Research Square

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Comparative study of nucleus accumbens shell and core lesions in relation to body weight in healthy and activity-based anorexia female Wistar rats

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Abstract

Background. The nucleus accumbens (NAc) is a central node of the brain reward system and participates in feeding and motivated behaviour. Its two subregions, the shell and the core, differ neurochemically and may contribute differently to weight regulation. We compared the effects of bilateral radiofrequency lesions of the NAc shell versus core on body weight in healthy rats and in rats submitted to an activity-based anorexia (ABA) protocol.

Methods. Forty-eight female Wistar rats (250–400 g) were assigned to a healthy group (Group A, ad libitum feeding) or an anorexia group (Group B, ABA protocol). Each group was subdivided by lesion target: shell (subgroups A1, B1) or core (A2, B2). On day 14, bilateral radiofrequency ablation of the NAc was performed under stereotaxic guidance; no sham-operated group was included. Body weight was recorded at baseline (day 0), at surgery (day 14) and before euthanasia (day 28), and percentage change was analysed separately for the pre-lesion (days 0–14) and post-lesion (days 14–28) phases. Wheel-running activity was monitored at the cage level (four animals per cage) as an exploratory measure. Non-parametric tests (Friedman, Wilcoxon, Mann–Whitney) were used, with a 5% significance level.

Results. In healthy rats, weight increased before surgery in both subgroups. After core lesions, weight continued to rise (median change days 14–28, +1.9%; Wilcoxon $p = 0.016$), whereas after shell lesions the response was heterogeneous (median +0.8%, interquartile range −14.3 to +5.7%; $p = 0.70$), with four of ten animals losing 12–23% of their weight. In ABA rats, weight fell before surgery in both subgroups (median −21.0% and −11.7%); after shell lesions it continued to fall (median −5.0%; $p = 0.007$), whereas after core lesions it stabilised (median +0.6%; $p = 0.65$). Direct shell-versus-core comparisons of post-lesion weight change were not statistically significant (Mann–Whitney: healthy, $p = 0.67$; ABA, $p = 0.31$). Wheel-running data, recorded at the cage level, are reported descriptively only.

Conclusions. Post-lesion weight trajectories differed in direction between shell and core targets in both feeding conditions, but the between-subregion differences did not reach statistical significance and no sham-lesion group was included. The findings are hypothesis-generating and support a sham-controlled, histologically verified comparison of NAc subregions in weight regulation.

Plain English summary

Anorexia nervosa is a severe eating disorder in which people eat very little, fear weight gain, and often exercise excessively. The nucleus accumbens is a small brain region that helps control reward, motivation and eating, and it has two parts: the shell and the core. To understand how each part contributes to body weight, we studied female rats. Some rats ate freely and others followed an “activity-based anorexia” procedure that combines a short daily feeding window with free access to a running wheel, reproducing key features of the human disease. Two weeks into the study we made small lesions in either the shell or the core of the nucleus accumbens and followed the animals’ body weight for two more weeks. Weight changed differently after shell and core lesions: in healthy rats it kept rising after core lesions but was variable after shell lesions, and in the anorexia procedure it kept falling after shell

lesions but levelled off after core lesions. Because the study was small and had no sham-operated comparison group, these differences were not statistically conclusive. They nevertheless add to our understanding of how brain reward circuitry may contribute to weight regulation and point to what a larger, better-controlled study should test.

Background

Eating disorders are disabling and potentially deadly mental disorders characterised by disturbed eating patterns and marked concern about weight or body shape [1]. Anorexia nervosa (AN) is defined by restricted energy intake relative to requirements, an intense fear of weight gain, and a disturbance in the way body weight or shape is experienced [2]. AN carries one of the highest mortality rates of any psychiatric disorder; a meta-analysis of 36 studies reported a standardized mortality ratio of about 5.9, with one in five deaths attributable to suicide [3].

A prominent behavioural feature of AN is excessive physical activity. This is captured in rodents by the activity-based anorexia (ABA) model, in which restricted access to food combined with free access to a running wheel produces progressive weight loss, hyperactivity and, if uninterrupted, severe self-starvation, reproducing central features of the human disorder [4, 5].

