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RAPID ATROPHY AND HYPERTROPHY OF AN AVIAN FLIGHT MUSCLE

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ABSTRACT.—Eared Grebes (*Podiceps nigricollis*) use Mono Lake in eastern California as a rest stop during spring migration. Some nonbreeders remain for the summer, and in the autumn the lake becomes a staging area that may accommodate 750,000 returning breeders and young of the year. There the birds become obese by feeding on invertebrates and, if they have not already done so, molt. Most grebes remain several months until a decline in prey populations stimulates further migration. During this period the birds become flightless, and the flight muscles may lose up to 50% of their mass. Myofibers from atrophic birds show evidence of mitochondrial division (or fusion). Even severely atrophic fibers retain a high mitochondrial density (27% vs. 33% in migratory condition), so that relative volume remains stable although absolute volume is reduced. In contrast, intracellular triglyceride droplets are extremely sparse in atrophic fibers, even though most of the birds are carrying >200 g of subcutaneous fat. Mean myofiber diameter increases and decreases with atrophy and hypertrophy. In late autumn, as food availability declines, the birds engage in conspicuous flapping exercises. In the same period, intracellular lipid reappears in the muscles. Within several weeks the muscles are rebuilt to full size and the grebes emigrate. The benefits, if any, of this cycle of muscle atrophy and concomitant obesity, followed by muscle hypertrophy and weight loss, remain obscure. *Received 18 December 1989, accepted 16 June 1990.*

HIGH-resistance exercise, such as weight lifting, causes hypertrophy of skeletal muscle. Conversely, failure to exercise a muscle group for prolonged periods may lead to atrophy. Disuse atrophy can become debilitating when exercise is difficult or impossible (Booth and Gollnick 1983).

The effect of disuse on skeletal muscles has been investigated with experimental methods that include denervation, tenotomy, limb immobilization, and similar types of neuromuscular alterations (Shear 1981, Hikida and Bock 1972, Silverman et al. 1979, Reiser et al. 1987). Disuse brought on by these regimes generally reduces muscle mass, although other factors also influence muscle fiber size (Booth et al. 1986). Different responses to the various procedures show that disuse atrophy derives from a complex interaction of activity and neural influences.

Because of the variable responses to experimental protocols, a natural system is desired for studying atrophy and hypertrophy. This may be available in birds, in which the mass of the flight muscles may vary with the load. Several migratory species, including Pied-billed Grebes (*Podilymbus podiceps*), some hawks, and several passerines, show seasonal increases in the mass of flight muscles, purportedly in response to the deposition of premigratory fat (e.g. Fry et al. 1970, 1972; Evans and Smith 1975; Marsh 1984). Conversely, in some waterfowl, the flight muscles atrophy during the periods of flightlessness associated with breeding or molting

(e.g. Rosser and George 1987; Piersma 1988; Jehl 1988, 1990). Because both breeding and molt demand energy and protein, it has been supposed that muscular atrophy was a technique for shunting resources to other sites (e.g. Austin and Fredrickson 1987). The hypertrophy of leg muscles of ducks as the breast muscles atrophy is presumably in response to increased swimming and walking (Hanson 1962, Ankney 1979). However, atrophy of breast musculature in Great Crested Grebes (*Podiceps cristatus*) is not associated with the condition of the leg muscles, which remains constant with unchanging activity (Piersma 1988).

A case of atrophy that seems to be free of the complicating effects of energy limitation is found in Eared Grebes (*Podiceps nigricollis*) at Mono Lake, California, where food resources of brine shrimp and brine flies are not a limiting resource (Jehl 1988) until late in the year. Northward migrating grebes arrive at the lake in late March through early May. Some nonbreeding birds remain there until autumn migration (6–8 months). Breeders continue migration to the north-central prairies of the United States and Canada. Because breeding depends on the presence of emergent vegetation (to which nests are attached), the timing and extent of the breeding season varies. Depending on locality and season, young may be fledged between early July and early September. Autumn migration begins after the young have fledged, with the first adults and young arriving at Mono Lake in late July to early August (Jehl 1988).

A few adults molt wing feathers on the breeding grounds, but most do not initiate this molt until after their arrival at Mono Lake. In this molt, shortly after arrival, all flight feathers are lost simultaneously. This renders the birds flightless. Body molt occurs slowly over the next two months. Similar wing and body molts are undertaken by nonbreeders that have remained on the lake, but their wing molt may begin as early as mid-May. Juveniles replace body plumage but not wing feathers in the autumn (Storer and Jehl 1985, Jehl 1988).

The grebes remain at Mono Lake until food supplies fail in late autumn. During this time, they more than double their arrival weights by laying down immense deposits of subcutaneous fat (Jehl 1988). Their flight muscles begin to atrophy within a few days of arrival. Hence, breeders and juveniles may be flightless for 2–4 months, nonbreeders for up to 8 months.

