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Compensatory responses to food restriction in juvenile green turtles (*Chelonia mydas*)

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Abstract. The purpose of this study was to assess the compensatory responses to food restriction and subsequent increased food availability in juvenile green turtles (*Chelonia mydas*). Turtles were fed an ad libitum ration for 12 weeks (AL), a restricted ration for 12 weeks (R), or a restricted ration for 5 weeks and an ad libitum ration for 7 weeks (R-AL). Analysis of covariance was used to test the relationships between (1) growth and body size, (2) intake and body size, and (3) growth and intake for each of the three treatment groups. Body composition of turtles in each group was also evaluated at the beginning of the study and after weeks 5 and 12. After the switch to ad libitum feeding, R-AL turtles consumed comparable amounts of food and grew faster than AL turtles on a size-adjusted basis, but mean body sizes did not converge, although the overlap in their size ranges increased with time. The R-AL turtles also converted food to growth more efficiently and allocated proportionally more nutrients to protein accretion, thereby restoring body composition (except mineral content) to AL levels by the end of the study. Thus, accelerated size-specific growth without hyperphagia restored body condition but not size. These results indicate that (1) intake in juvenile green turtles is maximal when food is readily available and cannot be increased to compensate for a previous period of food limitation, (2) growth rates of ad libitum-fed turtles are only mildly plastic in response to past nutritional history, and (3) priority rules for nutrient allocation favor the attainment of an optimal condition rather than an optimal size. Nutritional setbacks experienced during the vulnerable juvenile stage could therefore have long-lasting consequences for wild turtles in terms of size-specific mortality risk, but these risks may be mitigated by the potential benefits of maintaining sufficient body stores.

Key words: body composition; body size; catch-up growth; Chelonia mydas; *compensatory growth; conversion efficiency; food restriction; green turtle; hyperphagia; nutrition; organ size; reptile.*

Introduction

Growth rates of wild animals can be highly variable, particularly in response to resource availability. This variation increases the size disparity among individuals. Because smaller individuals are typically more susceptible to predation and starvation, these costs of growth limitation should select for adaptations that allow previously food-limited individuals to exploit better conditions whenever they are encountered.

One adaptive response to fluctuating resource availability is compensatory growth (CG), a period of accelerated growth during improved food conditions following growth restriction (Wilson and Osbourn 1960). Compensatory growth causes growth trajectories of individuals with different intake histories to converge toward a putative optimum, thereby minimizing the variance in body size among same-age individuals (Atchley 1984, Wilson and Réale 2006). Compensatory growth presumably allows organisms to avoid the costs of being small. This growth pattern has been documented in numerous animals, with the majority of CG studies focusing on commercially important teleost (Ali et al. 2003) or livestock (Mitchell 2007) species. However, the capacity for compensatory growth is not universal (e.g., Altwegg and Reyer 2003, Brzęk and Konarzewski 2004), and knowledge of its occurrence in non-teleost ectotherms is limited.

When CG does occur, the magnitude of compensation depends on the species in question, the developmental stage of the organism at the times of restriction and realimentation, and the length and severity of the period of food restriction (Wilson and Osbourn 1960, Ali et al. 2003, Mitchell 2007). However, the mechanisms underlying compensatory growth are relatively conserved. To grow more quickly than their well-fed counterparts, previously restricted animals demonstrate hyperphagia (i.e., increased intake) and/or are more efficient at converting food to growth during the realimentation period (Wilson and Osbourn 1960, Broekhuizen et al. 1994, Gurney et al. 2003).

Studies in fish and mammals have confirmed that the standard response to food limitation is to become

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hyperphagic relative to continuously well-fed animals when food availability increases (Wilson and Osbourn 1960, Ali et al. 2003, Gurney et al. 2003). A long-standing hypothesis (Kennedy 1953) posits that appetite and food intake are regulated lipostatically, with intake responding to intrinsic signals of adiposity that are independent of body size (Jobling and Johansen 1999, Johansen et al. 2001). Because lipid reserves tend to be mobilized before protein stores during food limitation (Cherel et al. 1993, Tian and Qin 2004), food-restricted animals are generally leaner than continuously well-fed individuals. This altered body composition then purportedly fuels compensatory hyperphagia upon refeeding.

Altered body composition may also facilitate enhanced food conversion efficiency (FCE) during growth compensation. Typically, the early phases of CG are characterized by protein deposition (e.g., Qian et al. 2000). This differential accretion of lean tissue provides a mechanism for accelerated growth, as protein deposition requires less energy than fat deposition (Hornick et al. 2000). Alternatively, FCE can be increased by decreasing metabolic costs (Skalski et al. 2005), thereby freeing a larger proportion of ingested energy to be routed to production. Although this inverse relationship between FCE and standard metabolic rate has been demonstrated in reptiles, it does not necessarily facilitate CG after a period of food restriction because FCE in these animals was not affected by intake history (Cox and Secor 2007).

In this study, we assessed the compensatory responses to food restriction and subsequent increased food availability in juvenile green turtles (*Chelonia mydas*). The green turtle leads an oceanic existence for the first several years of life and consumes a largely carnivorous diet during that time (Reich et al. 2007). Intake in this stage most likely varies stochastically due to heterogeneous prey distribution. At a size of ~20–25 cm carapace length (for Atlantic *C. mydas*) or 35 cm carapace length (for Pacific *C. mydas*), green turtles undergo an ontogenetic shift in habitat use and diet by recruiting to neritic habitats, where they consume a largely herbivorous diet of algae and seagrasses (Bjorndal 1997). Although growth dynamics of juveniles during the oceanic stage are unknown, post-recruitment growth rates are known to vary temporally as a result of variation in oceanographic conditions (Limpus and Chaloupka 1997) and population density (Bjorndal et al. 2000). This variation in juvenile growth rates may have substantial fitness effects, as body size is correlated with juvenile survivorship (Chaloupka and Limpus 2005) and clutch size (Broderick et al. 2003) in *C. mydas*.