The nucleus accumbens (NAc), in the ventral striatum, is a pivotal node of the brain reward system and contributes to motivation, reward processing and the regulation of food intake [6]. It is classically divided into a shell and a core, which differ in cytoarchitecture, connectivity and neurochemistry [6, 7]. Dopamine and serotonin signalling within accumbal and related striatal circuits shape the hedonic and motivational aspects of feeding, and imaging studies indicate that altered ventral and dorsal circuit function—possibly related to altered serotonin and dopamine metabolism—contributes to the symptoms of AN [8].

Because the shell and core differ functionally, selective lesions of each subregion may reveal distinct contributions to weight regulation. Targeted NAc interventions—ablative or neuromodulatory—have been explored as research tools and, in severe treatment-refractory AN, as candidate therapies: the NAc is among the most frequently reported deep brain stimulation targets for AN [9, 10], and NAc/ventral-capsule stimulation has an established investigational role in related disorders [11]. Direct comparisons of shell versus core lesions on weight regulation within an ABA framework, however, remain scarce.

The aim of this study was to compare body-weight changes following bilateral radiofrequency lesions of the NAc shell versus core in healthy female Wistar rats and in rats submitted to the ABA protocol, using the pre-lesion phase as a within-animal reference. Wheel-running activity was recorded as an exploratory, cage-level measure.

Methods

Animals

Forty-eight female Wistar rats (Rattus norvegicus), 250–400 g and aged 6–8 months, were obtained from the FCMS–PUC-SP animal facility. Animals were group-housed (four per cage) in polypropylene cages under controlled humidity and a 12-h light/dark cycle (lights on 07:00–19:00) at 21 ± 1°C, with ad libitum access to standard chow (Nuvilab®) and water. All procedures were approved by the Ethics Committee on Animal Use (CEUA) of PUC-SP (Process No. 2020/126) and complied with Brazilian National Council for the Control of Animal Experimentation (CONCEA) guidelines. Reporting follows the ARRIVE 2.0 guidelines, and a completed checklist is provided as an additional file.

Animals were assigned to a healthy group (Group A, ad libitum feeding) and an anorexia group (Group B, ABA protocol). Each group was subdivided by lesion target: shell (subgroups A1, B1) or core (A2, B2), with a planned n = 12 per subgroup (a convenience sample; no a priori power calculation was performed). Animals were allocated to subgroups sequentially (four per cage) rather than by a formal randomisation procedure, and no blinding of surgery or outcome assessment was used; both are acknowledged as limitations. Inclusion criteria were healthy females aged 6–8 months weighing 250–400 g. Predefined exclusion and humane-endpoint criteria were body-weight loss exceeding 25% relative to day 0, or body temperature below 33°C; body weight and clinical condition were monitored daily throughout the study.

Activity-based anorexia (ABA) protocol

Both groups were housed in cages fitted with a running wheel throughout the 28-day study. Group A had ad libitum access to standard rodent chow. Group B followed the ABA protocol: chow was available for a single 2-h daily feeding window (11:30–13:30). After each feeding period, the animals and the remaining chow were weighed. The protocol ran for the 28-day study period.

Surgical procedure

On day 14, bilateral radiofrequency ablation of the NAc was performed under stereotaxic guidance (EFF-331 device). Anaesthesia used ketamine (90 mg/kg) and xylazine (5 mg/kg) with local lidocaine (2%); carprofen (5–15 mg/kg) provided peri- and post-operative analgesia. Coordinates were taken from the rat brain atlas of Paxinos and Watson [12]. Target coordinates (from bregma) were: shell — anteroposterior + 2.04 mm, mediolateral ± 1.72 mm, dorsoventral + 0.90 mm; core — anteroposterior + 2.04 mm, mediolateral ± 1.64 mm, dorsoventral + 1.20 mm. The stereotaxic ablation procedure has been described in detail elsewhere [16].