In most years, food supplies fail by early December, and the grebes move to wintering areas farther south. In the fortnight or so before their departure, the grebes metabolize much of the accumulated fat reserves, and their body weights drop from 550–600 g to approximately 400 g. At the same time, exercise flapping of the wings becomes obvious, and the flight muscles hypertrophy and exceed the size found in newly arrived birds in late summer (Jehl 1988). We report here cellular measurements and changes during the atrophy-hypertrophy sequence.

Methods and Materials

Birds were taken throughout the autumn migration period. It was not possible to collect selectively for age, sex, or condition of molt. Most were shot, but a few had recently died of natural causes. Measurements from the naturally deceased specimens were confined to muscle and body mass. All birds were weighed, and the entire breast-muscle mass (pectoralis and supracoracoideus) was removed from one side and weighed. Small strips from various portions of the pectoralis were fixed in a modified Karnovski (paraformaldehyde-glutaraldehyde) fixative buffered with 0.1 M phosphate, pH 7.2, and then stored in phosphate buffer. Tissue for histochemistry was frozen in dry-ice chilled methyl butane and transported in dry ice. Upon arrival in the laboratory in Columbus, Ohio, it was stored in a low-temperature freezer at −70°C. Frozen 10-μm sections were cut in a Reichert cryostat and stained for either myofibrillar ATPase activity after acid (pH 4.35) or alkaline (pH 9.7) preincubation (Brooke and Kaiser 1970) or for NADH-TR activity (Lojda et al. 1979). Histochemical fiber-typing was performed on frozen tissues of the August sample only.

Tissue for electron microscopy was stored in buffer for several days to weeks before being processed in Athens, Ohio. The samples were diced into strips, postfixed in 1% osmium tetroxide in 0.2 M cacodylate buffer, and dehydrated through an ethanol series. After a propylene oxide rinse, tissues were infiltrated with resin and embedded in a mixture of Epon 812 and Araldite. Using diamond knives on a Reichert OMU2 ultramicrotome, we made sections contrasted ("stained") with aqueous uranyl acetate and lead citrate, and examined them with a Zeiss EM 109 electron microscope.

Electron micrographs of the central region of the fibers enlarged to 28,000× were analyzed by measuring the cross-sectional areas occupied by triglyceride and mitochondria within a measured area of the center of the fibers with a Bioquant System IV program (R & M Biometrics, Nashville, Tennessee). Statistical analysis was done with the Bioquant *t*-test analysis or a one-way analysis of variance.

TABLE 1. Correlation matrix of data for adult birds. Each cell of the matrix contains, from top to bottom, the Pearson's correlation coefficient, the calculated probability, and the sample size.

	Date	Body weight	Wet muscle weight	Cell diameter	Volume % mitochondria
Body weight	0.05591 0.7251 42				
Wet muscle weight	0.21833 0.1648 42	−0.41518 0.0063 42			
Cell diameter	0.31970 0.1114 26	−0.49695 0.0098 26	0.64022 0.0004 26		
Volume % mitochondria	−0.02388 0.9118 24	−0.50578 0.0117 24	0.15049 0.4828 24	0.55765 0.0047 24	
% Intracellular lipid	0.45522 0.0254 24	−0.52807 0.0080 24	0.33069 0.1145 24	0.47780 0.0182 24	0.33800 0.1062 24

Semi-thin (0.5 μm) sections were mounted on glass slides, and the cells stained with 1% toluidin blue in 1% borax. We measured cell diameters from tracings made at ×500 magnification with a Wild microscope equipped with a drawing tube. Both the least diameter and cross-sectional areas were measured, the latter with the Bioquant system.

In some birds collected on 1 October 1987, both pectoralis and leg muscles were analyzed to compare the pectoralis with "normal" muscles. Fiber-types were not determined for the leg muscles.

Peterson's correlation coefficients among all the variables were calculated on data from both years integrated by Julian date (Table 1). August and September data were combined in graphic analyses because the samples (especially August) were small, highly variable (as this is the period of arrival for most birds), and statistically similar. These graphic displays (Figs. 1 and 2) suggested a common pattern of change over time for the parameters examined, but because the variances of the samples were high (because of the prolonged arrival time for migrating grebes), we were unable to show statistical significance for the changes. Because the effects of body weight might obscure the pattern (Table 1), we performed analyses of covariance (ANCOVA) in the general linear model (PROC GLM, SAS 1985) with body weight as the independent variable, muscle weight, cell size, and mitochondrial and lipid contents as dependent variables, and dates as treatments. The dependent variables were tested for homogeneity within treatment and analyzed with models incorporating the assumption of homogeneity or heterogeneity as appropriate. ANCOVAs run with three (combined August–September) or four dates did not differ in interpretation. The data (Table 2) are from the four date analyses.

