to have standardized schooling and to pressure people to conform to dominant society's way of thinking and doing? What is the value of diversity? What does it mean to tolerate others and respect individual opinion? Do we all have to think and be alike? Is new meaning given to the word "rehabilitation?" What is so comforting (or discomforting) about "kinship" and spiritual relationships with the land and the sky?

In examining why *Little Tree* is a classic, I looked at many reading lists that the book appeared on—for eighth and ninth grade middle-school summer reading, corporate leadership training, environmental organization recommendations, and as a suggestion for "Top Ten Reads of the 20th Century." Oregon State University posted it on an annotated bibliography for students in a course on Multicultural Issues in Education. The Literary Society of San Diego read it as its November 2000 selection, and chose it as one of its "top choices for the year 2000." Praised by some Cherokees and criticized by others, *Little Tree* has grown to appeal to a wide audience in just a few decades.

Read this "hidden treasure" and decide for yourself whether it is a classic. You will meet some great characters. You will laugh. You will cry. And you will get an "education" about culture and perspective, about "wild" life and habitat, and about human behavior and the ways indigenous people compare to those of dominant society. It is unforgettable. JWR

Reviewer Elsa C. Bumstead is an interpretive naturalist, writer, environmental scientist, and sometime wildlife rehabilitator who is based in Albuquerque, New Mexico.

Conservation Classics is intended to provide our readers with suggestions for building a collection of must-have wildlife and ecology reference books.

Wildlife Under the Microscope

CD-ROM by Stuart L. Porter, V.D.M.

To order, see end of review.
28.6 MB; U.S.$28, students; $43 others
(cost includes shipping in U.S., Canada)

I recently purchased Dr. Stuart Porter's *Wildlife Under the Microscope* CD-ROM, and as a microbiologist, instructor, and volunteer raptor rehabilitator, all I can say is "Wow!" This is the most comprehensive work of hematology, urine analysis, and parasitology of non-companion, wildlife animals I have ever seen in one reference. Dr. Porter's 27 years of experience as a veterinarian, treating domestic animals and wildlife, as well as his experience as an instructor teaching clinical laboratory procedures is apparent in this work.

Everything and more that an individual rehabber or a larger rehabilitation center would need to enhance a pre-existing laboratory or set up a small-scale diagnostics laboratory is included in this CD. The CD would also make an excellent in-house training tool if supported by hands-on lab work. Even if a rehabber were not doing in-house testing, the information and the ease with which tests are explained would help one navigate through the medical terminology used by veterinarians, thus facilitating communication with consulting vets and/or reference labs.

Presented in a PowerPoint format, *Wildlife Under the Microscope* is a click-and-go CD. The user can cover as little or as much information as needed. The fifteen programs are well supported by references (both text and websites). The breadth of coverage of each program is outstanding There are approximately 800 full-color microscopic images. The supporting text slides are clearly written and easy to understand.

Each program deals with a different area of clinical pathology, ranging from the selection and care of a microscope through urinalysis, parasitology, mycology, hematology, and avian necropsy. The easy-to-follow introduction offers a discussion of the pros and cons of doing in-house testing vs. sending samples out to a reference lab.

Several programs are dedicated to hematology. One program demonstrates different methods for doing a complete blood count (CBC) using domestic animal blood. A discussion of the proper choice of tubes and anticoagulants, views of collection sites from various animals, information on smear preparation and proper sample storage, and various sources of error that are encountered in routine hematology are presented. Three other programs deal exclusively with reptile, avian, and exotic mammal hematology. Within each program are microscopic images of blood smears from the various species.

An omission in many clinical texts but a strong point in these programs is that, unless otherwise noted, all images are displayed at the same power of total magnification and stained with the same histological stains. Another strong point in these programs is the inclusion of several different cells in one field. In one field, the viewer might see an opossum neutrophil and a lymphocyte together; or an avian basophil, eosinophil, and a monocyte together. The beauty of these images is the ease with which one can make detailed, direct comparisons between sizes of the different cells, their cytoplasmic colors and textures, and nuclear morphologies. These are critical parameters when doing a differential white blood cell count.