Given its dietary habits and life history, *C. mydas* is an excellent model species for studying the effects of food limitation. Its size-specific survival and reproduction suggest that green turtles should be capable of growth compensation, as has been shown for wild juvenile loggerheads (Bjorndal et al. 2003). The physiological adjustments that permit growth compensation in marine turtles are currently unknown and could differ from those utilized by other taxa. Understanding how marine turtles respond to nutritional stress in a captive setting will improve our ability to predict the effects of fluctuating resource availability in wild populations.

Materials and Methods

Animal care

The feeding trial was conducted at the Cayman Turtle Farm in Grand Cayman, British West Indies, in compliance with the University of Florida Institutional Animal Care and Use Committee. *Chelonia mydas* hatchlings ($n = 115$) were housed individually in seawater in 68-L tanks arranged in an outdoor enclosure. Seawater was continuously circulated within the large enclosure at a depth of ~20–25 cm to maintain consistent temperatures within individual tanks. The water in each tank was replaced daily, and water temperature was monitored at five locations within the array of tanks. Treatment groups were systematically arranged within the enclosure to minimize position effects.

Turtles were fed 2.6-mm turtle pellets (Melick Aquafeed, Catawissa, Pennsylvania, USA) twice daily and were allowed to feed for 7–10 h each day. Pellets remaining in each tank were counted every afternoon, and approximate intake was calculated based on the average mass per pellet (determined weekly), the known mass of pellets offered, and the number of pellets remaining. Intake for each turtle was quantified six days per week (weather permitting), and tanks were cleaned once per week. Straight carapace length (CL) and body mass of each turtle were measured weekly. Five food samples were weighed and dried every two weeks for nutrient analyses.

Turtles were fed ad libitum for seven days (week 0) prior to the beginning of the experiment to establish daily ad libitum intake. Turtles were then assigned to one of three treatment groups: ad libitum (AL), restricted (R), and restricted-ad libitum (R-AL). The AL turtles were fed ad libitum for 12 weeks. The R turtles were fed ~50% of the initial ad libitum intake on a percentage of body mass basis for 12 weeks. This ration slightly exceeded basal maintenance costs, as food-restricted turtles continued to gain both mass and length throughout the trial. The R-AL turtles were food-restricted for five weeks and then fed ad libitum for seven weeks.

Assimilatory organ sizes and body composition

Turtles were euthanized with an intramuscular overdose injection of ketamine (Ketaset, Fort Dodge, Iowa, USA; 100 mg/kg body mass) prior to, during, and after the study for analysis of organ sizes and body composition. Ten turtles were euthanized after week 0 (when all turtles were feeding ad libitum) and are hereafter referred to as week 0 AL turtles. Ten AL, five

R, and five R-AL turtles were euthanized after week 5. Because the five R and five R-AL turtles were all food-restricted in weeks 1–5, these 10 turtles were pooled into a group hereafter referred to as week 5 R turtles. Ten AL, 10 R, and 10 R-AL turtles were euthanized after week 12.

The liver and digestive tract (from the lower esophageal sphincter to the distal end of the hindgut anterior to the cloaca) of each turtle were removed. Gut contents were gently removed from the excised gut using forceps and weighed. Wet masses (at dissection) of liver, stomach, and total intestine were determined. Masses of midgut and hindgut could not be evaluated separately because the distinction between the two intestinal segments was not easily discernible.

Tissues and carcasses were dried at 60°C for a minimum of seven days. Dried body tissues (including blood) were recombined for each turtle and then ground in a mill (C.W. Brabender Instruments, South Hackensack, New Jersey, USA) with dry ice. Dried food samples were also ground in the mill without dry ice. Ground samples were dried again overnight at 60°C, and subsamples of each food sample and turtle were then analyzed for dry matter (DM), organic matter (OM), mineral, water, lipid, nitrogen, and energy contents. Dry matter content was determined by drying at 105°C for 16 h, and OM content was determined by mass lost during combustion at 500°C for 3 h, with residue (ash) equaling mineral (osseous + nonosseous) content (AOAC 1960). Water content of each turtle was calculated as the mass lost from the original wet mass of the whole animal (minus gut contents) during drying at 60°C. Lipid content was determined by ether extraction using a soxhlet apparatus (AOAC 1984). Nitrogen content was determined with a modified Kjeldahl procedure. Samples were digested for at least 4 h at 375°C using a modification of the aluminum block digestion procedure of Gallaher et al. (1975). Nitrogen in the digestate was determined by semiautomated colorimetry using a Technicon Autoanalyzer (Hambleton 1977; Pulse Instrumentation, Saskatoon, Sasakatchewan, Canada). Energy content of each food sample and turtle was determined by bomb calorimetry (Parr Instrument 1960; Parr Instrument, Moline, Illinois, USA). Nutrient analyses were performed in duplicate unless relative error exceeded 2.0%, in which case additional analyses were performed.