RESULTS

We collected samples on 3 August, 13 September, 14 October, and 28 October (all in 1986), and on 1 October 1987. Muscle and body masses were taken from all birds, but tissue was not preserved from the 14 October 1986 sample. Samples represent birds that had been in residence on the lake for periods that varied from a few hours to several months. Hence, the variation in these samples was high. The 1 October 1987 sample was the most homogeneous. Examination of the molt patterns showed that all birds in this sample had been on the lake for at least 3 weeks, and all were atrophic.

In 1986, the grebes began their southward migration in October, a month earlier than usual, because brine shrimp numbers declined earlier. In contrast, the following year grebes did not leave Mono Lake until late January 1988. The combination of an unusually early migration in 1986 and an unusually late one in 1987 required that direct comparisons be made with caution. Nevertheless, breast-muscle masses from 1987 are similar to those of other, early October samples (Jehl 1988, unpubl.) and are plotted accordingly (Figs. 1 and 2).

Analysis of covariance.—Of the dependent variables in the ANCOVAs, only muscle weight was significantly heterogeneous with respect to treatment and was so analyzed. All dependent variables showed significant change with respect to date (Table 2). Only muscle weight varied significantly with body weight, although

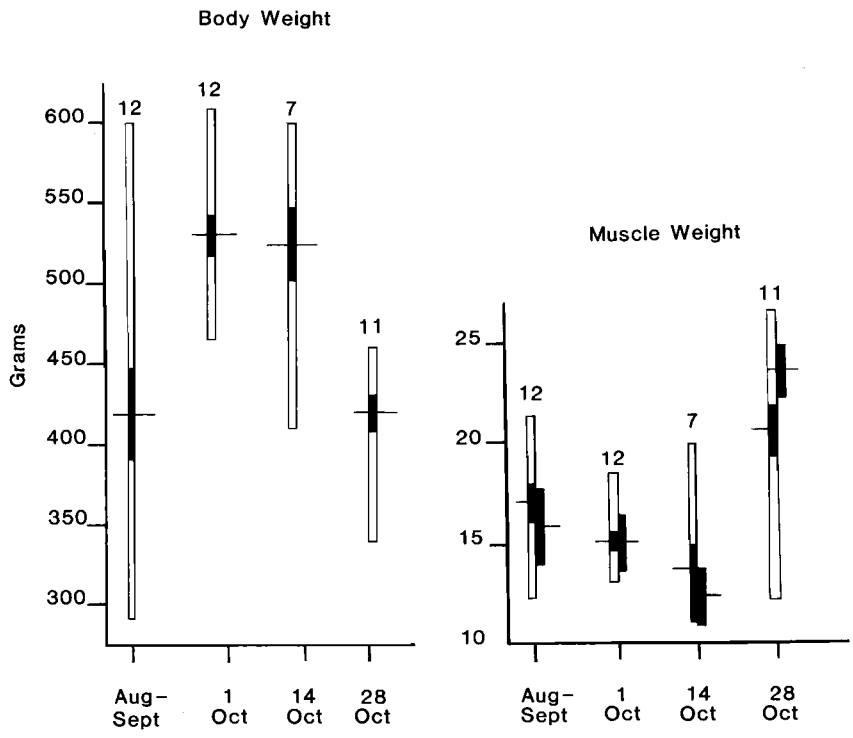

Fig. 1. Body weight and weight of pectoralis and supracoracoideus muscles from one side of adult birds by date. For body weight, vertical bar indicates range, horizontal bar indicates mean, and shaded zone indicates ± 1 SE. For muscle weight, left vertical, left directed horizontal, and shaded zone as for body weight; right directed horizontal and right vertical indicate least squares mean ±1 SE as adjusted by ANCOVA (see text). Number above vertical bar is sample size. Note difference in scale between body and muscle graphs.

lipid content of the cells also approached significance with respect to body weight. Juvenile birds differ considerably from adults in weight throughout the autumn, averaging 20–30% lighter. ANCOVAs including all specimens generally show an increased significance of date and a decreased significance of body weight as an explanation for variation in all dependent variables. Thus, if juveniles are included, body weight erroneously seems not to be a significant factor for explaining variation in intracellular lipid. When we included juvenile birds in the samples of pectoralis mass, the effect of both date and body weight decreased but both remained significant ($P = 0.0235$ and 0.0369, respectively).