Similar presentations are found in the three programs dedicated to parasitology. A basic program introduces parasitology and various methods or techniques used to isolate the ova or cysts of parasites. The other parasitology programs deal specifically with parasites found in reptiles, mammals, and birds. Many images contain more than one diagnostic parasitic form, which enables the viewer to compare relative sizes, shapes, and textures of the different cysts and ova critical for making accurate identifications. In addition to the numerous internal parasites, the external or ectoparasites are well represented. Especially noteworthy in this section is an excellent image of a squirrel with a warble.

Although the sections dealing with cytology and effusions might be beyond what is needed in a rehabilitation setting, these are very strong units for anyone interested in the collection and processing of these samples.

As I browsed through this CD there were only a few changes that I would like to see included, perhaps in the next edition. One change would be either a compiled or searchable index to access a specific image or test more quickly. Another minor change would be the inclusion of more images of reproductive cytology—normal vs. abnormal sperm morphologies, as well as estrous cycle images would enhance the cytology program.

Despite these minor shortcomings, the information contained in this CD is directed toward a wide range of practioners with varying levels of experience. Vets, veterinary technicians, veterinary technology students, and wildlife rehabilitators who have the common interests of clinical laboratory work and a love of wildlife will greatly benefit from the information presented in this CD, as will the animals in their care.

JWR

Reviewer Kathy Dolan is an instructor in the veterinary technology program at Trident Technical College (Moncks Corner, South Carolina). She has been a wildlife rehabilitator, primarily birds of prey, for 10 years.

(This CD is available from Stuart L. Porter, V.D.M., Professor, Veterinary Technology, Blue Ridge Community College, 1 Community College Lane, P.O. Box 80, Weyers Cave, VA 24486, USA; 540/453-2283.)

Wild Health:

How Animals Keep Themselves Well and What We Can Learn from Them

by Cindy Engel

New York: Houghton Mifflin Co.
288 pp; U.S. $24 (hardcover)
ISBN: 0-618-07178-4

At times, rehabilitators receive wild animals that have healed fractures, scars from old wounds, or amputated digits. Until a new book by Cindy Engel, *Wild Health*, there been little published about how wild animals manage these and other conditions in the wild. Dr. Engel,

who has a Ph.D. in animal behavior, offers insights into how wild animals stay healthy in their natural environment – and without rehabilitation. She admits that many scientists previously dismissed the idea of wild animals treating themselves. Rapidly accumulating research by reputable scientists, however, shows that it is no longer acceptable to dismiss this as romantic anthropomorphism. Growing awareness of this subject is similar to acknowledgement by the veterinary community in the late 1980s that animals feel pain.

Dr. Engel provides numerous interesting examples of birds, mammals, herptiles, and insects treating their own health. As well as fascinating information about animals, she offers ideas for further study and suggestions for human health. The following illustrations from *Wild Health* provide food for thought:

- Canadian snow geese often have significant tapeworm burdens in summer. Researchers observed them eating different types of grasses just before migration, and then promptly defecating large piles of the undigested grasses with the tapeworms. Since a heavy parasite infestation could reduce their energy for migration, this de-worming seems to be useful activity.
- Anting (rubbing live ants over their feathers) is a process used by more than 200 bird species. One scientist captured four wild birds in the process of anting and confirmed that the feather mites were dying. The ants preferred in this activity secrete toxic fluids, such as formic acid. In laboratory tests, the formic acid vapor alone has killed lice and feather mites.
- Researchers have observed many bird species select specific herbs and weave the fresh greenery into their nests, especially just before egg hatching. In several studies, young starlings were found to have more ectoparasites after researchers removed the fresh herbs from their nests. Young starlings in nests with the green herbs had fewer mites and higher hemoglobin counts than those with herbs.
- Some wild mammals have been seen licking wounds in the wild (rehabilitators may have also observed this when animals are in rehabilitation). Since saliva of all mammals has disinfectant qualities, this can be very helpful. Studies on rodent saliva have shown that it contains nerve growth and epithelial factors that accelerate healing of wounds.
- Wild bears have been seen rubbing resinous fir trees until they become covered in thick resin. This same resin was used by local indigenous people to repel black flies. When captive bears were offered pungent plants, they rubbed them on their fur – also repelling insects.
- Many wild species have been observed eating soil, a process called geophagy. Certain types of natural clay seem to be especially popular. While they suspected that the clay had some micronutrients the animals needed, researchers learned that many indigenous cultures use similar clay to help bind toxins and reduce gastrointestinal upset. Black or brown topsoil, however, does not seem to be attractive for consumption, possibly since it may be contaminated with parasite eggs, harmful bacteria, heavy metals, and other pollutants.