Statistical analyses

Body size (carapace length, CL) and intake (in grams per day) in turtles from all three treatment groups throughout the study were evaluated using repeated-measures analysis of variance (RM ANOVA) with Tamhane's T2 post hoc test. Average weekly intake (percentage of body mass per day) in AL turtles was also compared to that in R-AL turtles for each of weeks 6–12 (after the diet switch for R-AL turtles) using ANOVA or the appropriate nonparametric alternative. Intake, growth, and food conversion efficiency in AL and R-AL turtles in each of weeks 6–12 were then compared using analysis of covariance (ANCOVA). Intake for a given week was assessed using CL at the middle of that week as the covariate. Growth was assessed as change in CL in a given week using CL at the beginning of that week as the covariate. Conversion of consumed food to growth (food conversion efficiency) was assessed as change in CL in a given week using cumulative intake in that week as the covariate. Data were log-transformed prior to analysis to satisfy the assumptions of ANCOVA. Carapace length but not body mass was used as a body size covariate because changes in CL are not affected by gut filling. For all RM ANOVAs, if Mauchley's test indicated that the sphericity assumption was violated, Greenhouse-Geisser F and P values are reported.

To evaluate initial ad libitum intake (week 0) and initial and final body size (CL in weeks 0 and 12), we used one-way ANOVAs. Data were first transformed if doing so was deemed necessary because of a significant result for the Shapiro-Wilk test (for normality) and/or Levene's test (for homogeneity of variances). Pairwise post hoc comparisons of week 12 data were evaluated using Tukey's honestly significant different (hsd) test. If the assumptions of ANOVA could not be met, data were analyzed using a Kruskal-Wallis test.

Organ masses and body composition (as a percentage of body mass, data not shown) were both correlated with body size within treatment groups at each sampling time, thus necessitating a regression approach to compare among groups (Shearer 1994, Hayes and Shonkwiler 2001). We therefore tested these data using ANCOVA with body mass (excluding gut contents) as the covariate. All ANCOVAs were performed using log-transformed data to improve normality, and results are presented as estimates of each parameter at a common mean body size.

To determine the composition of growth, absolute masses (rather than size-adjusted estimates) of water, minerals, and lipid were averaged for each treatment group at each sampling time. These averages were then used as estimates of the masses of water, minerals, and lipid in a hypothetical turtle from each treatment group at each sampling time. For these estimates, total wet body mass was assumed to be equal to the actual mean body mass (excluding gut contents) of turtles from each treatment group at each sampling time. Protein was then estimated as the remainder of wet body mass, assuming that carbohydrate content was negligible (as in Johansen et al. 2001). Net change in body composition was then estimated by calculating the change in mass of each component between weeks 0 and 5 (for AL and R turtles) and weeks 5 and 12 (for AL, R, and R-AL turtles).

Data were analyzed using SPSS, release 11.0.0 (SPSS, Chicago, Illinois, USA). Only data for apparently

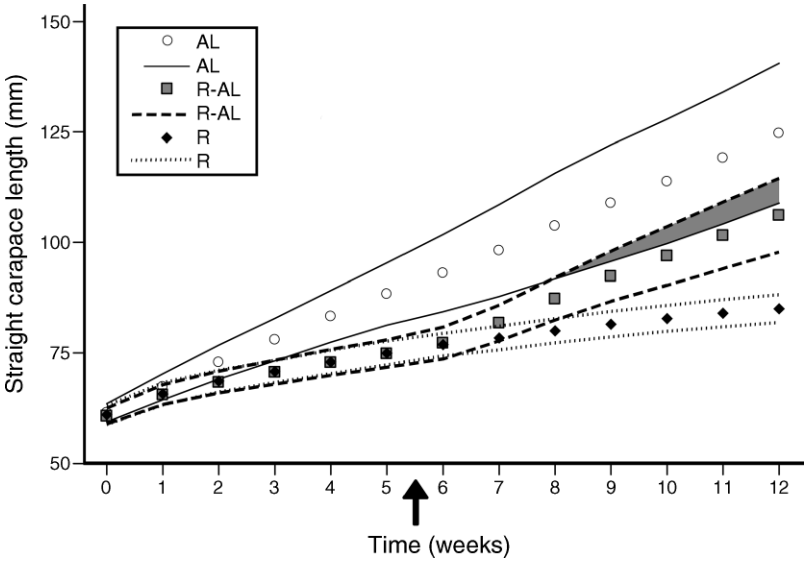

FIG. 1. Straight carapace length at the midpoint of each week for juvenile green turtles (*Chelonia mydas*) in three treatment groups: AL, ad libitum for 12 weeks; R, food-restricted for 12 weeks; R-AL, food-restricted for five weeks and ad libitum for seven weeks. The arrow indicates when R-AL turtles were switched from a restricted to an ad libitum diet. Points represent means, lines represent ±SD from the mean, and the shaded area is the region of overlap in standard deviations of AL and R-AL turtles. Sample sizes: $n = 37$ AL, 39 R-AL, and 39 R turtles (weeks 0–5); $n = 17$ AL, 29 R-AL, and 29 R turtles (weeks 6–12).

healthy turtles were analyzed. Data are expressed as mean ± SE, unless otherwise noted, with $\alpha = 0.05$.

