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THE EFFECT OF RESTRICTED FEEDING ON PLASMA GROWTH HORMONE (GH) CONCENTRATIONS IN GROWING AMERICAN KESTRELS[1]

DIANE LACOMBE[2] AND DAVID M. BIRD
Avian Science and Conservation Centre, Macdonald Campus of McGill University, 21,111 Lakeshore Road, Ste. Anne de Bellevue, Quebec H9X 3V9, Canada

COLIN G. SCANES
Department of Animal Sciences, Rutgers State University of New Jersey, New Brunswick, NJ 08903

KATHY A. HIBBARD
Department of Range Ecology, Texas A&M University, College Station, TX 77843

Abstract. In intact growing American Kestrels (*Falco sparverius*), plasma growth hormone (GH) concentration was correlated with body mass ($r_{Pearson} = -0.7504$; $P = 0.0001$; $n = 44$). High GH concentrations were observed during rapid growth periods (11 and 17 days of age) and lower values were present during slow growth periods (23 and 30 days of age: age at fledging). This maturational decline in the plasma GH concentration is due to age-related changes in the structure and the sensitivity/responsiveness of the somatotrophs of the pituitary gland to GH releasing and inhibiting factors. Restricted feeding (i.e., 90% and 70% of the control ad libitum intake) increased plasma GH concentrations above those of control birds during rapid growth periods but not during slow growth periods. Growth delay was observed with kestrels having lower body mass (16–19 and 24–26 days of age) and shorter antebrachium (10–12 and 16–19 days of age) than control birds according to the severity of the food restriction. As kestrels reached fledging (30 days of age), body mass was reduced only in birds fed the most restricted diet (70% at 30–33 days of age) and the antebrachium length was similar in birds fed the control diet and those fed the restricted diet (24–26 and 30–33 days of age). Food restriction increases the responsiveness of the pituitary gland to thyrotropin-releasing hormone (TRH: a GH releasing factor) and suppresses thyroid function (inhibiting factor of GH secretion), both resulting in an increased GH secretion. Changes in plasma hormone concentrations are needed to secure an adequate energy supply to vital organs during growth and food restriction.

Key words: American Kestrel; growth; growth hormone; body mass; antebrachium length; Falco sparverius.

INTRODUCTION

Plasma growth hormone (GH) has been studied extensively in domestic birds but less in wild bird species. The developmental pattern of GH concentrations has been characterized in domestic precocial species—the domestic fowl (*Gallus domesticus*) (Harvey et al. 1979, Scanes and Harvey 1981), the domestic turkey (*Meleagris gallopavo*) (Harvey et al. 1977a, Proudman and Wentworth 1980), the domestic goose (*Anser anser*), and the peking duck (*Anas platyrhynchos*) (Harvey and Phillips 1980, Foltzer et al. 1981)—and in one non-domesticated altricial species, the Ring Dove (*Streptopelia risoria*) (Scanes and Balthazart 1981). In all bird species studied, plasma GH concentrations are high after hatching and during the rapid growth period. Then, plasma GH concentrations decline during the slow growth period to reach low concentrations in adult birds.

Growth hormone lacks specific target organs, but it has widespread effects on the growth and the metabolism (Harvey 1990). The best characterized effects of GH in birds are on lipid metabolism (Scanes et al. 1983). Growth hormone stimulates lipolysis and exerts a glucose-sparing effect which favors both fatty acid utilization and decreased metabolism of glucose to fatty acids. This could be important in young rapidly growing birds in affecting the availability of circulating nutrients/metabolites to meet metabolic demands of growth (Scanes et al. 1983).