Wild Health provides an abundance of information, but at times raises more questions than it provides answers. (More research is certainly needed!) Since it takes considerable attention to read, its probably more likely to be enjoyed when the phone is not ringing off the wall with rescuers needing help or baby wild orphans needing to be fed. It is worth reading to expand our thinking about how wild creatures live in the wild—and even about our rehabilitation practices.

JWR

Reviewer Shirley Casey has been a rehabilitator since 1986 and is cofounder of WildAgain Wildlife Rehab in Evergreen, Colorado. She conducts training and publishes on a wide range of wildlife-related topics.

SELECTED ABSTRACTS

AVIAN SPECIES

Effects of Hardwood Reduction on Winter Birds in Northwest Florida Longleaf Pine Sandhill Forests
L. Provencher, N. M. Gobris, and L. A. Brennanc
The Auk 119(1): 71–87.

Reversing decades of fire exclusion by hardwood midstory reduction is now used to recover populations of the federally endangered red-cockaded woodpecker (*Picoides borealis*) in longleaf pine (*Pinus palustris*) forest ecosystems. The effects of red-cockaded woodpecker management on winter birds in longleaf pine sandhill forests are largely unknown. Examining habitat use of winter migrants, some of which are declining, may influence the selection of habitat management techniques used for red-cockaded woodpeckers to benefit overwintering migrants. During December–February of 1997–1998 and 1998–1999, the authors tested experimentally the effects of hardwood reduction treatments applied in 1995 on winter birds at Eglin Air Force Base in fire-excluded northwest Florida longleaf pine sandhills. Treatments were (1) prescribed spring burning, (2) herbicide application, (3) mechanical felling and girdling, and (4) a control where decades of fire exclusion was maintained. The authors also sampled winter bird flocks in frequently burned, nonexperimental reference plots to measure management success. Hardwood reduction techniques had no effect on flock species richness, which averaged 7.9 and 7.2, respectively, during 1997–1998 and 1998–1999. Larger flocks in felling and girdling and in herbicide plots were primarily due to significantly higher numbers of overwintering chipping sparrows (*Spizella passerina*), as well as resident red-cockaded woodpeckers and an influx of temperate migrant pine warblers (*Dendroica pinus*). In contrast, flocks in control plots were smaller and composed of hardwood-associated species, such as tufted titmouse (*Baeolophus bicolor*) and Carolina chickadee (*Poecile carolinensis*). The relative uses of longleaf pines and hardwoods by red-

cockaded woodpeckers, pine warblers, and brown-headed nuthatches (*Sitta pusilla*) during both winters best explained that winter birds present in herbicide, felling and girdling, and reference plots were more likely to forage on the same tree species and substrates than birds in spring-burned plots, and least likely to forage on the same species and substrates as birds in the control plots. Those differences corresponded to the following increasing order of hardwood stem mortality among treatments: control, spring burn (41%), felling and girdling (62%), and herbicide (92%). Repeated burning is recommended to restore the reference foraging condition because it was eight times less expensive than other techniques, which favored mostly chipping sparrows.