Body and muscle weights.—The samples used in our study are too small to reveal significant differences related to sex or age. However, juveniles rarely achieve adult weight during autumn (Jehl 1988), and we present data for adults only. Changes of body weight mainly reflect changes of subcutaneous fat content. The arrival weight of adult grebes is ca. 270–300 g. Subcutaneous fat increases gradually throughout the late summer and autumn. By mid-October, most adult grebes weigh 500–600 g. Before migration, body weight decreases to approximately 400 g (Fig. 1). Through the sampling period, body weight is correlated negatively with measures of flight-muscle weight and cell diameter, percent volume mitochondria, and intracellular lipid (Table 1). The weight of the flight muscles follows an almost opposite pattern (Table 1; Fig. 1). The size of these muscles decreases during the late summer and then increases dramatically just before migration.

Myofiber diameter.—Cell diameter correlated strongly with muscle size (Table 1). Average fiber diameter in September 1986 was smaller than that of the sample taken 6 weeks later. Fiber diameter of the 1987 sample is signifi-

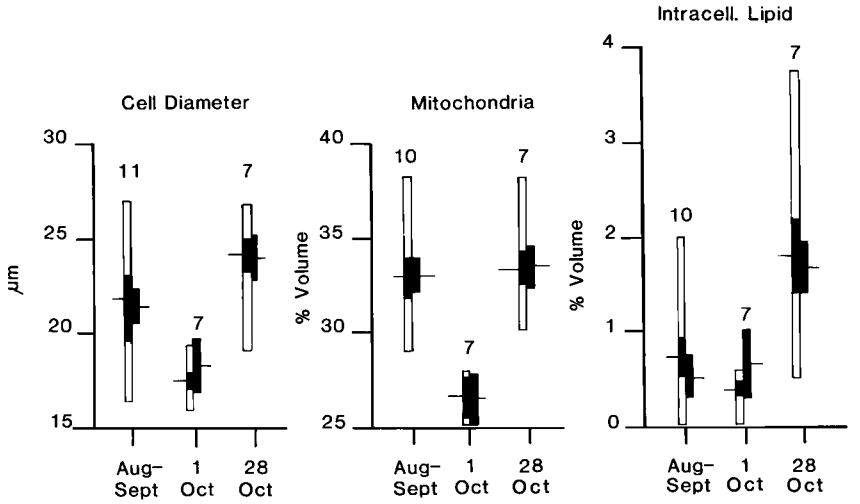

Fig. 2. Measurements from adult grebe pectoralis myofibers by date of sample. Format as for muscle weight in Fig. 1.

cantly smaller than either of the 1986 samples (Fig. 2).

Mitochondrial volume density.—The portion of cellular volume occupied by mitochondria (mitochondrial volume percent) correlated inversely with body mass and positively with cell diameter (Table 1). If we expand the sample to include juvenile birds, then it also correlated marginally with intracellular lipid ($r = 0.36$, $P = 0.049$, $n = 31$).

Mitochondrial volume percents in 1987 samples were significantly lower than in 1986 (Fig. 2). Indeed, the mean for 1987 is lower than the minimum for 1986.

Lipid volume density.—The late October 1986 sample shows a large increase of both range and mean value from the August–September sample (Fig. 2). The minimum value for 28 October 1986 is only slightly lower than the mean for September. Intracellular lipid is positively correlated with other muscle measures, negatively with body mass (i.e. *negatively correlated with subcutaneous fat* [Table 1]). The apparent positive correlation with date is spurious, resulting from the high values of the 28 October 1986 sample.

The 1 October 1987 sample had a limited range of quite low values. Here the maximum value is lower than either the mean for early autumn or the minimum for 28 October 1986.

Ultrastructure.—No pathological lesions were observed in any of the pectoralis samples. Specimens with the smallest muscle fibers had a normal muscle ultrastructure and did not differ from specimens with the largest fibers (Figs. 3–7). Pectoralis muscles were obviously different from leg muscles (Fig. 7). Cross or longitudinal sections of pectoralis myofibers showed many mitochondria that adhered closely to each other, not only in subsarcolemmal regions, but cen-

TABLE 2. Results of ANCOVAs. All but the pectoralis muscle assume homogeneity within treatment (see text). Adult birds only. Levels of significance: * = $0.05 < P > 0.01$; ** = $0.01 > P < 0.001$.

Dependent variable/ effect	df	Type III SS	F	$P \geq F$
Wet muscle weight				
Date	3	116.861	5.27	0.0043**
Weight (date)	4	129.999	4.39	0.0057**
Error	34	251.488		
Cell diameter				
Date	2	76.621	4.10	0.0307*
Weight	1	8.999	0.96	0.3371
Error	22	205.550		
Volume % mitochondria				
Date	2	113.966	7.44	0.0038**
Weight	1	0.625	0.08	0.7781
Error	20	153.233		
Intracellular lipid				
Date	2	5.506	5.19	0.0153*
Weight	1	2.265	4.27	0.0519
Error	20	10.605		