Short-term or chronic food restriction during growth, as well as fasting, increase plasma GH

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[2] Corresponding author.

concentrations above those observed in domestic birds fed normally and it could be accompanied by growth delay (Harvey et al. 1978, Scanes and Pethes 1979, Proudman and Opel 1981, Nir et al. 1983, Lauterio and Scanes 1988). The magnitude of the increase in GH concentration reflects the severity of the food restriction (Proudman and Opel 1981). Again, high plasma GH concentrations stimulate lipolysis. The effect of food restriction on plasma GH concentrations has not been studied in wild bird species, although food shortage can reduce survival of both nestlings and adults.

Studies of American Kestrels in our laboratory showed that nestling growth was delayed when food availability was reduced by 30% and nestlings fledged at a lower body mass than those fed normally (Lacombe et al., unpubl. data). We used the American Kestrel, a semi-altricial species, to determine the pattern of GH concentrations during normal growth and to examine whether plasma GH concentrations are modified by food restriction in nestlings.

MATERIAL AND METHODS

Housing of kestrels. This experiment was conducted at the Avian Science and Conservation Centre (ASCC) at McGill University (Ste. Anne de Bellevue, Quebec, Canada) where a captive colony of over 300 American Kestrels are maintained. American Kestrels are ideal laboratory animals due to their relative ease of acclimation to confinement and high reproductive success (Bird 1982, 1985).

GH concentrations during growth. To study plasma GH concentrations during growth in intact kestrels, 14 chicks were randomly selected from the colony's spring production. Chicks up to two weeks of age were fed ad libitum with mashed, day-old cockerels without down, head, feet or wings. After two weeks of age, the young were fed mashed cockerels with down and without head, feet, and wings. Cockerels were mashed less as the chicks' digestive system developed and the young learned to feed themselves. Calcium and mineral supplements were given alternately with each feeding, as cockerels may not contain the full calcium and mineral diet that kestrels require. Blood samples were taken in the morning by venipuncture of the brachial or the tarsal vein at 11, 17, 23, and 30 days of age. Capillary tubes containing sodium citrate as an anticoagulant were used to collect about 500 μl of blood. Blood samples were centrifuged and plasma was stored frozen at −20°C until GH assay. For birds at 11 days of age, when less than 500 μl of blood was collected, blood samples were pooled (equal volumes) for the subsequent GH assay. Chicks were weighed before blood collection. They were sexed at 17 days of age and the sex ratio was nine females: four males. One bird died before reaching 17 days of age.

GH concentrations during food restriction. Sixteen chicks were randomly assigned to four treatments at hatching. Sex ratios were evenly distributed for each treatment. Chicks were fed with day-old cockerels as in the first experiment. The following treatments were determined from an average of two previous years' data at ASCC. Birds were fed their respective diets from day 5 until day 15, after which time the amount of food given was held constant until day 35. The four treatments were: (1) ad libitum (100%): 2.7 g of food/feeding on day 5 to 9.6 g of food/feeding on day 35; (2) 90 % of ad libitum: 2.4 g of food/feeding on day 5 to 8.6 g of food/feeding on day 35; (3) 80% of ad libitum: 2.2 g of food/feeding on day 5 to 7.6 g of food/feeding on day 35; (4) 70% of ad libitum: 1.9 g of food/feeding on day 5 to 6.7 g of food/feeding on day 35. Chicks were fed four times daily at 08:30, 12:00, 16:00 and 20:00 hr. The amount of food consumed by each chick was measured by weighing each bird before and after each feeding.

In each group, eight birds were blood sampled at 10–12, 16–19, 24–26, and 30–33 days of age. Blood samples were processed as in experiment one for GH assay. Linear measurements of antebrachia were made with vernier calipers accurate to 0.1 mm. Measurements were taken daily for the first 10 days and then every third day until day 30. All birds were measured on day 30 and 35. The experiment was terminated at day 35. After the experiment, birds were monitored for several days to ensure that they could feed themselves before being permanently banded and released into the colony.

