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Treatment of capture myopathy in shorebirds: a successful trial in northwestern Australia

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ABSTRACT. Shorebirds held during banding activities can develop muscle cramps, especially when temperatures are high and birds are heavy. Such capture myopathy can be fatal or render birds vulnerable to predators. We rehabilitated Great Knots (*Calidris tenuirostris*), Red Knots (*C. canutus*), Bar-tailed Godwits (*Limosa lapponica*), and Red-necked Stints (*C. ruficollis*) in northwestern Australia. We kept birds in slings (if cramped) or in a small cage (if able to walk) and gave them daily standing exercises. Recovery of severely cramped birds took up to 14 d, which may reflect a critical period of tissue regeneration. Of 15 knots (8 Red and 7 Great) taken into captivity, 12 were rehabilitated and released. The resighting rate after the breeding season of the rehabilitated birds was the same as for other birds color-banded during our research, indicating that the rehabilitation was successful. We conclude that rehabilitating cramped shorebirds is possible though time-consuming. A sex bias in susceptibility to capture myopathy is suggested by seven of the eight Red Knots treated being male; the sex ratio in the local population was 1:1.

SINOPSIS. **Tratamiento de miopatía en playeros: casos exitosos en la parte noroeste de Australia**
Los playeros atrapados durante actividades de anillamiento pueden desarrollar calambres o miopatía muscular particularmente cuando las temperaturas son altas y las aves son pesadas. La miopatía puede ser fatal o dejar a las aves sumamente vulnerables a depredadores. Recientemente, rehabilitamos individuos de *Calidris tenuirostris*, *C. canutus*, *C. ruficollis* y *Limosa lapponica* en trabajos llevándose a cabo en la parte noroeste de Australia. Mantuvimos a las aves en cabestrillos si estaban acalambradas o en una pequeña jaula, (si no podían caminar) y los sometimos a un régimen diario de ejercicios. La recuperación de individuos con miopatía severa, tomó hasta 14 dias, lo que pudiera reflejar un periodo crítico para la regeneración de tejidos. De un total de 15 playeros (ocho *C. canutus* y siete *C. tenuirostris*) que fueron llevados a cautiverio 12 se rehabilitaron y fueron liberados. La tasa de reavistamiento de estas aves después de la época de reproducción fue la misma que para otros playeros anillados durante el periodo de investigación, lo que indica que la rehabilitación fue exitosa. Concluimos que la rehabilitación de aves con miopatía es posible, aunque consume mucho tiempo. Se sugiere una suceptibilidad mayor (a la miopatía) en los machos por la cantidad de capturados (siete de los ocho individuos capturados de *C. canutus*), cuando la proporción de hembras a machos en la población silvestre es de 1:1.

Key words: capture myopathy, migration, rehabilitation, shorebirds

Capture myopathy is a state of muscle tissue degradation that can result in cramped legs and wings, rendering a bird incapable of standing, walking, or flying. The condition is dangerous and potentially lethal. Shorebirds can develop these symptoms during catching operations, and although the condition is generally uncommon or rare, it occurs frequently enough to have prompted several reports on ways to alleviate the problem (Minton 1993; Taylor 1994; Clark and Clark 2002). Since 1998 we have rehabilitated a small number of shorebirds that suffered capture myopathy during cannon-netting in Northwestern Australia. Although apparently successful, the long-term fate of the birds was not known. In 2000, as part of another research project (Rogers et al. 2001), we rehabilitated several Great Knots (*Calidris te-*

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nuirostris) and Red Knots (*Calidris canutus.*) Because most of these birds were individually color-banded, we could assess their post-release survival and more adequately evaluate the success of our rehabilitation efforts. Here we describe and discuss the methods used and report on the subsequent survival of treated birds.

At the outset we should stress that our approach is a last resort. Our research involved intensive handling of many knots in hot conditions, as we attached radio-transmitters and took detailed information on them. Aware of the potential for capture myopathy, we were prepared to put a major effort into rehabilitation. We treated seven of 98 Great Knots (7.1%) and eight of 47 Red Knots (16%) captured. Such high incidences of capture myopathy would be unacceptable in ordinary banding operations. The best way to deal with capture myopathy is to organize trapping operations in such a way that animals do not develop it in the first place (Minton 1993; Williams and Thorne 1996; Clark and Clark 2002).