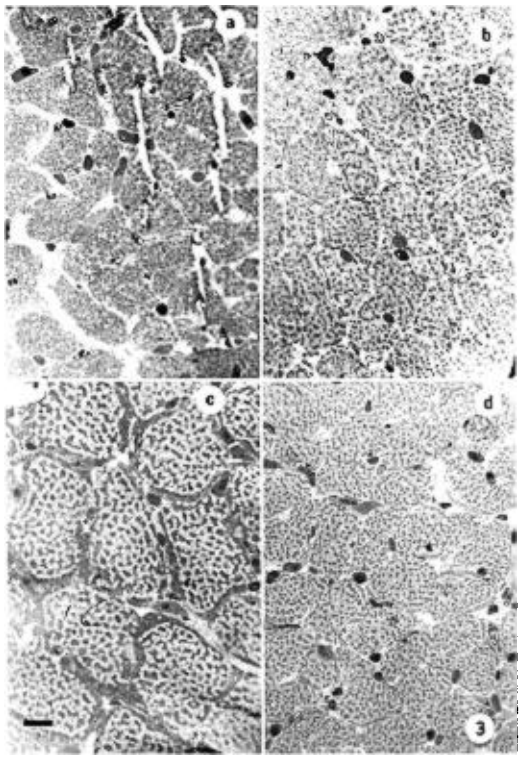

Fig. 3. Photomicrographs of pectoralis myofibers from various birds, all at ×795 (reference bar [in *c*] ≈ 10 μm). Sections cut at 0.5 μm thickness and stained with toluidine blue. (a) Bird I, 14 September 1986, mean fiber diameter 10 μm; (b) Bird M, 14 September 1986, mean fiber diameter 18 μm; (c) Bird H, 14 September 1986, mean fiber size 26 μm; and (d) Bird J, 1 October 1987, mean fiber size 15 μm. Fiber sizes among individuals presumably reflect the conditions in recently arrived (large fibers) vs. resident (small fibers) birds.

trally (Figs. 4–6). These interconnected mitochondria appeared most frequently in the early autumn samples and were less frequent in both October samples. No interconnected mitochondria were observed in leg muscles. Many of the mitochondria from pectoralis muscles had partition figures or, in a few cases, profiles that pinched in at certain points to divide the mitochondrion into unequal segments. We observed neither of these conditions in leg muscles.

The partitioned mitochondria were long and often branched. The partitions consisted of parallel membranes that were denser than the other cristal membranes. Often the parallel membranes were complete, connecting to the outer and inner mitochondrial membranes, producing a figure that appeared as two closely juxtaposed mitochondria. In other profiles, the partitions were incomplete. When mitochondria contained partitions, the cristae within one compartment were arranged at right angles to those of the adjacent compartment.

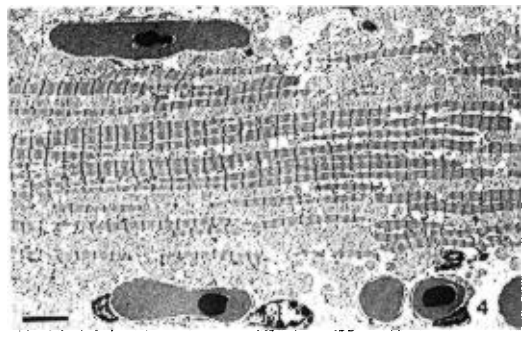

Fig. 4. Low power electron micrograph showing a fiber from Bird 12, 28 October 1986, with a high concentration of mitochondria. Portions of a capillary containing red blood cells are present in the extreme upper and lower portions of the micrograph (×3,850; reference bar ≈ 3 μm).

Histochemistry.—Myosin ATPase activity stains showed the pectoralis to contain a homogeneous population of fast-twitch fibers (Fig. 8). The NADH-TR activity was also uniformly high, which indicated that all cells were oxidative. These results concur with those of Rosser and George (1986) for another species of grebe.

DISCUSSION

Although our data portray the general pattern of events concerning the atrophy and hypertrophy of flight muscles of grebes staying at Mono Lake, it seems prudent to indicate certain events that may have exaggerated some effects. By mid- to late October 1986, brine shrimp numbers dropped to an unexploitable density, which forced birds to anticipate migration. On 14 October, approximately 750,000 birds were present and beginning to exhibit exercise flapping. Breast muscle weight at this time ranged from 11 g to 20 g, with a mean of 13.7 g (Fig. 1). By 28 October, 500,000 birds had departed, and the breast muscles of most of those that remained were in migratory condition, with muscle weights from 12–26 g with a mean of 20.6 g. Indeed, the only bird with flight muscles of <17