RESULTS

Intake (ANOVA, $F_{2,112} = 0.946$, $P = 0.392$) and CL (ANOVA, $F_{2,112} = 1.109$, $P = 0.333$) of all turtles ($n = 37$ AL, 39 R-AL, and 39 R) in week 0 (prior to the beginning of the experiment) were not significantly different among the three treatment groups. During the study, carapace length (CL, Fig. 1) of the three groups diverged substantially as a result of differences in intake. Analyses of intake and growth as the study progressed were complicated by a decrease in sample sizes after week 5. To elucidate properly the effects of time and treatment on intake and growth, we restricted our analyses of data collected weekly to include only those animals that survived through week 12. Doing so permitted the use of a repeated-measures approach for intake and body size. Using this approach, we found significant effects of time, treatment, and the interaction between time and treatment on intake and CL throughout the trial (Appendix A). For all effects, the three treatment groups differed significantly (Tamhane's T2 post hoc test, $P < 0.001$ for all comparisons).

Significant RM ANOVA results were explored further using univariate analyses within individual weeks. However, interpretation of the data was complicated by the confounding influence of body size, which differed among treatment groups except during week 0. For this reason, we used ANCOVA to compare estimated means for intake, growth, and food conversion efficiency. Because assessment of the capacity for and mechanisms of CG requires comparison of previously restricted animals with those feeding ad libitum continuously, we excluded R turtles and included only data from AL and R-AL turtles in weeks 6 through 12 for these ANCOVA analyses.

Although mean sizes of AL and R-AL turtles did not converge by the end of the study, the overlap in their size ranges increased with time (Fig. 1). On a size-specific basis, R-AL turtles grew faster than AL turtles after the switch to ad libitum feeding (Fig. 2). This faster growth was revealed by covariate analysis that corrected for initial carapace length and was thus not an allometric artifact resulting from size differences among smaller R-AL turtles and larger AL turtles. Despite growing faster, R-AL turtles were still significantly smaller than AL turtles at the end of week 12 (Kruskal-Wallis test, $\chi^2 = 14.535$, $P = 0.0001$).

We found no evidence of hyperphagia when intake was compared either by ANCOVA using CL as the covariate (Fig. 2) or by ANOVA on a percentage of body mass basis (Fig. 3), although the possibility of transient hyperphagia in week 6 could not be explored statistically because of a significant interaction between treatment group and body size. Despite consuming similar size-adjusted quantities of food, R-AL turtles converted this food into growth more efficiently than AL turtles in weeks 6–11 (Fig. 2).

After five weeks of food restriction, livers in R turtles were smaller with higher water content than in turtles feeding ad libitum (Fig. 4). Continued food restriction caused both intestine and liver in R turtles to be smaller with a higher water content than in AL or R-AL turtles