BACKGROUND ON CAPTURE MYOPATHY

Capture myopathy is not unique to shorebirds and has been studied thoroughly in other groups of birds and mammals (Williams and Thorne 1996). The physiological basis appears to be altered blood flow to the tissues and exhaustion of normal aerobic energy (Beringer et al. 1996; Harthoorn 1983). These effects are largest in the skeletal muscles, and cause decreased delivery of oxygen and nutrients, increased production of lactic acid, and inadequate removal of cellular waste products. Damage to muscle tissues follows, sometimes relatively slight but often severe enough to cause extensive necrosis of muscle tissue. Resultant pallor of skeletal muscles is typical, and the extent of tissue necrosis is closely correlated with the severity of capture myopathy (Shepherd et al. 1988). Necrosis of skeletal muscle tissue can render animals incapable of locomotion (Spraker et al. 1987; Beringer et al. 1996); furthermore, the myoglobin released from dead muscle cells can cause fatal renal failure (Harthoorn 1983). Necrosis of cardiac tissue can also occur, so subsequent death through heart failure can result even if the animal survives and is released in apparently good health (Williams and Thorne 1996; Nicholson et al. 2000).

The risk of capture myopathy is increased by several side effects of capture: intense exertion in restrained conditions (e.g., Harthoorn and van der Walt 1974), cramped postures (e.g., Dabbert and Powell 1993), and heat stress (Chalmers and Barrett 1982; Williams and Thorne 1996; Nicholson et al. 2000). These all increase the likelihood of switching to anerobic respiration in some muscles. Workers comparing different capture methods in ungulates (Harthoorn 1983; Beringer et al. 1996; Hamilton et al. 1996) and kangaroos (Shepherd 1983; Coulson 1996) agree that the safest techniques are those that minimize the amount of time a captured animal spends struggling before it is released, anesthetised, or put in a dark, quiet place. Administration of a balanced electrolyte solution and sodium bicarbonate to reverse metabolic acidosis has proved helpful in the treatment of capture myopathy in ungulates (Harthoorn 1983; Williams and Thorne 1996). This treatment does not appear to have been tested in birds and would require expert veterinary supervision.

The muscle necrosis, clinical signs, and apparent causes of capture myopathy in shorebirds (Minton 1993; Taylor 1994; P. McWhirter, pers. comm.) seem similar to those reported in other animals in the broader veterinary literature. Levels of creatine kinase (which probably reflect muscle damage) of shorebirds during handling far exceed those incurred by migratory flight (Guglielmo et al. 2001). Three contributory factors may be of particular importance in shorebirds. First, they are gregarious and captures often involve many birds at one time. Second, heat stress greatly increases the susceptibility of shorebirds to capture myopathy (Clark and Clark 2002), and there are two reports of wild shorebirds developing clinical signs identical to capture myopathy on very hot days (Clark and Clark 2002; J. Schoenjahn, pers. comm.). Finally, condition at capture may be important; shorebirds carrying large fat loads just before migratory departure are especially prone to capture myopathy (Minton 1993; J. Clark pers. comm.).

METHODS AND GENERAL COURSE OF TREATMENT

From February to April 2000 we were involved in a research program (“Tracking 2000”)