GH assay. The plasma concentration of GH was determined by a radioimmunoassay using chicken GH standard. Serial dilutions of kestrel plasma samples inhibited ^{125}I-chicken GH to antisera to chicken GH in a parallel manner to that of the chicken GH standard (C. G. Scanes, unpubl. data). Inter-assay and intra-assay coefficient of variation were respectively 11% and 6%. Sensitivity of the GH assay was 5.0 ng/ml.

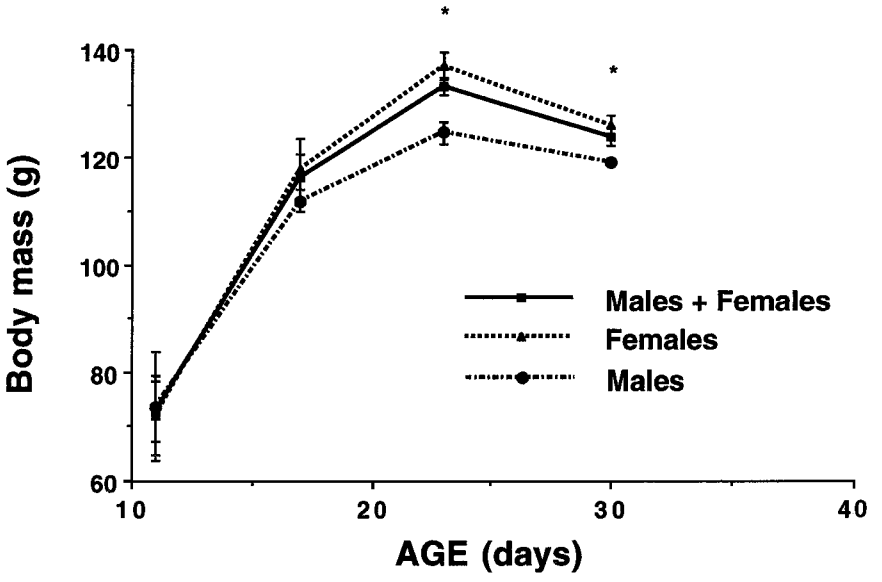

FIGURE 1. Changes in body mass in intact growing American Kestrels. Values shown are means ± SEM. A significant difference in body mass between males and females is indicated by a * ($P < 0.05$); $n = 3$ to 4 for males; $n = 3$ to 9 for females; $n = 6$ to 13 for males + females.

Statistical analysis. Normality of the data was checked with the Shapiro-Wilk test (Shapiro and Wilk 1965, SAS Institute Inc. 1985). To establish the degree of association between plasma GH concentrations and the body mass of kestrels we used the Pearson's coefficient of correlation. A regression (Model I) of plasma GH concentration against age was performed and significance of the regression was tested with an analysis of variance. The significance of the regression coefficient and that of the intercept was tested with a Student's *t*-test. Differences between males and females for the body mass and the plasma GH concentration were evaluated with the Student's *t*-test. Data that were not normally distributed were logarithmically transformed to satisfy the assumption required for parametric statistics. Differences among real means or logarithmically transformed means were evaluated by one-way analysis of variance (ANOVA: Model I) followed by Scheffé's test (SAS Institute Inc. 1985). When log-transformed data had a non-normal distribution, we used a one-way ANOVA applied to ranks (which is equivalent to a Kruskal-Wallis test) (SAS Institute Inc. 1985: 651) and then followed by a Student-Newman-Keuls test to detect any difference between means. Combined effects of age and feeding regimen on plasma GH concentrations, body mass and antebrachium length were tested with a two-way ANOVA followed by a Scheffé's test (SAS Institute Inc. 1985).