Nutritional Consequences of Gastrolith Ingestion in Blue-Winged Teal: A Test of the Hard-Seed-for-Grit Hypothesis

M. H. Sherfy, R. L. Kirkpatrick, and K. E. Webb Jr.
J. Wildlife Mgmt. 65(3): 406–10.

The authors investigated whether young blue-winged teal were able to more efficiently digest available food with grit in their crops. Although the researchers expected to find that their subjects would be more able to derive energy from both pelleted and seed diets if they were provided with grit and/or hard seeds (substitute grit), in fact, they saw no difference in total metabolizable energy between birds with grit and without grit. This might change in wild birds on a varied diet but for the purposes of captive husbandry, providing grit to waterfowl does not seem to enhance digestion.

MAMMALIAN SPECIES

Population Ecology of Swift Foxes (*Vulpes velox*) in Southeastern Colorado

E. R. Schauster, E. M. Gese, and A. M. Kitchen
Can. J. Zool./Rev. Can. Zool. 80(2): 307–19.

Swift foxes (*Vulpes velox*) were once distributed across most of the shortgrass prairie of North America. The arrival of settlers and the concurrent loss of habitat, trapping, incidental poisoning, and shooting brought about a decline in its numbers until the swift fox remained only in the southern part of its historic range. A current understanding of swift fox population ecology is important to management and conservation efforts.

The authors examined the population ecology of swift foxes on a native shortgrass prairie ecosystem in southeastern Colorado. From January 1997–December 1998 we investigated home-range size and space use, spatial overlap, dispersal patterns, survival rates, and reproduction using a sample of 90 (42 males, 48 females) radio-collared foxes. Home ranges were largest (9.4 ± 4.9 km^2) and spatial overlap was greatest using nighttime locations when foxes were active, while daytime locations indicated that the use of dens was exclusive among social units, and space use (2.8 ± 2.2 km^2) was reduced during the day. Among resident foxes, survival rates were higher for adults than for juveniles. For dispersing foxes, adults that dispersed had higher survival rates than juvenile dispersers. Social units ranged in size from 2–4 foxes. About 58% of the social units produced pups; litter size averaged 2.4 pups at den emergence. Among the surviving pup cohort, some pups were philopatric, some dispersed long distances, while others dispersed to neighboring territories. Swift fox density was negatively associated with both coyote (*Canis latrans*) and lagomorph abundance, but positively correlated with rodent abundance. *(Abstract © NRC Research Press)*

Behavior, Movements, and Apparent Survival of Rehabilitated and Free-Ranging Harbor Seal Pups

M. E. Lander et al.
J. Wildlife Mgmt. 66(1): 19–28

The authors compared diving behaviors, activity patterns, movements, and survival between rehabilitated pups (n=29) and wild pups (n=24). Pups were followed for 5 months after release. Rehabilitated pups spent a greater proportion of their time in the water than did wild pups. Survival curves between rehabilitated pups and wild pups were similar after pooling data across 1995 and 1996. The overall behavior, movement and apparent survival of rehabilitated pups appeared similar to wild pups. It was recommended that a longer term study be done since 5 months only covered initial survival.

CONSERVATION

The Effects of Low Levels of Pollutants on the Reproduction of Golden Eagles in Western Norway

T. Nygård and J. O. Gjershaug
Ecotoxicology 10(5): 285–90.

The reproductive success of golden eagles was studied in an area in western Norway between 62° and 63°N between 1973 and 1999. Addled eggs were collected for analysis of chemical pollutants from nine territories; five from coastal areas and four from inland. The coastal sites had lower annual reproductive output than inland sites, and the eggs had a higher content of organochlorine compounds. There were relatively strong negative correlations between reproductive output and (1) shell thickness and (2) DDE concentration in eggs. The data indicate that the golden eagle may be a particularly sensitive species to DDE. It is proposed that the higher organochlorine content found in the eggs of coastal birds was caused by a contribution of marine birds to the diet, as opposed to inland eagles which have a prey basis consisting almost entirely of terrestrial herbivores such as grouse, mountain hare and cervids. The study's data-set on reproductive output is, however, too small to establish a general relationship between DDE contamination and reproductive output in golden eagles. *(Abstract © 2001 Kluwer Academic Publishers.)*

JWR

NEO-REHABILISM...
prehabilitation
back when you used to have a life

New Evidence Snowmobiles Harm Wolves and Elk

A study by Montana State University (MSU) has found that snowmobiling in U.S. national parks can stress wolves and elk so much that it harms their ability to fight off diseases. By measuring the glucocorticoids—stress hormones—in the feces of elk and wolves in Yellowstone, Isle Royale, and Voyageurs national parks, MSU's Scott Creel and his colleagues found that levels of glucocorticoids were higher when snowmobile use was higher. Although these elk and wolf populations remain stable, Creel says the results can be viewed as an early warning signal. "The populations we studied apparently can compensate for the current level of stress, under current ecological conditions," he explains, "but the physiological effect is there to be seen."