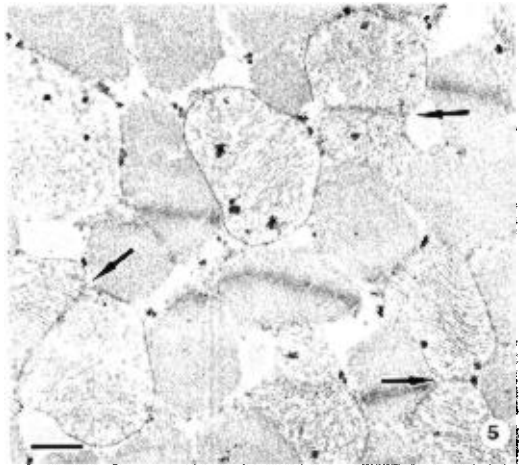

Fig. 5. Mitochondria are closely apposed (arrows), which suggests a stage just before separation of these organelles (from Bird 14, 28 October 1986; ×28,000; reference bar ≈ 0.5 μm).

g also had a remarkably low body mass for that season (340 g vs. mean of 420 g).

The next year, exploitable brine shrimp populations persisted until early January 1988, and few departures occurred before late January. The early October records show a homogeneous population with both body and muscle weights averaging slightly, but not significantly, higher than in mid-October 1986 (Fig. 1). Thus, despite the unusual conditions later in the season, the 1 October birds were of a physiological condition that fit the pattern seen in 1986 and other years (Jehl unpubl.).

Changes in food availability provide the major cues controlling the behavior of grebes at the staging areas. The ability to fatten quickly shortly after arrival may be the cue to molt (Jehl 1988) and to allow breast muscle to atrophy. Conversely, the shortage of food in late autumn is probably the stimulus to begin to resynthesize flight-muscle tissue. The presence of fat reserves per se cannot be the cue to reduce flight readiness, because the maximum reserves are attained long after muscle atrophy has occurred. Nor does the presence of fat reserves stimulate the rebuilding of muscle tissues; the small muscle mass (Fig. 1) and lack of variation in several measures of muscle size in early October (Fig. 2), when the birds were obese (Fig. 1), shows that the birds were unprepared for migration.

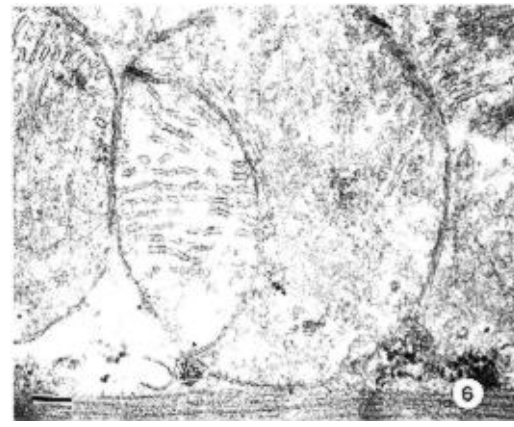

Fig. 6. High magnification of a group of mitochondria, one with a partition figure (from Bird 10, 28 October 1986; ×98,500; reference bar ≈ 0.1 μm).

The *absolute* volume of mitochondria in the pectoral muscles tends to change as the myofiber volume changes. This is reflected in a rather constant volume percentage, regardless of fiber size in most samples. The relationship does not hold for the 1 October 1987 sample, which contained both the smallest fiber size and the lowest *relative* mitochondrial volume percentage of all sampling periods. Thus, at that time the absolute mitochondrial volume had decreased much more drastically than the fiber volume.

The muscles of the 1987 samples were the most atrophied of any examined. The state of

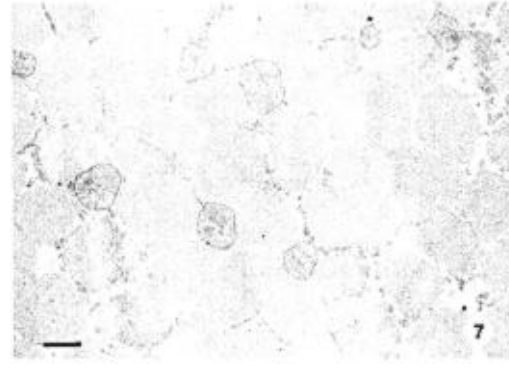

Fig. 7. Normal muscle fiber from a leg muscle of a bird taken 1 October 1987 (×20,000; reference bar ≈ 0.5 μm). The absence of partition figures in the mitochondria of leg muscles indicates that the abnormal mitochondria seen in pectoralis muscles are related to the atrophy-hypertrophy cycle and not an artifact of fixation.