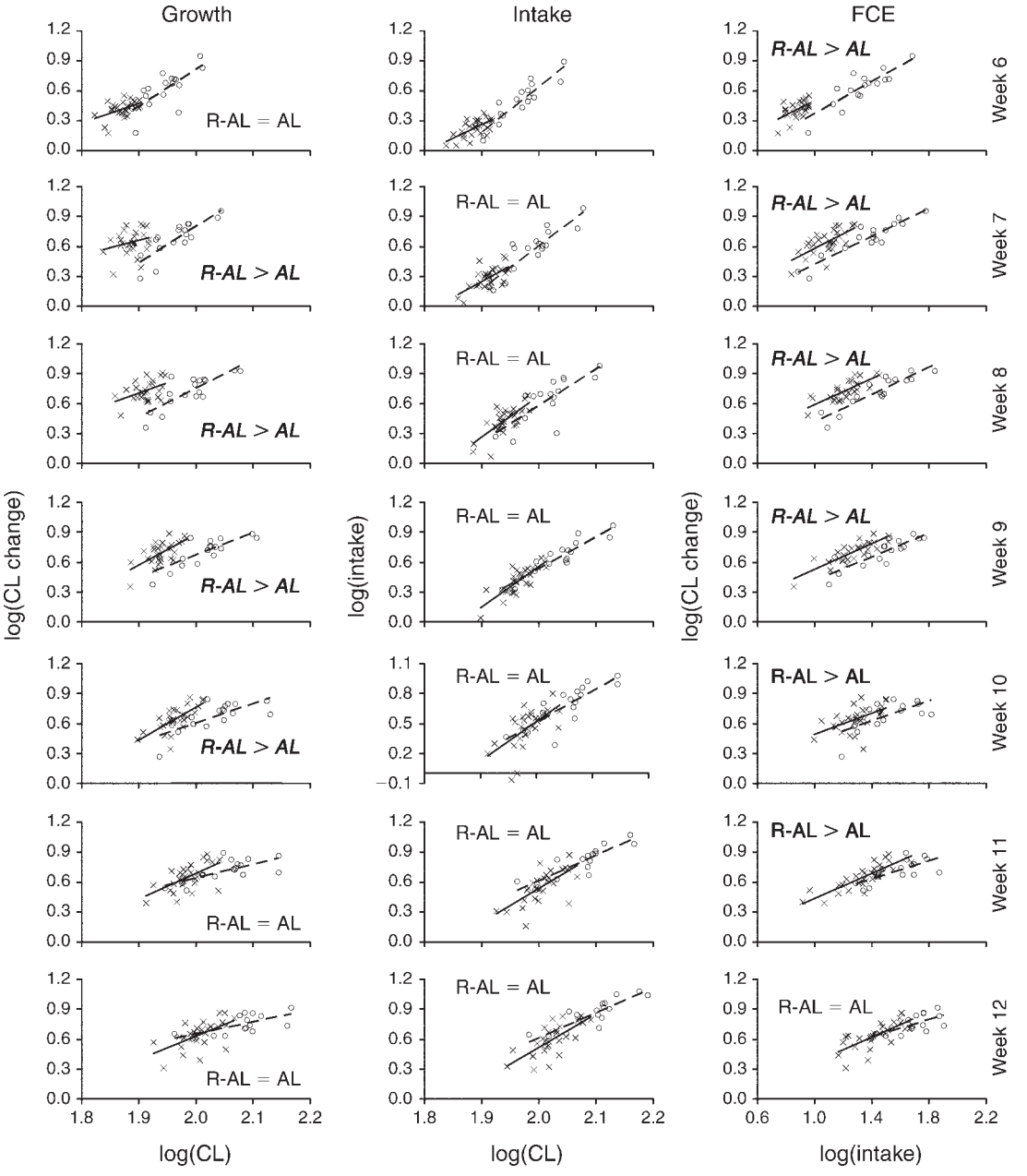

FIG. 2. Regressions for growth, intake, and food conversion efficiency (FCE) in AL (open circles, dashed lines) and R-AL (crosses, solid lines) juvenile green turtles in each of weeks 6–12, when both groups were feeding ad libitum. Data were log-transformed prior to analysis to satisfy the assumptions of ANCOVA. CL indicates straight carapace length; treatment groups are as in Fig. 1. The equation in each graph refers to ANCOVA results; boldface indicates $P < 0.05$; italicized boldface indicates $P < 0.01$. The log-transformed dependent variables were change in CL (for growth and FCE) and average daily intake $\times$ 7 (for intake). The log-transformed covariates were CL at the beginning of the week (for growth), CL at the middle of the week (for intake), and average daily intake $\times$ 7 (for FCE). A comparison of intake in AL and R-AL turtles in week 6 was not possible because of a significant treatment $\times$ covariate interaction. Sample sizes: $n = 17$ AL and 29 R-AL in each week.

by the end of week 12. Total intestine length followed the same pattern as total intestine mass (data not shown).

Turtles prior to and after the diet switch also differed significantly in body composition (Table 1), with R turtles having higher water and lower mineral, OM, energy, and nitrogen contents than those of AL turtles. Body size-adjusted lipid content did not differ among AL and R turtles in week 5 or week 12, although lipid content of R-AL turtles was significantly higher than that of R turtles in week 12. Body composition of R-AL turtles was not significantly different from that of AL

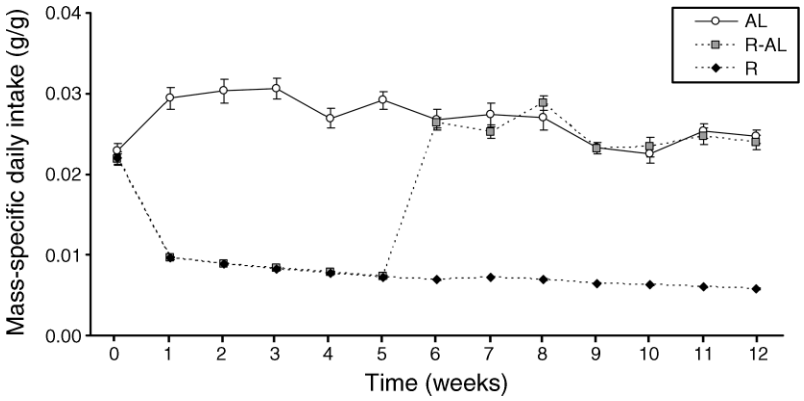

FIG. 3. Intake (mean ± SE) in each week as a percentage of body mass. No significant differences were detected among AL and R-AL turtles after the diet switch at the beginning of week 6. Sample sizes and treatment groups are as in Fig. 1.

turtles in week 12, with the exception of mineral content, which was lower in R-AL turtles than in AL turtles.

We used our body composition results to estimate the mass of water, minerals, protein, and lipid in a hypothetical turtle of average mass in each treatment group in weeks 0, 5, and 12. The estimated composition of gain was then calculated as the net change in the mass of each component from one sampling time to the next for each treatment group (Fig. 5). Based on these estimates, R turtles experienced a net loss of lipid mass between weeks 0 and 5 but were able to increase protein mass slightly as they grew in size. During this same interval, growth in AL turtles resulted from increases in both protein and lipid masses. Mineral gains accounted for approximately 4.7% and 4.6% of total body mass gains in R and AL turtles, respectively. Between weeks 5 and 12, all turtles gained protein and lipid masses, but both protein and lipid mass represented a smaller proportion of total body mass gain in R turtles than in AL or R-AL turtles. During this time interval, allocation to protein mass gain was proportionally higher than allocation to lipid mass gain in all groups, even when accounting for the higher energy density of fat (38.9 kJ/g of fat, 17.6 kJ/g of protein; Randall et al. 1997). Using our estimates of mass gains and known values for energy densities, we calculated the ratio of caloric allocation to protein gains vs. lipid gains in each treatment group and found that R-AL turtles preferentially allocated more calories to protein gains than AL turtles (Fig. 5). Mineral gains between weeks 5 and 12 accounted for approximately 5.5%, 5.3%, and 4.5% of total body mass gain in R, AL, and R-AL turtles, respectively.

Organic matter, energy, nitrogen, and lipid contents of food samples ($n = 7$) were consistent throughout the experiment (Appendix B). Daily water temperatures dropped slightly as the study progressed, with high and low temperatures respectively averaging approximately 32.5°C and 28.5°C at the beginning of the study and 29.5°C and 25.5°C by the end of the study. Occasional variation in temperatures was the result of rainfall from tropical weather systems, including a hurricane that occurred during week 8 (Appendix C).