The skull was drilled at the calculated coordinates using a saline-cooled dental drill, and a custom radiofrequency electrode with a 4-mm exposed (active) tip was lowered bilaterally; lesions were made at 60°C for 20 s. Incisions were closed with 3 − 0 nylon and topical 2% lidocaine gel was applied. Animals were individually housed for 12 h for recovery and then returned to group housing. Three animals did not survive surgery (two in A1, one in B1), reducing those subgroups to n = 10 (A1) and n = 11 (B1); these

were treated as exclusions under the criteria above. Euthanasia was performed on day 28 by halothane inhalation (5 mL); brains were removed and stored in 10% formalin and remain available for histological confirmation if required.

Lesion sites were defined by stereotaxic coordinates. Histological verification (e.g., cresyl-violet/Nissl staining) was not performed in the present study; the brains stored in 10% formalin remain available for confirmation of shell versus core targeting and of damage to adjacent structures should this be required (see Limitations).

Activity monitoring

Cages were equipped with a running wheel connected to a tachometer that recorded wheel rotations over 24-h periods. Because four animals shared each cage, wheel-running was recorded at the cage level and cannot be attributed to individual animals. Activity data are therefore reported descriptively and treated as exploratory; no inferential statistics assuming animal-level independence were applied to the activity measure (see Limitations).

Statistical analysis

Body weight was analysed at the individual-animal level from the day 0/14/28 records. Because surgery was performed on day 14, weight change was expressed as percentage change from the start of each phase—pre-lesion (day 0 to 14) and post-lesion (day 14 to 28)—and over the whole study (day 0 to 28); the pre-lesion phase served as a within-animal reference in the absence of a sham group. Given the small sample size and non-normal distribution, non-parametric tests were used: Friedman's analysis of variance for the three time points, the Wilcoxon signed-rank test for paired within-phase comparisons, and the Mann–Whitney test for shell-versus-core comparisons of percentage change and of baseline weight. Data are presented as median (interquartile range, IQR). Change in body weight from the pre-lesion value was the primary outcome; cage-level wheel-running activity was a secondary/exploratory measure. The significance level was 5%; no correction for multiple comparisons was applied and all results are interpreted as exploratory. Friedman analyses were performed in BioEstat 5.0 and phase-wise and between-subgroup analyses in Python 3 (SciPy).

Results

Body weight — healthy group (A1, A2)

Table 1 summarises body weight at each time point and the percentage change in each phase. Before surgery (days 0–14), body weight increased in both healthy subgroups (median + 2.8% in A1 and + 3.9% in A2; Wilcoxon $p = 0.002$ and $p < 0.001$). After core lesions (A2, n = 12), weight continued to increase (median change days 14–28, + 1.9%, IQR + 0.5 to + 4.0%; Wilcoxon $p = 0.016$; Friedman across days 0/14/28, $\chi^2 = 16.63$, $p = 0.0002$). After shell lesions (A1, n = 10) the response was heterogeneous: the

median change was + 0.8% (IQR − 14.3 to + 5.7%; p = 0.70), but four of ten animals lost 12–23% of their day-14 weight while the remaining six gained 3–23%, so that weight at day 28 did not differ from baseline (median 266 vs 289 g; p = 0.85; Friedman $\chi^2 = 6.20$, p = 0.045). The direct shell-versus-core comparison of post-lesion percentage change was not statistically significant (Mann–Whitney p = 0.67), nor was the comparison of total change from day 0 to day 28 (p = 0.41). The two subgroups differed in baseline weight (median 266 vs 278 g; p = 0.024), which is why percentage change rather than absolute weight was used for comparisons.

Body weight — anorexia group (B1, B2)

Before surgery, under the ABA protocol alone, body weight fell markedly in both subgroups (median change days 0–14, − 21.0% in B1 and − 11.7% in B2; Wilcoxon p = 0.042 and p = 0.002). After shell lesions (B1, n = 11) weight continued to fall (median change days 14–28, − 5.0%, IQR − 6.5 to − 0.9%; p = 0.007; 10 of 11 animals lost weight), whereas after core lesions (B2, n = 12) weight stabilised (median + 0.6%, IQR − 9.2 to + 4.2%; p = 0.65; 5 of 12 lost weight). Across the three time points the decrease was significant in both subgroups (Friedman $\chi^2 = 14.73$, p = 0.0006 in B1; $\chi^2 = 10.34$, p = 0.006 in B2). The shell-versus-core comparison of post-lesion change was not statistically significant (Mann–Whitney p = 0.31; total change days 0–28, p = 0.10). As in the healthy group, baseline weight differed between subgroups (median 271 vs 327 g; p = 0.016). Weight values were taken from the day 0/14/28 dataset, which is consistent across all archived versions of the source records.