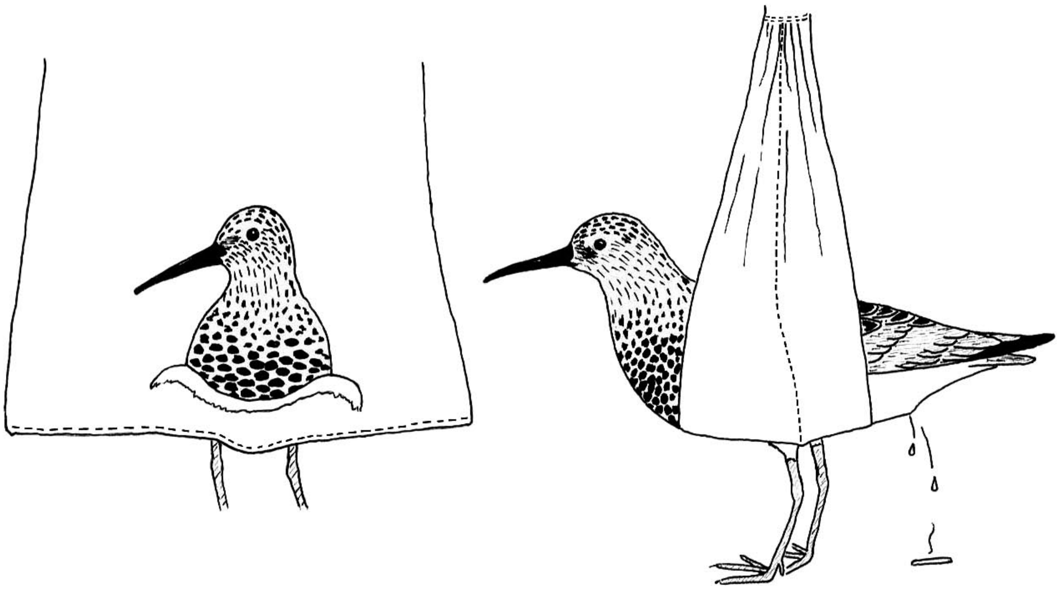

Fig. 1. Great Knot in a sling to treat capture myopathy. The sling should enable birds to take the weight off their legs if they so desire; the tail, wing tips and cloaca should all protrude through the tail-hole to prevent the bird from flapping or fouling its sling.

in Roebuck Bay (northwestern Australia) that involved several cannon-net catches of Great Knots and Red Knots during the pre-migratory period (Rogers et al. 2001). At this time of year adult knots were accumulating large fat deposits in preparation for northward migration. Although our field period in the late wet season was unusually cool and rainy by local standards, it was still warm and extremely humid; daytime highs were usually above 30°C. We made cannon-net catches on 26 and 28 February, and on 3, 4, 19 and 28 March. One to three birds needed rehabilitation after each of these catches because they were unable to walk or fly.

Birds were kept in an air-conditioned wetlab, in either a cage or in slings. The cage was 1.54 m long, 0.55 m wide and 0.7 m high, wooden framed with a covering of plastic shade-cloth. A large plastic tray of water in the cage was changed regularly through easily closed slits in the shade-cloth that also enabled us to reach into the cage when we needed to handle the birds. Birds frequently walked in the water tray; as it was somewhat slippery we put sand at the bottom to provide them with firmer grip. The main cage had a cardboard and newspaper floor which was replaced daily to prevent foot infections. A maximum of six birds was kept in the cage. They showed no signs of aggression to one another and if anything seemed more relaxed in the company of other shorebirds.

Some birds could walk a little from the outset and were placed in the main cage immediately. Birds that were unable to stand were initially suspended in slings (Fig. 1), following Green (1978). The slings were bird-bags modified by the addition of two leg-holes, a neckhole and a hole for the tail and cloaca. The bottom seam of the bird-bag was laid perpendicular to the belly of the bird, so that the sides of the bird were not in direct contact with the sling; this allowed air to circulate in the sling and prevented the bird from becoming too hot. The tips of the folded wings were also pushed through the tail-hole, preventing the bird from flapping or abrading its wing-tips. The slings were suspended from a rod, and the height of the sling adjusted to enable the bird to extend its legs and touch the ground, yet rest its weight in the sling. A water bowl was placed in front of the bird and could be reached by the bird. Food placed in front of each bird was invariably ignored. Each sling was placed in a separate cardboard box, which was open at the top.

Once birds began to improve they were moved into the main cage.

Neither species of knot fed itself in the early stages of rehabilitation (and Great Knots are particularly slow to learn to feed in captivity; A. Dekinga and T. Piersma, pers. comm.), so it was necessary to hand-feed the birds. We used soaked cat food ("Go-cat Friskies": 26% crude protein, 10% crude fat, 3% fibre, 1.2 % sodium chloride), a readily available low-fiber food previously tested and found adequate for Great Knots at Roebuck Bay (D. Rogers et al., unpubl. data) and similar in broad composition to the Trouvit pellets used to maintain a captive population of Red Knots at The Royal Netherlands Institute of Sea Research (T. Piersma, pers. comm.). Pellets were usually swallowed if placed in the base of the bill (or pushed to the back of the mouth with a wet cotton wool bud). Some birds took to eating the softened cat or kitten pellets directly from a fingertip. We also "drip-fed" knots with water or a mild saline solution from a syringe or pipette. In birds that were initially reluctant to drink, it helped to place water drops onto the nostrils; these seeped through into the mouth and were invariably swallowed. We fed and watered each bird at least twice a day; it took about 15 min to feed each bird. When time permitted we weighed each bird (to the nearest gram using a 300-g Pesola balance) once a day before a feeding session.