DISCUSSION

In this study, we assessed the capacity of juvenile green turtles for growth compensation. Experiments such as ours require the comparison of growth in previously food-restricted and continuously ad libitum-fed animals. However, this approach makes it difficult to

TABLE 1. Body composition of juvenile green turtles (*Chelonia mydas*) at 0, 5, and 12 weeks, expressed as mean (SE).

Group	Week	Water (g)	Organic matter (g)	Minerals (g)	Nitrogen (g)	Lipid (g)	Energy (kJ)
AL	0	28.0 (0.5)	5.4 (0.1)	0.91 (0.02)	0.75 (0.02)	1.27 (0.04)	130.6 (3.4)
AL	5	53.5^{A} (1.0)	11.4^{A} (1.0)	2.55^{A} (1.03)	1.76^{A} (1.02)	1.76 (1.09)	263.3^{A} (1.0)
R	5	55.3^{B} (1.0)	9.6^{B} (1.0)	2.29^{B} (1.03)	1.50^{B} (1.02)	1.41 (1.09)	220.3^{B} (1.0)
AL	12	101.3^{A} (1.0)	24.0^{A} (1.0)	5.87^{A} (1.03)	3.66^{A} (1.02)	4.11^{AB} (1.06)	566.2^{A} (1.0)
R-AL	12	102.1^{A} (1.0)	23.8^{A} (1.0)	5.30^{B} (1.02)	3.56^{A} (1.01)	4.48^{A} (1.04)	570.6^{A} (1.0)
R	12	104.9^{B} (1.0)	19.5^{B} (1.0)	5.46^{AB} (1.03)	2.95^{B} (1.02)	3.30^{B} (1.07)	458.6^{B} (1.0)

Notes: Groups are: AL, ad libitum for 12 weeks; R, food-restricted for 12 weeks; R-AL, food-restricted for five weeks and ad libitum for seven weeks. Data for week 0 represent actual means, whereas data for weeks 5 and 12 represent estimated marginal means (covariate = body mass excluding gut contents). Body composition data and body masses (without gut contents) were log-transformed prior to analysis. Within columns and time periods, values with different superscript letters are significantly different according to ANCOVA with a Bonferroni adjustment for multiple comparisons in week 12. Sample sizes: $n = 10$ turtles in each group at each sampling time.