Worldwide Extinction Crisis

Nearly a quarter of all kinds of mammals face extinction within 30 years, according to a new United Nations study. The list of the critically endangered include the black rhino, the Siberian tiger, and the Amur leopard of Asia. The report blames human activities, primarily habitat destruction and the introduction of alien species from one part of the world to another. To read the report, visit www.unep.org/GEO/geo3/index.htm.

"Extinct" Bird Discovered

A rare Amazonian bird that was thought to be extinct for 45 years has been re-discovered. Ironically, a road-paving team spotted the golden-crowned manakin in the heart of the rainforest of Brazil. But conservationists say the bird is likely to become extinct soon, for real this time, because of tree-cutting and road-building in the rainforest.

California Otter Population Continues to Decline

According to recent research conducted by the United States Geological Survey (USGS), the population of the threatened California sea otter has declined for the sixth year out of the last seven. Although scientists had expected the species to make a strong comeback since receiving federal protection in 1977, pollution, entanglement in fishing nets, infectious disease, and starvation have claimed many lives. "We are finding a higher percentage of otters dead on the beach every year," Steve Shimek of the Marina, California–based Otter Project told the *San Francisco Chronicle*. "All that adds up to a fairly bleak picture."

Birth Announcement

Two litters of highly endangered Florida panthers were recently born at the Florida Panther National Wildlife Refuge. The seven kittens weighed approximately 1 pound each at birth.

Florida panthers are among the world's most endangered mammals. There are only between 80 and 100 of them remaining in the wild. The biggest threat to panthers is loss of habitat. Another threat is being struck by cars on highways.

Sweet-Smelling Nests Are Best

Scientists at the Centre for Functional Ecology and Evolution in Montpelier, France, have found that birds on the island of Corsica make their own potpourri. The female blue tits gather 10 aromatic plants—including lavender, mint, and curry, which contain chemicals that ward off bacteria, parasites, and insects—to keep their nests clean and pest-free for their young. "They are real botanists," says the Centre's Marcel Lambrechts, "and do a great job of exploiting their environment to protect their chicks."

Squid on the Rise

A recent report in the Australian journal *Australasian Science* found that squid numbers around the globe are increasing at rapid rates and now exceed humans in terms of total bio-mass. The reasons include the climate changes attributed to global warming, as well as overfishing of rival species that compete with squid for food.

The findings may offer an answer to the mysterious appearance of a giant squid on the coast of Tasmania earlier this summer, and hundreds washed ashore in California—although El Nino also is being blamed.

Squid eat anything that comes their way, breed whenever and wherever possible, and keep growing until they die. "Many of the species have exponential growth," noted Dr. George Jackson, of the Institute of Antarctic and Southern Ocean studies in Hobart, Tasmania, Australia.

Request for Assistance

Diane Winn, Ph.D., seeks accounts of experiences with a syndrome in chimney swifts that was first noted by Paul and Georgean Kyle in their *Rehabilitation and Conservation of Chimney Swifts* (Driftwood Wildlife Association, 1996); it was dubbed "mung" and described in more detail by Kathy Frisbie in her article "Hand-Raising Chimney Swifts" (*Wildlife Rehabilitation Today*, Winter 1998, pp. 56–61). The illness appears to begin as a mouth infection with signs such as reluctance to eat, spitting out food, inflammation of mouth mucuous membranes, repeated and seemingly difficult swallowing, and thick, ropy saliva. The infection may progress through the GI tract; untreated birds may lose weight even if force fed, and they often die.