RESULTS

GH concentrations during growth. Young kestrels grew quickly from hatching to 17 days of age. Then, kestrels grew more slowly and body mass of nestlings levelled off. Kestrels showed a slight loss of body mass from day 24 to 30, which is the age at fledging (Fig. 1). A highly significant negative correlation was observed between body mass and plasma GH concentration in intact growing kestrels ($r_{Pearson} = -0.7504$; $P = 0.0001$; $n = 44$). The highest plasma GH concentration was observed at the lowest body mass (11 days of age) and plasma GH concentrations decreased significantly with increasing body size (Fig. 2A). Growth hormone concentrations decreased with age in intact growing kestrels as shown by the significance of the regression ($F = 49.234$; $P = 0.0001$), the coefficient of regression ($t = -7.017$; $P = 0.0001$), and the intercept ($t = 10.756$; $P = 0.0001$) (Fig. 2B).

If sexes are separated for the analysis, a sexual dimorphism is observed with females having higher body mass than males at 23 and 30 days of age (Fig. 1). Plasma GH concentrations showed a tendency to be lower in males compared to females during the rapid growth period (11 and

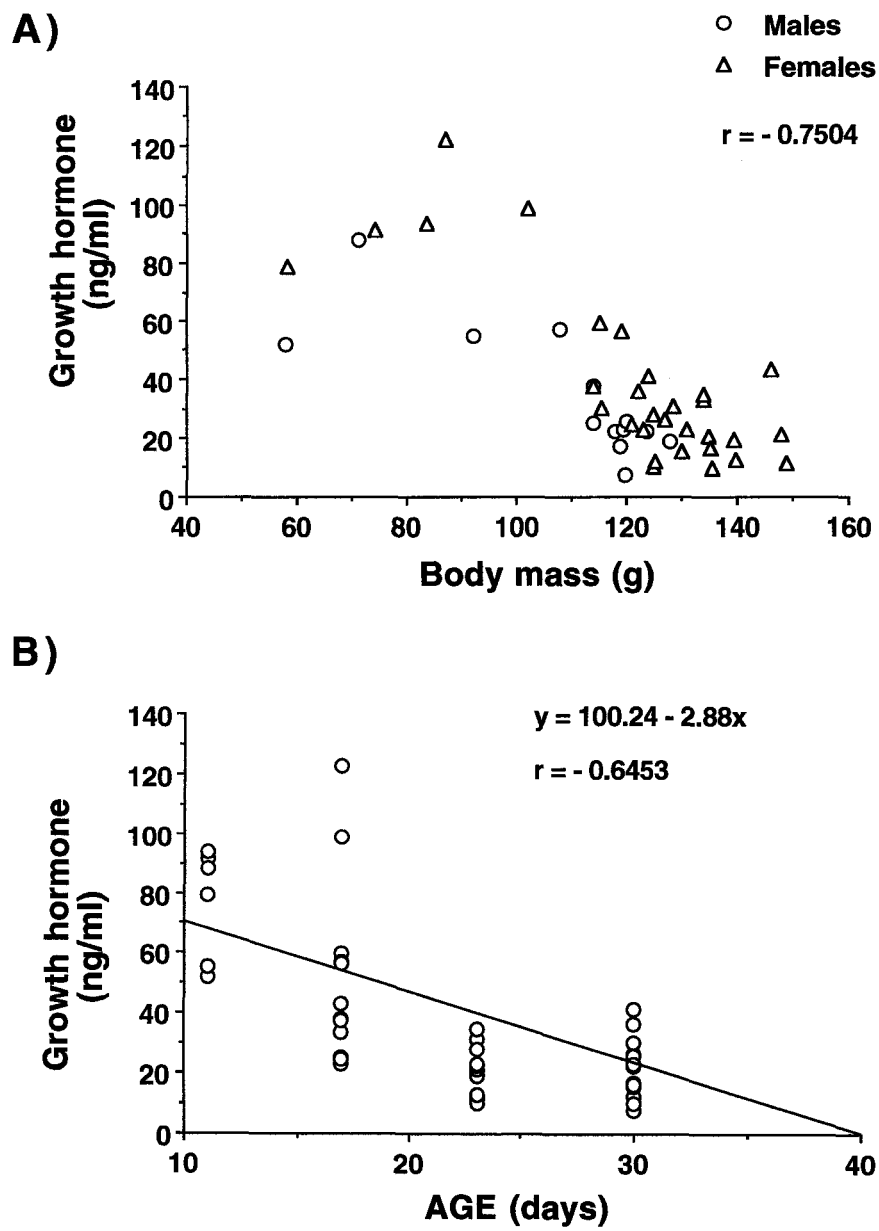

FIGURE 2. A) Scatter diagram showing the variation of plasma GH concentrations with body mass in growing intact American Kestrels ($n = 44$). B) Plasma GH concentration of intact growing American Kestrels as a function of age (male and female samples pooled; $n = 44$).