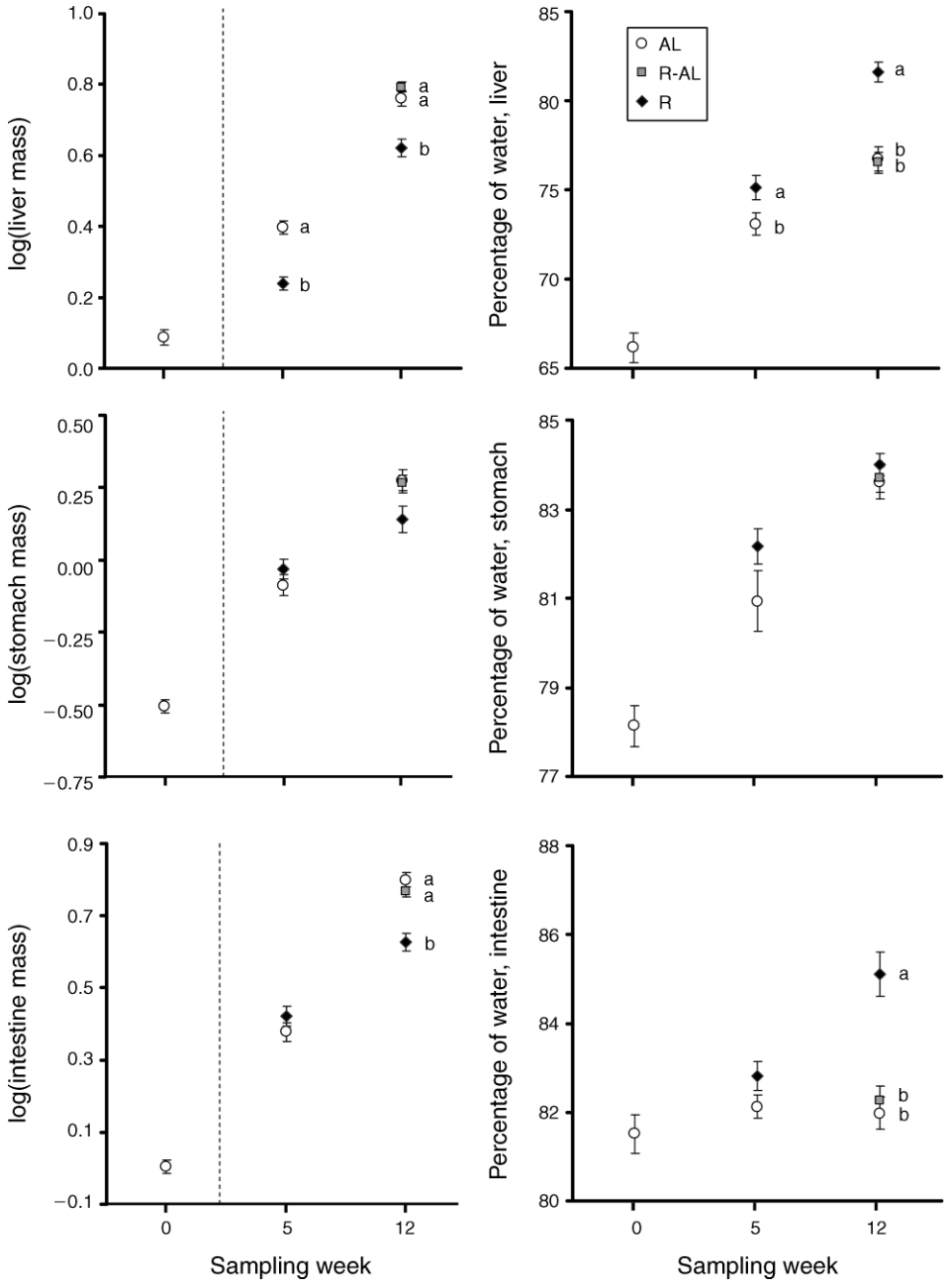

FIG. 4. Wet mass (left panels) and water content (right panels) of liver, stomach, and intestine from turtles at the end of weeks 0, 5, and 12 (mean ± SE). The dashed line in the left panels separates actual values in week 0 from estimated marginal means in weeks 5 and 12. For organ masses, different letters associated with data points indicate values that are significantly different ($P < 0.05$) within weeks according to ANCOVA [covariate = log(body mass excluding gut contents)] with a Bonferroni correction for multiple comparisons. For water content, different letters associated with data points indicate values that are significantly different ($P < 0.05$) within weeks according to ANOVA and Tukey's hsd post hoc test. Sample sizes: $n = 10$ turtles per group at each time point. Treatment groups are as in Fig. 1.

discriminate between accelerated growth and simple growth allometry because (1) growth rates vary with body size (Jobling 1983, Harris 1999), and (2) the food restriction that facilitates subsequent CG causes body size divergence between restricted and non-restricted individuals. Because CG is defined as faster growth in same-age animals (Bohman 1955, Wilson and Osbourn 1960), only comparisons within the same cohort at the same point in time are truly valid. Body size is therefore a confounding factor that must be addressed when analyzing CG data. Further, the proper body size covariate must be chosen to ensure that changes in size

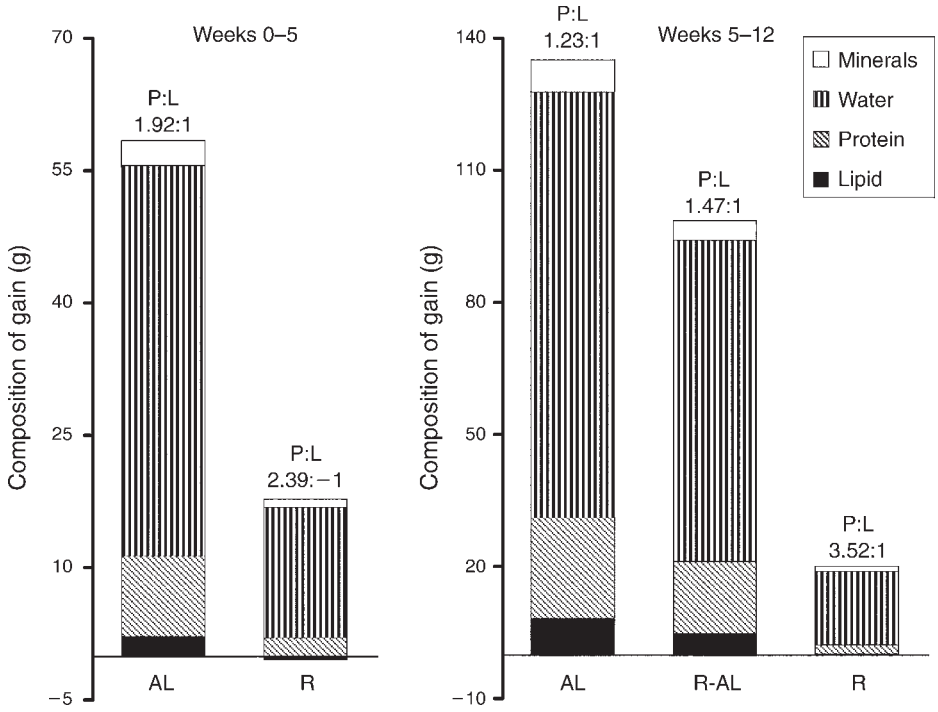

FIG. 5. Estimated composition of body mass gain (excluding gut contents) between weeks 0 and 5 (left) and between weeks 5 and 12 (right), calculated as the net change in mass of each component. Minerals, water, and lipid were determined empirically, and protein was estimated as the remainder of body mass. Sample sizes and treatment groups are the same as in Fig. 4. P:L is the ratio of calories allocated to protein gain to calories allocated to lipid gain. The negative denominator in the P:L ratio for R turtles between weeks 0 and 5 reflects net loss of lipid. Treatment groups are as in Fig. 1.

are not exaggerated by gut filling upon realimentation. For this reason, the assessment of length gain is preferable to that of mass gain.

By analyzing our data with body length as a covariate, we determined that growth of juvenile green turtles on a size-adjusted basis was indeed faster during recovery from food limitation than during continuous ad libitum feeding. This faster size-specific growth did not significantly diminish the divergence in mean body length between AL and R-AL turtles and therefore does not fit the standard definition of compensatory (or catch-up) growth. However, increased variation in individual growth rates with time in R-AL turtles led to some convergence of size ranges. Because mortality risk for juvenile sea turtles decreases as body size increases (Chaloupka and Limpus 2005), we expected the compensatory response to expedite progression of previously food-limited turtles through the vulnerable juvenile stage. Instead, we found that assimilated nutrients are preferentially allocated to restoring condition rather than to achieving a particular size-at-age trajectory when green turtles are recovering from food limitation.