If this syndrome sounds familiar, please send descriptions of its course and treatment in your practice to the address below. Histopathology reports on any birds that died are particularly important. Results will be shared with all contributors and any other interested parties.

Diane Winn
Avian Haven
418 North Palermo Road
Freedom, ME 04941
E-mail: avianhaven@northlandlink.com

JWR

NEO-REHABILISMS...

koikilotherms
animals dependent on carp as a source of heat

refurgitate
the action of a cat with a hairball

DITTO A REPRODUCEABLE PUBLIC SERVICE ANNOUNCEMENT

CATS

keep your cat indoors

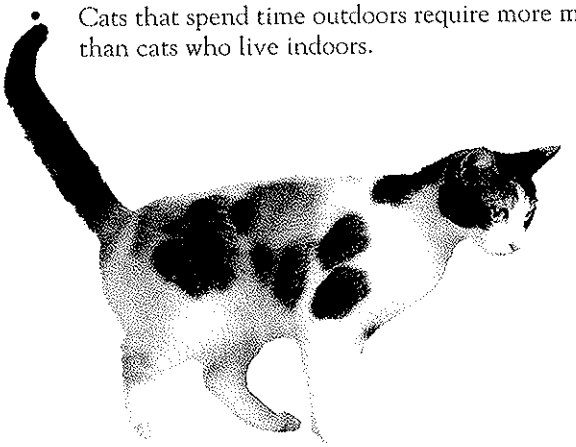


If you love wildlife, keep your cat indoors because:

- Every day, cats kill between 4 and 5 million birds in the U.S. alone.
- Collar bells don't work. Birds and other wildlife do not associate bells with being stalked.
- Ground-nesting birds are very susceptible to predation by cats.
- Even well-fed cats will hunt small wild animals.
- Most young birds leave the nest before they are able to fly well, spending a day or two on the ground as they learn. These fledglings are frequently caught by cats.
- Most of the birds caught by cats, but not killed outright, die of their injuries or infection.
- Cats that kill small rodents can eliminate a critical food source for owls and hawks.

If you love your cat, keep him or her indoors because:

- Cars kill millions of cats each year.
- Outdoor cats are exposed to serious—and often fatal—infectious diseases such as feline leukemia and rabies.
- Parasites such as fleas, ticks, and intestinal worms post a health threat to your cat. Some of these parasites can be transmitted to humans.
- Outdoors, cats can be chased by dogs or other cats and killed, injured, or become lost.
- Cats are often shot at, poisoned, trapped, or tortured by neighbors who are annoyed by cats using their gardens as a litterbox or hunting ground.
- Coyotes, great-horned owls, and other wild animals are known to regularly kill and eat house cats.
- Cats that spend time outdoors require more medical treatment and their life-span is much shorter than cats who live indoors.



(This space for your organization's name, address, website, and phone number.)

Ditto content provided by Lindsay Wildlife Museum, Walnut Creek, California

TAIL END

by Astrid MacLeod



Blue Gold Macaw (*Ara ararauna*)

"Look, everybody—Bob's back!"

INSTRUCTIONS FOR AUTHORS

POLICY Original manuscripts concerning any aspect of wildlife rehabilitation are welcome. Articles on topics including administration, law, education, veterinary medicine, animal husbandry, and public relations as they relate to wildlife rehabilitation are all invited. Book reviews are also welcome. Submissions are not confined to the continent of North America.

Associate editors and anonymous reviewers judge each submitted manuscript on appropriate subject matter, originality, accuracy, conciseness, clarity, and contribution to existing literature. Concurrent submission to other peer-reviewed journals may preclude publication in the *Journal of Wildlife Rehabilitation* (JWR). The primary author of each original article published receives either a $30 honorarium or a one-year individual membership or renewal. IWRC retains copyright on all original articles published in JWR, but will grant permission to reprint articles upon request, with credit given to IWRC.

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TABLES Start each table on a separate page and continue long tables onto 2 or more pages. Double-space throughout and leave margins of at least 1 inch. Annotate abbreviations.

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