Exercising the birds was a crucial part of the rehabilitation. With the most seriously affected birds, we began with "balancing exercises" after they had been hand-fed but before they were returned to their slings. We supported the weight of the bird from underneath, letting it balance on straight legs until it was standing, then gradually let the bird take its own weight. Progressively, the bird would increase its ability to support itself. This would be done for up to an hour in a session. Sometimes the first indication that a bird might walk again came through this activity. We also lightly stretched the legs in an attempt to help the bird regain the ability to hold its leg in a natural position.

Preening and bathing are important activities for shorebirds, but they cannot do either while in a sling. Periodically taking them out of the sling and giving them access to a tray of water always resulted in energetic bathing. If the bird had reasonable mobility, even on its haunches, then bathing and preening could be done successfully and safely. Once birds had recovered to the point that they could stand by themselves (albeit unsteadily) they were moved to the main cage and voluntarily spent a good deal of their time preening and bathing. When given the opportunity to bathe, birds also flapped their wings vigorously and lifted their bodies off the ground. This enabled them to get onto their legs properly, and proved to be the turning point for some of them.

Birds were released on a nearby beach when they had regained the ability to walk steadily and flap their wings effectively. We allowed them to bathe and dry naturally and preen before release. Birds were transported in a cardboard box and released by slowly upending the box, so that they could walk out onto the sand with a view of the water or mudflats. When possible we released them on ebbing low tides, so that they could immediately begin feeding on exposed mudflats, a time when raptors seldom hunt along the shores of Roebuck Bay. We always checked that released birds were capable of flight before we left them in the field, flushing them if necessary. When possible we tried to release more than one at a time.

During Tracking 2000, we placed a unique combination of three color-bands on 70 Great Knots and 49 Red Knots. These color-banded birds included all seven rehabilitated Red Knots and one of the five rehabilitated Great Knots. Daily searches for color-banded birds were made in Roebuck Bay throughout March and April 2000, and less frequent, often opportunistic, searches have been made since then. Blood samples were taken from all color-banded birds and used for subsequent genetic sexing (Baker et al. 1999).

RESULTS

We rehabilitated 12 of 15 knots treated during Tracking 2000 (Table 1). One Great Knot and one Red Knot were very badly affected, showed no signs of improvement, and were euthanazised; another Great Knot died after only one day in captivity, possibly due to causes other than capture myopathy.

Five of the rehabilitated Great Knots had an average (±SE) mass on capture of 208.4 (±14.0) g (range 196–230 g); two lighter birds weighed 132 and 140 g. The average non-

Table 1. Outcomes of rehabilitation attempts performed on shorebirds suffering capture myopathy in Roebuck Bay, Australia. Duration of rehabilitation is given as average number of days (± SD). Scientific names are given in the text.

Species	Year	Number cared for	Number rehabilitated and released	Duration of successful rehabilitations (days)
Great Knot	2000	7	5	8 ± (2.1), *N* = 5, range 6–11
Red Knot	2000	8	7	11 ± (2.3), *N* = 7, range 8–14
Bar-tailed Godwit	1998	3	2	
Bar-tailed Godwit	2000	2	1	
Bar-tailed Godwit	2001	1	0	
Great Knot	1998	1	1	
Great Knot	2001	2	2	
Red Knot	2001	2	2	
Red-necked Stint	1998	1	1	

breeding (pre-migratory) mass of Great Knots in northwestern Australia is about 140 g (Barter and Minton 1998), and the average departure mass is about 240–260 g (Battley et al. 2000), so it is apparent that most birds had undergone pre-migratory mass gain. The seven Red Knots weighed 109.3 (±8.67) g (range 101–127). They had undergone little or no pre-migratory mass gain; in northwestern Australia, pre-migratory mass of Red Knots is about 110 g and departure mass is about 160 g (Piersma et al., 2004).

The genetic sexing results showed an unexpected tendency for male Red Knots to predominate among the treated birds: seven of eight were males, compared with 21 males and 21 females among the healthy birds. Of the seven Great Knots, three were male, one was female, and three birds were not sexed. The sex ratio of healthy Great Knots was 50 males: 41 females.