17 days of age with $P = 0.1370$ and $P = 0.4759$ respectively), but the difference was not significant (Fig. 3). Plasma GH concentrations of both sexes were pooled for further analysis (Fig. 3, 4). Plasma GH concentrations (sexes pooled) were high during rapid growth periods at 11 and 17 days of age and low during slow growth periods at 23 and 30 days of age (Fig. 3, 4).

GH concentrations during food restriction. In the restricted feeding experiment, there was no significant interaction between age and restricted diet on plasma GH concentrations (Log-trans-

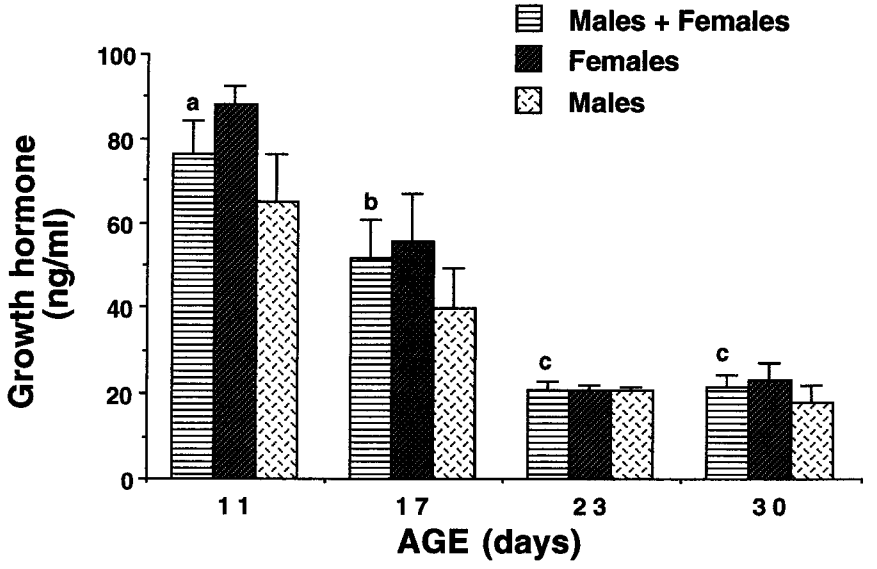

FIGURE 3. Changes in plasma GH concentrations in growing intact American Kestrels. Values shown are means ± SEM. Means with the same letter (Males + Females across age) are not significantly different ($P > 0.05$); no significant difference between Males and Females within a given age ($P > 0.05$); $n = 3$ to 4 for males; $n = 3$ to 9 for females; $n = 6$ to 13 for males + females.

formed data; two-way ANOVA: $F = 0.55$, $P = 0.8337$). At 10–12 days of age, plasma GH concentrations in birds fed 90% and 70% of the control diet (100%) were significantly higher than those of the control birds (100%) (Fig. 4). However, birds on the 80% diet had plasma GH concentrations similar to those of birds on the control (100%) and the 90% diets. The highest plasma