Table 1

Body weight and percentage weight change by subgroup and study phase. Values are median (interquartile range). Surgery (bilateral radiofrequency lesion) was performed on day 14; days 0–14 = pre-lesion phase, days 14–28 = post-lesion phase. † Wilcoxon signed-rank test $p < 0.05$ within phase (see text for exact values). Friedman test across days 0, 14 and 28. Shell-versus-core comparisons of Δ% (Mann–Whitney) were not significant in any phase (all $p \geq 0.10$).

Subgroup	n	Day 0 (g)	Day 14 (g)	Day 28 (g)	Δ% days 0–14	Δ% days 14–28	Δ% days 0–28	Friedman χ^2 (p)
A1 (healthy, shell)	10	266 (263–270)	276 (271–280)	289 (225–294)	+ 2.8 (+ 2.6 to + 3.0)†	+ 0.8 (− 14.3 to + 5.7)	+ 3.6 (− 12.0 to + 8.5)	6.20 (0.045)
A2 (healthy, core)	12	278 (268–293)	296 (280–304)	304 (293–307)	+ 3.9 (+ 2.2 to + 4.7)†	+ 1.9 (+ 0.5 to + 4.0)†	+ 5.8 (+ 4.1 to + 8.5)†	16.63 (0.0002)
B1 (ABA, shell)	11	271 (254–310)	242 (213–265)	230 (201–264)	−21.0 (− 23.8 to − 7.8)†	−5.0 (− 6.5 to − 0.9)†	−21.3 (− 27.4 to − 13.2)†	14.73 (0.0006)
B2 (ABA, core)	12	327 (300–363)	299 (262–315)	280 (262–319)	−11.7 (− 15.8 to − 10.2)†	+ 0.6 (− 9.2 to + 4.2)	−13.4 (− 16.8 to − 9.6)†	10.34 (0.006)

Wheel-running activity (exploratory, cage-level)

Wheel-running was recorded per cage (four animals per cage) and is reported descriptively. Because these measures are not independent at the animal level, they are presented as exploratory observations only and were not subjected to animal-level inferential testing (see Limitations). Cage-level wheel-running (n = 3–4 cages per subgroup) is shown in Supplementary Figure S1; because of very high inter-cage variability and the small number of cages, no directional conclusion is drawn from the activity data.

Discussion

In this exploratory comparison, post-lesion body-weight trajectories differed in direction between NAc shell and core targets in both feeding conditions. In healthy rats, weight continued to rise after core lesions but became heterogeneous after shell lesions, with four of ten animals losing weight. Under the ABA protocol, weight continued to fall after shell lesions but stabilised after core lesions. However, the direct shell-versus-core comparisons were not statistically significant and, in the absence of a sham-lesion group, the within-subgroup changes cannot be separated from the natural course of each feeding condition—weight gain in ad libitum-fed adult females and weight loss under ABA—or from non-specific effects of surgery. The most defensible reading is therefore that shell and core lesions were followed by different patterns of post-lesion weight change, without proof of a subregion-specific effect.

These observations are consistent with a role of the NAc—and particularly its core subregion—in weight regulation through reward-related mechanisms [6, 8]. The mesolimbic dopaminergic projection from the ventral tegmental area to the NAc is central to the hedonic evaluation of food, and disruption of accumbal signalling can alter feeding and body weight [6]. We emphasise that this study measured body weight and cage-level activity only; it did not assess neurotransmitter levels, receptor expression or neural circuitry, so any neurochemical interpretation is an extrapolation from the literature rather than a direct finding.

