Introduction

Both longitudinal bone growth and periosteal bone apposition are affected by mechanical loading [1]. End loads applied to the rat ulna have been shown to suppress longitudinal bone growth [2]. Using the rat ulna model, we investigated the effect of loading on longitudinal and appositional bone growth by varying the magnitude and mode (static vs. dynamic) of applied load.

Materials and Methods

Twenty-nine growing male Sprague Dawley rats were divided randomly into 3 experimental groups (group $n = 9–10$). All rats were subjected to a daily 10-minute session of axially-applied compressive end-loading of the right ulna while under general anesthesia (ethyl-ether inhalation). The ulnar loading model is a nonsurgical preparation that applies mechanical force through the olecranon and flexed carpus [3]. The natural curvature of the diaphysis translates most of the axial load into a bending moment that puts the lateral cortex in tension and the medial cortex in compression—a strain distribution similar to that resulting from normal ambulation in vivo [2]. The 3 loading groups differed in the nature (static vs. dynamic) and magnitude (17N vs. 8.5N) of the forces applied. Group 1 received 1200 cycles of dynamic loading, applied as a haversine waveform at a frequency of 2 Hz. The range of the load magnitude was monitored from a load cell and was equal to 17N. Group 2 was subjected to 10 minutes of static load at 17N. Force was applied and released at a rate approximating 2N sec$^{-1}$. Group 3 received 10 minutes of static load at 8.5N. Force was applied and released at a rate approximating 1N sec$^{-1}$. Left ulnae were not loaded and served as controls for the right ulna. All animals were allowed normal cage activity between the daily loading sessions.

Loading was administered on days 1–5 and 8–12 of the experimental period. On days 5 and 12, the rats were given an IP injection of flurochrome label (either calcein or alizarin) at a dose volume of 7 mg/kg. All animals were sacrificed on day 16, immediately after which the right and left ulnae were dissected out and measured for maximum proximo-distal length using digital calipers. Transverse thick sections (~70 µm) were prepared from the right and left diaphysis at a point 2 mm distal to midshaft. Primary histomorphometric data were collected from the endocortical and periosteal envelopes, and derived bone formation indices (BFR, MAR, MS) were calculated using standard formulae. To control for individuals differences in growth rates and hormonal influences, relative histomorphometric indices (rBFR, rMAR, rMS) and relative gross lengths (rL) were calculated by subtracting left ulna (nonloaded control) values from right ulna (experimental) values. Differences among group means were tested for significance by ANOVA, and followed up with Fisher’s PLSD post-hoc comparisons.

Results

Mean body mass increased by approximately 90 g over the experimental period (day 1 mean = 213 ±27 g; day 16 mean = 305 ±16 g).

Longitudinal Growth: Right ulnae were significantly shorter (paired $t$-test, $p < .001$) than left ulnae in all 3 groups (Figure 1). Post hoc tests revealed no difference in relative length (rL) between the 17N dynamic and the 17N static groups; however, both were significantly greater than the 8.5N group.

Appositional Growth: The periosteal and endocortical surfaces of the 17N dynamic group expressed significantly greater BFR and MAR in the right versus the left ulna, but no right–left difference was detected in MS (paired $t$-test, $p < .05$). The 2 static-load groups exhibited significant load-induced suppression of BFR and MAR on the periosteal surface, but static loads produced no detectable right–left difference in endocortical bone formation (Figure 2). Comparisons of mean relative BFR and MAR reveal significant differences (PLSD, $p < .05$), both periosteal and endocortical, between the dynamic group and both static groups.

Discussion

These data suggest that during growth, daily bouts of loading suppresses longitudinal bone growth significantly, in proportion to the peak load magnitude. Dynamic loading clearly has an anabolic effect on periosteal bone growth while static loading can suppress periosteal apposition. Dynamic, but not static, loading has stimulatory effect on endocortical bone formation.

The effects of mechanical forces on longitudinal and appositional bone growth appear to work through different mechanisms: load-related suppression of longitudinal growth was proportional to peak load magnitude and not the dynamic nature of the loading, whereas appositional bone growth on the periosteal and endocortical surfaces was stimulated by dynamic loading only.