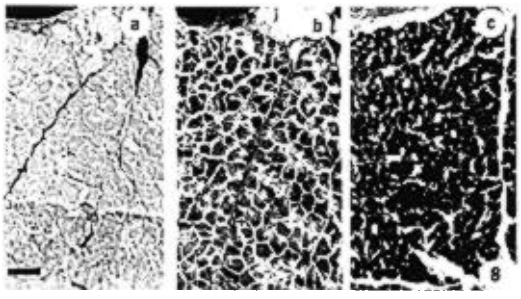

Fig. 8. Serial sections of pectoralis stained for myosin ATPase activity after (a) acid (pH 4.35) and (b) basic (pH 9.7) preincubation, and for (c) NADH-TR activity. The combination of little reaction in acid with intense reactions in basic medium and with NADH-TR is diagnostic of fast-oxidative (IIA) myofibers. Reference bar (in *a*) ≈ 50 μm.

atrophy is correlated with the time until migration was initiated. The 1987 populations had not left the lake by 31 December, whereas the 1986 population migrated by early November. In 1986, premigratory activity began as early as 14 October. Thus, the 1987 preparations were from birds unprepared for flight, whereas those of 28 October 1986 were from fully, or almost fully, recovered birds.

We observed partitioned mitochondria in all breast muscle samples, but not in those of leg muscles. These structures were most common in the September and 1 October 1986 samples, and less common in the 1987 preparations. The presence of partitioned mitochondria in the late October sample is surely associated with mitochondrial proliferation. Mitochondrial partitions are characteristic of mitochondriogenesis in developing or regenerating tissues (Tandler et al. 1969, Tandler and Hoppel 1970, Ghadially 1988). By 28 October 1987, the muscle fibers had returned to their flight size and, correspondingly, by that time mitochondria had achieved their greatest *absolute* volume in preparation for flight.

The presence of partition figures in the September 1986 samples is more puzzling. At that time food was still abundant, and newly arrived birds were apparently becoming atrophic as usual. We postulate that, at least in grebes, the presence of partitioned mitochondria is associated with major changes in the mitochondrial volume, by either growth or reduction. It is tempting to speculate that partitioned mitochondria of the September sample represent a fusion process that permits reduction of mitochondrial numbers with less change in volume.

Both atrophied and normal pectoralis muscles in grebes have a high mitochondrial density. Although some forms of atrophy (e.g. ubiquitin-activated proteolytic activity) may require ATP (Ciechanover et al. 1984), protein production clearly uses ATP in the formation of peptide bonds. Thus, the high volume of mitochondria in small myofibers may anticipate myofibrillar protein synthesis, and this permits a "jump-start" for rapid recrudescence.

The variation in intracellular lipid is higher in the migratory-ready birds of 28 October and considerable in the different samples. Further, our field notes (taken before dissection) indicate that, on the basis of age and molt, we had designated four birds in the September sample as probable recent arrivals. Their muscles have a mean intracellular lipid value of 1.19% vs. a sample mean of 0.68%, and these four values are included in the top five values of the sample. Thus, birds taken just before and just after migration have "fueled motors." Interestingly, the individuals in the 28 October sample with the largest values for intracellular lipid (2.37, 2.51, and 3.77%) have only moderately heavy muscles (18, 18.5, and 19.5 g) and cells of moderately large diameter (24.5, 23.6, and 25.2 μm). Thus, fat appears to be deposited into the myofiber early in recrudescence. It is not known whether the fat is derived from subcutaneous fat stores or is manufactured in situ.

The diameters of myofibers in atrophic birds were only 60% of the diameters of myofibers in the largest, normal flight muscles. Despite the large differences in cell size, the samples did not show obvious abnormal morphology or degenerative changes. The absence of lysosomes or cell fragments indicates that cell death was not a factor in reduced muscle bulk. Therefore, atrophic changes undergone by grebe pectoral muscles differ from those described for Canada Geese (*Branta canadensis*) (George et al. 1987) in which atrophic and degenerative changes of pectoral muscles were induced by both inactivity and starvation. Degenerative changes in geese include tubular aggregates, Z-line streaming, presence of lipofuscin and autophagic vacuoles, and loss of mitochondria. Mono Lake grebes are similar only in the possible loss of mitochondria. In this, they more closely resemble the atrophy in molting Canada Geese described by Rosser and George (1987).

Because the peak tension that a myofiber can develop is directly proportional to its cross-sectional area (e.g. Josephson 1975), smaller cells are direct evidence that atrophied muscles are weaker. Given the increased body size, and consequent wing loading, of the nonmigratory birds, it is scarcely surprising that they show little inclination or ability to fly, even when molt is complete. Similarly, birds that emigrate in autumn migration require larger muscles than the lighter, summer immigrants. The mean body weights of the August–September and 28 October samples are similar, but mean cell diameter is significantly greater in the late October population. Some of this discrepancy may be exaggerated because the early sample contains birds that already reduced their muscles and added body fat. Hence, the sample means were drawn to higher values for body weight and to lower values for cell diameter. Nevertheless, the mean cell diameter in the migratory-ready birds in October is close to the maximum for the earlier sample, and the October maximum is almost double that of the earlier sample.