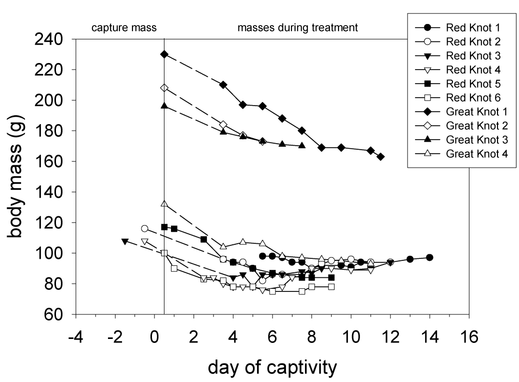

Fig. 2. Mass loss for Red Knots and Great Knots held in captivity in 2000. Because of other activities, most birds were not weighed regularly in the early parts of their rehabilitation. "Half-days" represent afternoon weighings.

The duration of rehabilitation varied from 6 to 14 d (Table 1), and recovery was often sudden rather than gradual. For example, a heavy Great Knot hospitalized on 28 March had lost almost all use of its legs and showed no sign of improvement over the first 7 d in captivity. On 4 April it managed to stand up (with assistance), so it was placed in the larger cage, where it repeatedly managed to raise itself to its feet. By the next morning it was walking strongly, and it was released that afternoon.

All birds lost mass during their stay in captivity, despite our attempts to feed them (Fig. 2). After an initial drop in mass, some birds did level off and maintain body mass or even recover slightly. Birds with a low initial mass may approach critically low body mass levels before release; for example, a second-year Great Knot (#3 in Fig. 2) weighed 132 g when caught and had dropped to 93 g when released. Because it was so light, this bird was released almost immediately upon gaining use of its legs.

Of the eight rehabilitated knots released with color-bands (one Great and seven Red), all but one (a Red Knot) were resighted 1–3 times in Roebuck Bay during April up to 10 km from the release site. Their tide-related movements appeared similar to those of radio-tracked birds that had not experienced capture myopathy. In all cases, the rehabilitated birds accompanied flocks of conspecifics that included unbanded knots. We could not see any differences between rehabilitated birds and unbanded birds in terms of appearance or behavior, and were generally unaware that we had seen rehabilitated birds until we returned from the field and could check the color-banding paperwork.

Color-band resightings showed that two rehabilitated birds did not migrate: a Great Knot resighted in Roebuck Bay on 10 July 2000 and a Red Knot resighted on 4 August 2000. The Red Knot was a first-year bird and was not expected to migrate (Red Knots in northwestern Australia typically skip their first northward migration and remain on the non-breeding grounds). The Great Knot was an adult with extensive breeding plumage that should have migrated. In contrast, there were no austral winter resightings of the six color-banded, rehabilitated adult Red Knots. Although we do not have conclusive proof, we suspect that these Red Knots were not found in the austral winter because they had migrated north with healthy adults.

In the subsequent austral summer, after adult knots had returned from a breeding attempt in Siberia, we resighted the single color-banded rehabilitated Great Knot and three of the seven color-banded rehabilitated Red Knots. By comparison, the resighting rates for birds that did not suffer capture myopathy were 52% in Great Knots (36 of 69 birds color-banded) and 52% in Red Knots (22 of 42 birds color-banded). The overall resighting rate for rehabilitated birds of 50% corresponds well with the resighting rates of color-banded birds that had not suffered capture myopathy.

DISCUSSION

We consider the capture myopathy treatment used on knots to have been successful. The rehabilitation rate of 80% was good, given that the condition is often fatal and the prognosis for animals displaying clinical signs of capture myopathy is generally regarded as poor (Williams and Thorne 1996). The resightings of color-banded birds indicated that their post-release survival rates were similar to those of color-banded birds that had not suffered capture myopathy.

Rehabilitations involved simple animal husbandry, and the core requirement was a sufficient labor force to carry out the work required. While our sample is too small to know whether different species respond equally well to captive rehabilitation, the fact that we have rehabilitated shorebirds ranging in size from a Red-necked Stint (*Calidris ruficollis*) to a Bar-tailed Godwit (*Limosa lapponica*) suggests that the techniques may prove successful with other species.

We had a quiet, shady, and well-ventilated room for hospitalization; air-conditioning was available, reducing the risk of heat stress contributing to capture myopathy. In field circumstances where such facilities are not available, it helps to treat incipient cramp in shorebirds by bathing the legs in cold water (Clark and Clark 2002; J. Schoenjahn, pers. comm.), presumably because this lowers body temperature. Rehabilitators usually spent several hours a day working quietly in the same room as the hospitalized birds. This may have been beneficial by helping to habituate the birds to humans and so reducing their stress levels when they had to be hand-fed and exercised.