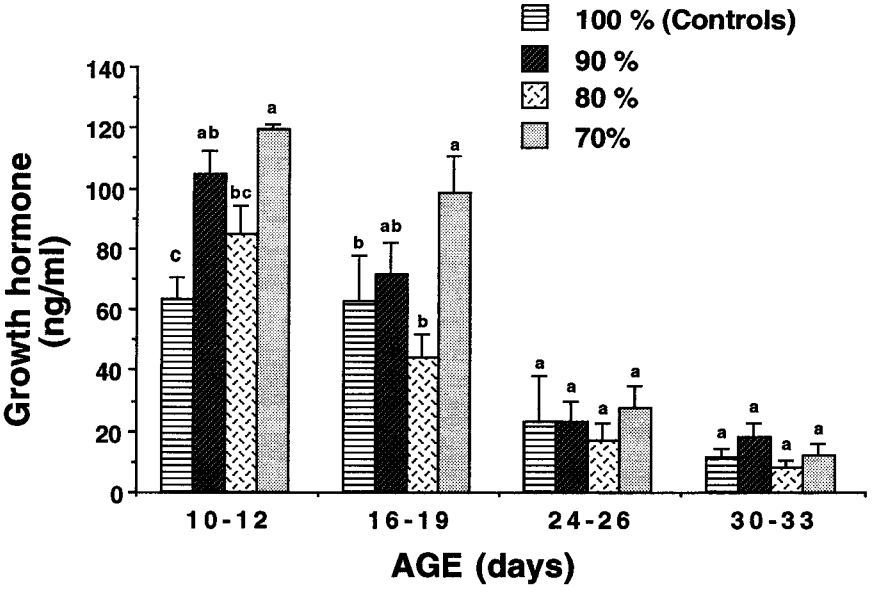

FIGURE 4. Comparison of plasma GH concentrations in growing American Kestrels fed on restricted diets. Values shown are means ± SEM. For a given age, means with the same letter are not significantly different ($P > 0.05$); $n = 5$ to 8.

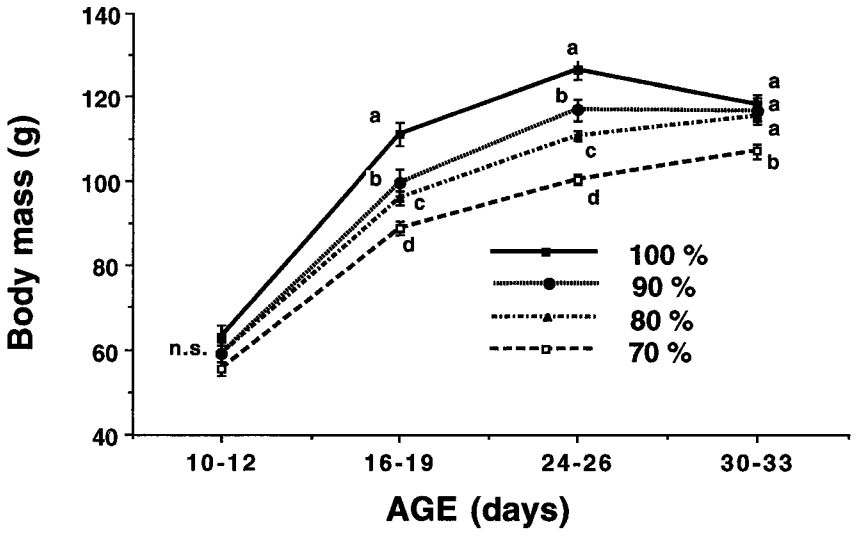

FIGURE 5. Comparison of body mass in growing American Kestrels fed on restricted diets. Values shown are means ± SEM. For a given age, means with the same letter are not significantly different ($P > 0.05$); n = 14 to 16. N.S.: no significant difference between means ($P > 0.05$).

GH concentration was observed in birds on the 70% diet. Similarly, at 16–19 days of age, plasma GH concentrations were significantly higher only in birds on the 70% diet compared to the control birds (100%). Plasma GH concentrations were no longer affected by restricted feeding at 24–26 and 30–33 days of age. For each diet, plasma GH concentrations were significantly reduced during slow growth periods compared to rapid growth periods.