A differential involvement of the two subregions is biologically plausible given their distinct connectivity and neurochemistry [6, 7]. The response after shell lesions—weight loss in a subset of healthy animals and continued loss in ABA animals—is, however, difficult to reconcile with the classical observation that pharmacological inhibition (GABA-agonist microinjection) of the medial NAc shell elicits robust feeding in satiated rats [13, 14], and may instead reflect variable lesion placement, lesion size relative to the subregion, or extension into adjacent structures. In the absence of histological confirmation, subregion-specific interpretations must remain provisional—particularly because the target labelled “shell” was placed more laterally and dorsally than the “core” target, which is atypical relative to standard atlas descriptions and must be reconciled with histology [12]. The clinical relevance of accumbal targeting is underscored by the growing, though still investigational, use of NAc deep brain stimulation in treatment-refractory AN [9, 10, 11].

Limitations

Several limitations apply. First, no sham-operated or unlesioned group was included in either feeding condition; the pre-lesion phase served as a within-animal reference, but changes after surgery cannot be attributed to the lesion rather than to the natural course of each condition or to surgery itself. Second, lesion sites were defined by stereotaxic coordinates without histological verification; the brains were preserved in 10% formalin and confirmation of shell versus core targeting and of the extent of damage to adjacent structures could be obtained if required. Third, wheel-running activity was recorded at the cage level (four animals per cage) and cannot be attributed to individual animals; activity is therefore reported as exploratory only, and any animal-level statistical test of activity would violate the assumption of independence (pseudoreplication) and is not reported. Fourth, the sample size was modest, no a priori power calculation was performed, allocation was sequential rather than randomised, and shell and core subgroups differed in baseline weight; analyses therefore relied on within-animal percentage change, and no correction for multiple comparisons was applied. Fifth, only female rats aged 6–8 months were studied; the oestrous cycle was not monitored, and adult animals are generally less susceptible to ABA than adolescents [15], which may account for the moderate weight loss observed over 28 days. These limitations constrain the strength of the conclusions, which should be regarded as hypothesis-generating.

Conclusions

In female Wistar rats, post-lesion body-weight trajectories differed in direction between NAc shell and core targets in both healthy and ABA conditions—continued gain after core versus heterogeneous change after shell lesions in healthy animals, and continued loss after shell versus stabilisation after core lesions in ABA animals—but the between-subregion differences were not statistically significant and no sham group was included. These exploratory findings are compatible with a subregion-specific contribution of the NAc to weight regulation and support a sham-controlled, histologically verified comparison of accumbal subregions in the context of eating disorders.

Abbreviations

ABA
activity-based anorexia
AN
anorexia nervosa
CEUA
Ethics Committee on Animal Use
CONCEA
National Council for the Control of Animal Experimentation
DA
dopamine
NAc
nucleus accumbens
SEM
standard error of the mean
5-HT
5-hydroxytryptamine (serotonin)
VTA
ventral tegmental area.

Declarations

Ethics approval and consent to participate

All procedures were approved by the Ethics Committee on Animal Use (CEUA) of PUC-SP (Process No. 2020/126) and complied with CONCEA guidelines.

Consent for publication

Not applicable.

Availability of data and materials

The individual-animal body-weight data and cage-level wheel-running data generated and analysed during the current study are provided as Additional file 2 (Supplementary_data_raw.xlsx); further materials are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

LC: conceptualization, methodology, investigation (stereotaxic surgery and data collection), and writing — original draft. RVGV, FN and CSM: investigation (surgical procedures). YJ and NFN: formal analysis (statistical analysis). WMGF: supervision and writing — review and editing. PHAR and VKS: writing — review and editing (English-language preparation of the manuscript). VBC: data curation, formal analysis, visualization (figures), manuscript formatting, and writing — review and editing. PHPA: supervision and writing — review and editing. All authors read and approved the final manuscript.