Slings proved crucial for some birds. Our original intention was to give them the opportunity to relax their leg muscles without sitting down. We reasoned that such cramped postures, seldom used by wild shorebirds, would probably restrict blood-flow to the legs and could therefore increase the likelihood of anaerobic respiration. In practice, another advantage was that the slings restrained the movement of birds before they were habituated to humans. Such exertion could potentially have caused anaerobic respiration and thus prolonged the period in which cell necrosis occurred. In general, birds were relaxed in the slings and did not attempt to flap. However, monitoring was needed to ensure that they at least sometimes attempted to stand.

Although the slings were important in the early stages of recovery of badly affected birds, none recovered sufficiently for release before we took them out of their slings and placed them in the main cage. Some exercise seems to be essential for knots to recover from capture myopathy, even if it only involves splashing around in a tray of water. Perhaps the birds simply need to exercise muscles that have not been used for several days, or perhaps moderate exercise encourages blood circulation which helps clear away waste products from necrosis that remain in the muscle tissues. The freedom to preen might also be important; plumage became scruffy after long periods in the slings, a problem that birds readily attended to themselves when given the opportunity. Whatever the exact cause, we are certain that an unrestrained period in a cage before release, with access to clean water for bathing, is beneficial; the most dramatic improvements always occurred after birds had been moved from slings into the main cage.

Of concern, even in successfully rehabilitated birds, is mass loss while in captivity. Most of the Great Knots we rehabilitated had undergone some pre-migratory mass gain before capture and therefore had substantial stores to fall back on. While this meant that they could be rehabilitated for long periods without starving, mass loss of the magnitude observed could easily prevent birds gaining the mass required for migration, thus forcing them to skip a migration; this probably happened to one of our rehabilitated Great Knots. In contrast, the rehabilitated Red Knots probably migrated. Great Knots typically migrate north from Roebuck Bay in late March and early April (Battley et al., 2004), but Red Knots typically migrate later, many birds not departing until early May (D. Rogers et al., unpubl. data). Our shorebird catches occurred in late February and March, so it is possible that rehabilitated Red Knots had time to catch up to normal mass schedules whereas Great Knots did not. Lean birds with capture myopathy could starve if they underwent excessive mass loss in captivity. Workers rehabilitating shorebirds should monitor mass changes carefully, increasing the amount of hand-feeding if necessary. Administering a saturated glucose solution may help maintain mass of birds reluctant to be hand-fed (Taylor 1994).

The duration we kept birds may seem extraordinary to banders accustomed to releasing birds as quickly as possible. Some birds were kept for two weeks before they showed any striking improvement, a large commitment for a banding team since looking after four birds can take a single person 2–3 h per day. However, we believe patience was the key and that even longer periods of rehabilitation may be justified in some circumstances. Studies in quokkas (*Setonyx brachyurus*) and kangaroos have demonstrated that muscle tissue killed by capture myopathy will regenerate, but that the process can take 5–8 wk (Kakulas 1966; Shepherd 1983). A similar process may have been occurring in the shorebirds we rehabilitated. In general, we believe the key to rehabilitating birds with capture myopathy is to provide them with an environment in which myopathy is arrested, and to keep them there long enough for muscle regeneration to occur.

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REFERENCES

Baker, A. J., T. Piersma, and A. D. Greenslade. 1999. Molecular vs. phenotypic sexing in Red Knots. Condor 101: 887–893.

Barter, M. A., and C. Minton. 1998. Can pre-migratory weight gain be used to predict departure weights of individual waders from north-Western Australia? Stilt 32: 5–15.

Battley, P. F., T. Piersma, M. W. Dietz, S. Tang, A. Dekinga, and K. Hulsman. 2000. Empirical evidence for differential organ reduction during trans-oceanic flight. Proceedings of the Royal Society of London B 267: 191–195.

———, ———, D. I. Rogers, A. Dekinga, B. Spaans, and J. van Gils. 2004. Do body condition and plumage explain departure date of individual Great Knots (*Calidris tenuirostris*) from north-west Australia? Ibis 146: 46–60.

Beringer, J., L. P. Hansen, W. Wilding, J. Fischer, and S. L. Sheriff. 1996. Factors affecting capture myopathy in white-tailed deer. Journal of Wildlife Management 60: 373–380.