The recovery process in Eared Grebes does not seem to require prolonged or high-load exercise. Our unquantified observations seem similar to those of Piersma (1988) for Great Crested Grebes in that individuals may engage in wing-flapping for a total of only approximately 5 min (in 3–10 s bursts) in a period of 24 hours.

Whether the recovery process is unusual in either duration or mechanics is unclear. The time for recovery to full capability varies depending on degree of atrophy and fiber-type (Booth and Seider 1979, Witzman et al. 1982). In rats, type I (slow-oxidative) fibers require 14–28 days to reacquire peak tension capabilities after 60 days of immobilization, and far longer times for longer periods of disuse. The contractile properties of type IIB (fast-glycolytic) fibers appear to be little affected by atrophy, and recovery in diameter is rapid. We did not find data on type IIA (fast-oxidative) cells such as occur in grebe pectoralis, but we may reasonably assume a time course intermediate between those of types I and IIB.

Exercise regimes leading to muscular hypertrophy also vary with fiber-type (McDonagh and Davis 1984). Repetitive exercise with high load conditions normal muscles for strength, whereas more repetitions at lower loads promote endurance capability. Fast-twitch fibers respond more rapidly. As muscles recover from atrophy, they may not require the amount of exercise necessary to promote hypertrophy of normal muscles. Not only might atrophic cells respond more rapidly to stimulus, but it is known that growth of developing muscle in chicks is stimulated by simply stretching (Barnett et al. 1980). If the 1986 grebe population began to recover in mid-October, then a recovery sufficient to depart in late October would be rapid (especially for birds that had been on the lake for several months), but probably near normal physiological limits.

Decreases in grebe flight muscles cannot be attributed either to food shortage or to demands for shunting limited proteins to other sites in the body. Atrophy here is a matter of reduction, not degeneration. The maintained high density of mitochondria predisposes the cell to rapid growth. Fuel, in the form of intracellular lipid, disappears from cells early in the atrophic process and reappears early in recovery. This pattern has the characteristics expected of an adaptive response to a regularly encountered sequence of events. The response may have evolved from disuse-use conditioning, but appears to have been modified to facilitate recovery. Clearly there is a profound relationship between the stages of the sequence and the "decision" to stay or migrate, but it is unclear which is cause and which is effect. To resolve this conundrum, we must elucidate the nature of the physiologic link between morphology and behavior.

An even greater puzzle is the reason for the prolonged reduction of flight muscles. In the present case, flight muscles (including those in spring migrants that do not breed but spend the summer on the lake) atrophy within a few days of arrival. In autumn, the flight muscles are in prime condition upon arrival, atrophy rapidly, and remain reduced for weeks or months after wing molt is complete until just before migration. For most of autumn the grebes at Mono Lake live in an environment of abundant food (aquatic invertebrates), which is not a limiting resource. The risk of predation is essentially nil, and in any event, grebes escape danger by diving, not by flying. Thus, they have no need to fly for weeks or months. Hence, some reduction in the flight musculature arising from disuse is expected. During this time, however, the grebes lay down huge fat stores, and they should have no difficulty synthesizing

or maintaining protein reserves at minimal cost. Accordingly, we do not yet understand why the breast muscle atrophies so completely rather than maintains a flight mechanism in a low-level of readiness for emergency use. The events of 1986 and 1987 show that duration of abundant food is variable, and other conditions might also change unexpectedly in ways to favor a resumption of migration. In addition, Mono Lake is not the only lake on which grebes stage for migration, and conditions clearly vary among lakes. Even a minimal level of flying competence would further facilitate the rapid build-up to migratory condition whenever that becomes necessary.

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100 Years Ago in The Auk

From "Notes on habits of a few birds of Orange County, Florida" by D. Mortimer (1890, Auk 7: 339):

Ceophlœus pileatus. PILEATED WOODPECKER.

The Pileated Woodpecker is among the birds most limited in the variety of their notes, and indeed its only cry seems to be the wild clatter that has been so often described. On one occasion I discovered a pair of birds of this species apparently at play amongst the trees of a dense hummock. Wishing to secure them, I shot the female as she clung to a broken limb on a large oak. The male, who had been making a great noise, was silent a minute upon the report of the gun, but directly began again, and at the same time flew about rapidly as if trying to discover his mate. Presently he alighted on the very limb from which the other had fallen, and then I fired at him in the midst of one of his outbursts. Although he fell, he did not pause in his clatter for an instant, but came tumbling down until he caught in some moss at a distance from the ground, where he continued to vociferate without apparently allowing himself to draw a breath. Very soon he fell to the earth, but became quiet only when I pressed my hand upon his lungs. It would seem that this bird must have felt pleasure, fear, and pain during the time I observed him, all of which he expressed by the same sounds.