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Figures

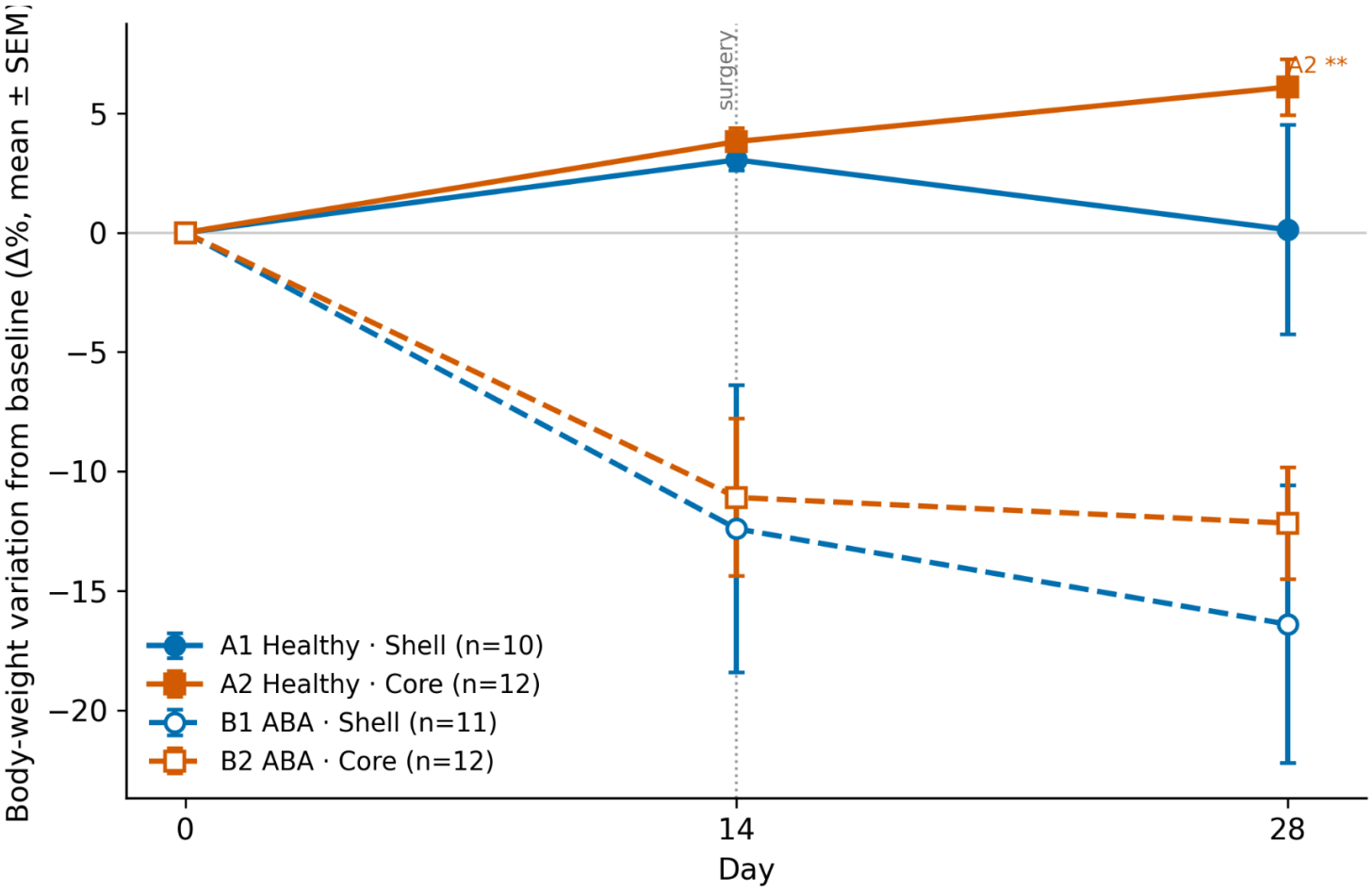


Figure 1

Body-weight variation from baseline (Δ%, mean ± SEM) across days 0, 14 and 28 in healthy (Group A, solid lines) and activity-based anorexia (Group B, dashed lines) female Wistar rats, following bilateral nucleus accumbens shell (A1, B1; blue) or core (A2, B2; orange) lesions. Surgery was performed on day 14 (dotted line). Within-subgroup Friedman tests: A2, $\chi^2 = 16.63$, $p = 0.0002$ (**); A1, $\chi^2 = 6.20$, $p = 0.045$; B1, $\chi^2 = 14.73$, $p = 0.0006$; B2, $\chi^2 = 10.34$, $p = 0.006$. Shell-versus-core comparisons of post-lesion (days 14–28) percentage change were not statistically significant (Mann–Whitney: Group A, $p = 0.67$; Group B, $p = 0.31$). Sample size per subgroup as indicated in the legend.