Restricted feeding also affected growth parameters in the kestrel. A significant interaction between age and restricted feeding was detected for body mass (two-way ANOVA: $F = 5.02$, $P = 0.0001$). Body mass was similar in control birds and birds fed the restricted diets at 10–12 days of age (Fig. 5). However, body mass of birds on restricted diets was significantly lower than that of the control group (100%) at 16–19 and 24–26 days of age (Fig. 5), and it decreased with the severity of the food restriction. As the birds reached adult size at 30–33 days of age, significant differences in body mass were only observed in birds fed the 70% diet.

A significant interaction between age and restricted diet was also observed for the antebrachium length (two-way ANOVA: $F = 2.16$, $P = 0.0138$). During rapid growth periods (i.e., at 10–12 and 16–19 days of age), the antebrachium was significantly shorter in birds fed the restricted diets compared to those fed the control diet (except for birds fed the 80% at 10–12 days of age) (Fig. 6). During slow growth periods (i.e., at 24–26 and 30–33 days of age), birds fed restricted diets reached the same antebrachium length as birds fed the control diet.

DISCUSSION

GH concentrations during growth. The maturational decline in the plasma GH concentrations observed in the kestrels and other bird species (Harvey et al. 1977a, 1977b; Scanes et al. 1979; Foltzer et al. 1981; Scanes and Balthazart 1981; Scanes et al. 1981; Scanes and Lauterio 1984) is due to age-related changes in the structure (decreased size and GH content of secretory granules) of the somatotrophs of the pituitary gland and to a decline in their sensitivity/responsiveness to releasing (TRH: thyrotropin-releasing factor and GRF: growth hormone-releasing factor) and inhibiting factors (SRIF: somatostatin) (Scanes et al. 1990). Peripheral hormones (somatomedin or thyroid hormones) that inhibit GH secretion in birds may also be involved (Scanes et al. 1990).

The sexual dimorphism in size observed in kestrels at 23 and 30 days of age is normal in this species (Bird and Clark 1983) and coincides with the maturational decline in plasma GH concentrations near fledging (30 days of age). Both

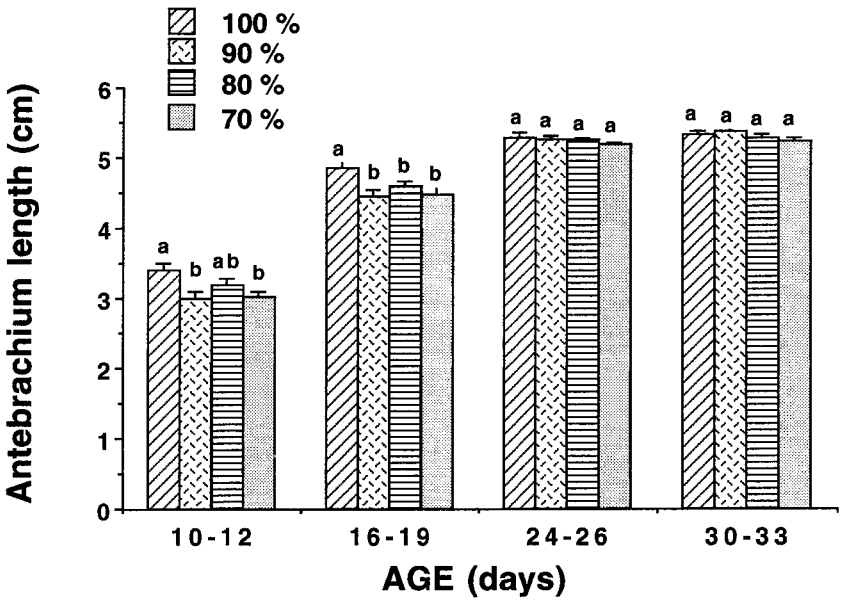

FIGURE 6. Comparison of the antebrachium length in growing American Kestrels fed on restricted diets. Values shown are means ± SEM. For a given age, means with the same letter are not significantly different ($P > 0.05$); $n = 14$ to 16.