Rapid restoration of body condition is common in hyperphagic animals (Metcalfe and Thorpe 1992, Morgan and Metcalfe 2001, Ali et al. 2003) because surplus nutrients can be routed into production after metabolic requirements have been fulfilled. Conversely, the composition of gain should be similar for animals with comparable intake, assuming no difference in allocation strategies. However, R-AL turtles in our study accelerated their protein deposition without becoming hyperphagic after the switch to ad libitum feeding. This shift in allocation is a potential mechanistic explanation for their transient fast growth, as protein deposition requires less energy per gram than fat deposition (Hornick et al. 2000).

Enhanced conversion efficiency and faster size-adjusted growth in R-AL turtles may also have resulted from a shift in their overall energy budget compared to AL turtles. Assimilatory organs such as the liver are energetically expensive to maintain (Hornick et al. 2000) and often shrink in the face of food restriction or starvation (Lee et al. 2002, Karasov et al. 2004). In our study, the liver was significantly smaller after five weeks of food restriction, and more prolonged food restriction was associated with smaller intestines as well. The decreased mass-specific metabolic costs associated with smaller visceral organs (Skalski et al. 2005, Cox and Secor 2007) coupled with a return to ad libitum feeding could have permitted a larger proportion of nutrients to be available for production in R-AL turtles (see also Yambayamba et al. 1996). An energy surplus also appears to have resulted from decreased investment in mineral mass, implying that decreased bone density may be a cost of accelerated growth.

Body composition was restored to AL levels before R-AL turtles achieved any measurable size compensation, suggesting that growth in green turtles is dictated more by condition than by overall body size. In fish, condition (namely adiposity) is thought to modulate appetite, thereby fueling a compensatory response during realimentation that ends once reserves are replenished (Bull and Metcalfe 1997, Johansen et al. 2001). The extent to which this lipostatic mechanism may regulate appetite and intake in green turtles could not be evaluated because size-adjusted adiposity did not differ among R and AL turtles. However, our inability to induce hyperphagia suggests either that green turtles do not adjust their intake in response to fat-free body composition or that ad libitum intake rates are already maximal because of physiological constraints (Speakman and Król 2005). The latter explanation is supported by evidence that juvenile loggerhead sea turtles (*Caretta caretta*) also do not increase ad libitum intake in response to nutrient dilution of their diet (McCauley and Bjorndal 1999).

Prioritizing condition over size could be adaptive in two senses. The capacity for rapid growth is more likely to evolve when the time available for achieving a size threshold is constrained (Arendt 1997, Metcalfe et al. 2002), as it is with seasonally dependent life-history transitions such as smolting (Schmitz 1995) and metamorphosis (Rowe and Ludwig 1991). In these scenarios, animals compensating for previous food limitation typically allocate more nutrients to growth and less to storage (Gurney et al. 2003, Stoks et al. 2006), so body size recovers but body condition does not. In the absence of such time constraints, funneling extra nutrients into storage rather than size may increase starvation resistance and therefore decrease the risk of mortality from future nutritional stresses (Owen-Smith 2004).

Alternatively, fast growth may be associated with performance and/or fitness costs (Blanckenhorn 2000, Metcalfe and Monaghan 2001) including delayed skeletal ossification (Arendt and Wilson 2000), weakened musculature (Christiansen et al. 1992), reduced locomotor performance (Álvarez and Metcalfe 2005), accelerated telomere degradation (Jennings et al. 1999), and decreased longevity (Olsson and Shine 2002). The proximate determinant of many of these costs could be the accumulation of cellular damage during rapid growth (Mangel and Munch 2005). Our results from another study suggest that cellular antioxidant potential of R-AL turtles was lower than that of AL turtles, at least in mitotically active tissue (Roark et al. 2009). If such costs place an upper limit on growth in green turtles, they would explain the transient and incomplete nature of the growth response we observed.

Our results provide insights into compensatory responses of non-teleost ectotherms, animals that have received relatively little attention in this regard. Juvenile green turtles that experience a period of nutritional stress respond after food availability improves by growing faster than expected for their size and by compensating for altered body condition. Accelerated size-specific growth results from enhanced conversion efficiency rather than hyperphagia, but this faster growth does not permit size compensation. Conversely, a nearly complete compensatory response to altered body composition (with the exception of mineral content) is achieved during the recovery period. These results suggest that (1) intake in juvenile green turtles is maximal when food is readily available, (2) growth rates of ad libitum-fed turtles are only mildly plastic in response to past nutritional history, perhaps because of inherent costs to fast growth, and (3) priority rules for nutrient allocation favor the attainment of an optimal condition rather than an optimal size. Nutritional setbacks experienced during the vulnerable juvenile stage could therefore have long-lasting consequences for wild turtles in terms of size-specific mortality risk, especially if intake and growth rates early in life entrain later growth trajectories (Madsen and Shine 2000). However, these risks may be partially mitigated by the potential benefits of maintaining sufficient body stores.

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APPENDIX A

Repeated-measures ANOVA results for weekly means of intake and body size in three groups of turtles over 12 weeks (*Ecological Archives* E090-178-A1).

APPENDIX B

Kruskal-Wallis test results for nutrient content of biweekly food samples (*Ecological Archives* E090-178-A2).

APPENDIX C

Daily water temperatures throughout the feeding trial (*Ecological Archives* E090-178-A3).