Chalmers, G. A., and M. W. Barrett. 1982. Capture myopathy. In: Noninfectious diseases of wildlife (G. L. Hoff, and J. W. Davis, eds.), pp. 84–94. Iowa State University Press, Ames, IA.

Clark, J., and N. Clark. 2002. Cramp in captured waders: suggestions for new operating procedures in hot conditions and a possible field treatment. Wader Study Group Bulletin 98: 49.

Coulson, G. 1996. A safe and selective draw-string trap to capture kangaroos moving under fences. Wildlife Research 23: 621–627.

DABBERT, C. B., AND K. C. POWELL. 1993. Serum enzymes as indicators of capture myopathy in Mallards (*Anas platyrhynchus*). Journal of Wildlife Diseases 29: 304–309.

GREEN, G. H. 1978. Leg paralysis in captured waders. Wader Study Group Bulletin 24: 24.

GUGLIELMO, C. G., T. PIERSMA, AND T. D. WILLIAMS. 2001. A sport-physiological perspective on bird migration: evidence for flight-induced muscle damage. Journal of Experimental Biology 204: 2683–2690.

HAMILTON, S. M., W. E. PORTER, AND B. A. RUDOLPH. 1996. Evaluating four methods to capture white-tailed deer. Wildlife Society Bulletin 29: 255–264.

HARTHOORN, A. M. 1983. Mechanism and prevention of capture myopathy. In: Wildlife diseases of the Pacific Basin and other countries: proceedings of the 4th International Conference of the Wildlife Disease Association (M. E. Fowler, ed.), pp. 248–252. Wildlife Disease Association, Ames, IA.

———, AND K. VAN DER WALT. 1974. Physiological aspects of forced exercise in wild ungulates with special reference to overstraining disease. Journal of the South African Wildlife Management Association 4: 25–28.

KAKULAS, B. A. 1966. Regeneration of skeletal muscle in the Rottnest quokka. Australian Journal of Experimental Biology and Medical Science 44: 673–688.

MINTON, C. D. T. 1993. Stress myopathy in captured waders. Wader Study Group Bulletin 70: 49–50.

NICHOLSON, D. S., R. L. LOCHMILLER, M. D. STEWART, R. E. MASTERS, AND D. M. LESLIE. 2000. Risk factors associated with capture-related death in eastern Wild Turkey hens. Journal of Wildlife Diseases 36: 308–315.

PIERSMA, T., D. I. ROGERS, P. M. GONZALEZ, L. ZWARTS, L. J. NILES, I. DE LIMA, S. DO NASCIMENTO, C. D. T. MINTON, AND A. J. BAKER. 2004. Fuel storage rates before northward flights in Red Knots worldwide: facing the severest ecological constraint in tropical intertidal environments. In: Birds of two worlds: the ecology and evolution of migratory birds. R. Greenberg, and P. P. Marra, eds., Johns Hopkins University Press, Baltimore, MD.

ROGERS, D., P. BATTLEY, AND T. PIERSMA. 2001. Radiotracking in the tropics. Wingspan 11: 16–20.

SHEPHERD, N. C. 1983. Capture myopathy in the Red Kangaroo (*Macropus rufus*). In: Wildlife diseases of the Pacific Basin and other countries: proceedings of the 4th International Conference of the Wildlife Disease Association (M. E. Fowler, ed.), pp. 239–247. Wildlife Disease Association, Ames, IA.

———, P. R. HOPWOOD, AND P. L. DOSTINE. 1988. Capture myopathy: two techniques for estimating its prevalence and severity in Red Kangaroos, *Macropus rufa*. Australian Wildlife Research 15: 83–90.

SPRAKER, T. R., W. J. ADRIAN, AND W. R. LANCE. 1987. Capture myopathy in Wild Turkeys (*Meleagris gallopavo*) following trapping, handling, and transportation in Colorado. Journal of Wildlife Diseases 23: 447–453.

TAYLOR, N. 1994. A technique for the treatment of capture myopathy. Stilt 25: 33–34.

WILLIAMS, E. S., AND E. T. THORNE. 1996. Exertional myopathy (capture myopathy). In: Non-infectious diseases of wildife (A. Fairbrother, L. N. Locke, and G. L. Hoff, eds.), pp. 181–193. Iowa State University, Ames, IA.