occur considerably prior to puberty, since the gonads of young kestrels remain in a regressed state for up to eight months. It is assumed that sex steroids have only minor effects on GH secretion and they do not appear to be involved in either the maturational decline of plasma GH concentrations or the marked sexual dimorphism observed in the chicken (Scanes and Johnson 1984, Fennel et al. 1990). Other factors, such as genetic differences at the level of organ or cellular growth, are more likely mechanisms for sex dimorphism (Fennel et al. 1990).

Lack of significant differences in plasma GH concentration between males and females at 11 and 17 days of age may result from small sample sizes and high individual variation, as reported in other birds (Scanes et al. 1983). However, there is some evidence that plasma GH concentrations differ between male and female chickens at various ages (Harvey et al. 1979, Burke and Marks 1982, Stewart and Washburn 1983). Moreover, a sexually dimorphic ontogeny of GH secretion occurs in some breeds of chicken (Johnson 1988).

GH concentrations during food restriction. Restricted feeding increased plasma GH concentrations in the kestrel (i.e., at 11 and 17 days of age) above concentrations observed in kestrels fed normally. The highest concentration was observed with the most severely restricted diet (70% of the ad libitum control diet). Similar results for plasma GH concentration during food restriction were observed during the growth of the domestic chicken (Harvey et al. 1978), the turkey (Proudman and Opel 1981), and the duck (Scanes and Pethes 1979).

However, with kestrels reaching fledging (23 and 30 days of age), plasma GH concentration was low in intact kestrels and restricted feeding did not affect plasma GH concentration. Food restriction increases the responsiveness of the pituitary gland to TRH (releasing factor for GH secretion) (Harvey 1983, Harvey and Baidwan 1989) and suppresses thyroid function (inhibiting factor for GH secretion) (Lauterio and Scanes 1988). Both result in an increase in GH secretion and plasma GH concentrations are elevated above those observed in birds fed ad libitum. However, the age-related decline in the responsiveness of the somatotrophs to TRH and peripheral hormones (somatomedins or thyroid hormones) as well as changes in their structure could explain the lack of effect of food restriction on plasma GH concentrations when kestrels reach fledging.

Growth delay was observed in the kestrel as the diet became more restricted, and it was ac-

companied by high plasma GH concentrations as seen in other bird species (Harvey et al. 1978, 1981, Engster et al. 1979, Nir and Nitsan 1979, Scanes and Pethes 1979, Hoshino et al. 1980, Scanes et al. 1981, Scanes and Harvey 1982, Nir et al. 1983). Also, young kestrels fed on restricted diets had significantly lower body mass at fledging and they stored less fat than kestrels fed ad libitum (Lacombe et al., unpubl. data). Since GH exerts a short-term control on body metabolism, inhibiting lipogenesis and stimulating lipolysis (Harvey et al. 1977c, Scanes 1992), it is likely that increased plasma GH concentrations caused by restricted feeding in the kestrel mobilize fatty acids from adipose tissue and that stored carbohydrates are not used to synthesize fat (Scanes et al. 1984).

Therefore, reduced growth due to a lower availability of substrate during times of nutritional deprivation is likely to occur. Under these circumstances, GH could be aiding the homeostatic response by increasing lipolysis (Scanes et al. 1990). Other hormones such as thyroid hormones, cortisol, and prolactin may also be involved in this action because all of these can be lipolytic in certain circumstances (Sheridan 1986). Further studies on plasma GH concentrations in wild bird species are needed to know the patterns of GH concentration during growth, as well as to fully understand the mechanisms involved in the increase of plasma GH concentrations during food restriction.

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