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12 days of altitude exposure at 1800m does not increase resting metabolic rate in elite rowers.

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1. Title Page

Title of the article: 12 days of altitude exposure at 1800m does not increase resting metabolic rate in elite rowers.

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2. Abstract

Four elite rowers completed a twelve-day altitude training camp living at 1800m, and training at 1800m and 915m, to assess changes in resting metabolic rate (RMR). RMR and body composition were assessed PRE and POST-camp. Downward trends in RMR and body composition were observed post-altitude: absolute RMR (percent change: -5.2%), relative RMR (-4.6%), body mass (-1.2%), and fat mass (-4.1%), likely related to the hypoxic stimulus and an imbalance between training load and energy intake.

Keywords: Hypoxia, basal metabolic rate, body composition, endurance athletes, energy availability, RMR, training load.

3. Text

Introduction

Altitude exposure is a common training stimulus in athletes seeking to enhance endurance performance, but has also received interest from a clinical perspective. Specifically, high (3000–5500m) and extreme (>5500m) altitudes (Bärtsch and Saltin, 2008) have been utilized as a therapeutic intervention to reduce body mass and cardio-metabolic risk factors in obese populations (Wee and Climstein, 2015); possibly related to hormone secretion, neurotransmitters involved in energy balance and increased resting metabolic rate (RMR) (Hamad and Travis, 2006). RMR is the minimum energy required to maintain the body's basic functions at rest, and can provide an indication of energy availability (EA), which, if low may impair physiological function, increase risk of illness and injury, and decrease an athlete's ability to adapt to the training prescribed (Mountjoy et al., 2014).

A loss of body mass is commonly reported at high altitudes (Hamad and Travis, 2006), but the extent of the loss, and any effects on training in athletes who frequent lower elevations are yet to be quantified. RMR is largely dependent on lean mass, thus any changes in body composition as a result of altitude exposure may affect RMR and EA, consequently impacting training and performance goals. Previous research from our group found significant increases in RMR following three weeks of continuous altitude exposure at 2200m in highly-trained distance runners (Woods et al., 2016b), however such elevations are often impractical in sports such as rowing. A lack of access to appropriate on-water training venues, and logistical complications

associated with travel and equipment transportation make naturally lower altitudes a practical alternative for training in these athletes, but it is unknown whether increases in RMR are induced at lower elevations.

The present case study sought to investigate whether RMR increases with twelve days of classical altitude training at 1800m in a group of four elite rowers preparing for the Rio 2016 Olympic Games.

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Method

Study Design

A case study was performed in conjunction with a pre-competition altitude training camp for elite male and female rowers. Both the Australian Institute of Sport Human Ethics Committee and the University of Canberra Human Research Ethics Committee approved the study. All participants provided written informed consent prior to involvement. For twelve consecutive days, athletes lived at Perisher, New South Wales, Australia (1800m), and undertook a combination of ergometer training sessions at Perisher, and on-water training in Jindabyne, New South Wales, Australia (915m), with an hypoxic exposure of $\sim 18\text{--}20$ h.day⁻¹. RMR and body composition were assessed PRE and POST-camp. Training and wellbeing were monitored throughout.

Participants

Four elite rowers [n=2 male (mean \pm SD height, body mass: 191 \pm 4cm, 94.9 \pm 2.8kg) and n=2 female (177 \pm 8cm, 76.3 \pm 2.3kg)] aged 21–28 years participated in the study. All athletes were selected to the 2015 Australian Rowing Team and categorized as ‘medal potential’ for the Rio 2016 Olympics. Due to the unique and highly applied nature of the study, athletes were unable to be pair-matched with a control group, nor assessed using an alternate study design.

RMR Measurement

RMR was assessed the day prior to, and the day after returning from Perisher using the Douglas Bag method of indirect calorimetry, which has been described previously

(Woods et al., 2016a). All athletes were overnight rested and fasted, and abstained from physical activity for at least eight hours prior to all measurements. Minute ventilation [$V_{E(STPD)}$] was assessed for each expirate collection. Typical error (TE) for the Douglas Bag method of RMR measurement in our hands is 286.8kJ, or 4.3% [90% confidence limits (CL): 3.1–7.2%] within days, and 455.3kJ or 6.6% (90% CL: 4.8–11.1%) between days, which compares favourably with other researchers (Compher et al., 2006).

Body Composition

Body composition was assessed immediately following each RMR measurement via Dual-Energy X-Ray Densitometry (DXA; GE Lunar Prodigy, GE Healthcare Asia-Pacific). Each DXA scan provided an assessment of fat mass, lean mass and bone mineral content (BMC). Fat-free mass (FFM) was calculated as lean mass plus BMC. Radiation safety approval was provided by the Radiation Safety Committee at the John James Hospital, Canberra. Athletes provided a urine sample at first void for assessment of urine-specific gravity (USG) from digital hand-held refractometer (ATAGO, USA).

Training and Wellness Monitoring

On-water sessions were monitored for duration, distance, velocity and stroke rate from boat-mounted GPS units (Catapult Sports, VIC, Australia). Ergometer (Concept 2 Model D, VIC, Australia) sessions during the camp were monitored for duration, distance, power output, heart rate, rating of perceived exertion (RPE, 1-10 Borg Scale (Borg, 1970) and blood lactate concentration (BLa) via earlobe capillary sample (Lactate Pro2, Arkray, Japan) upon completion of each specified work set. Fatigue,

muscle soreness, stress, sleep quality and external load were assessed daily to provide an indication of wellbeing. Each component was rated on a Likert scale anchored from 1 (“Not at all”) to 5 (“Extremely”) using online software (Sportlyzer, Tartu, Estonia). Training load was assessed by Training Stress Score (TSS), a training load index taking into account the duration and intensity of the activity using either heart rate or power output, whereby 100 TSS points is equivalent to one-hour of exercise at an individual’s functional threshold power (FTP) (Halson, 2014, TrainingPeaks, 2012).

Data Analysis

Raw RMR and body composition data between PRE and POST was log-transformed to reduce potential bias from any error, with outcomes presented as the difference in means (%) and standardized mean difference (Cohen’s Effect Sizes, ES), with associated 90% confidence limits (CL). ES were calculated as the difference in mean divided by the between-subject SD where a small effect is >0.2 , moderate >0.6 and large >1.2 (Hopkins et al., 2009).

Results

Twelve days of altitude exposure at 1800m elicited small-to-moderate decreases in absolute RMR (mean \pm SD of difference: $-550\pm 385\text{kJ}\cdot\text{day}^{-1}$), relative RMR ($-1.5\pm 0.9\text{cal}\cdot\text{kg}\cdot\text{FFM}^{-1}$, equivalent to $-6.2\pm 3.7\text{kJ}\cdot\text{kg}\cdot\text{FFM}^{-1}$), body mass ($-1.1\pm 0.9\text{kg}$), and fat mass ($-0.45\pm 0.34\text{kg}$, Table 1). Fat-free mass ($-0.50\pm 1.05\text{kg}$), lean mass ($-0.53\pm 1.06\text{kg}$), $V_{E(\text{STPD})}$ ($-0.03\pm 0.79\text{L}\cdot\text{min}^{-1}$) and hydration status via USG ($-0.0003\pm 0.008\text{kg}\cdot\text{m}^3$) remained stable. Individual responses are presented in Figure 1.

Mean on-water session distance, duration, velocity and stroke rate for the camp were: $22367\pm 308\text{m}$, $111.8\pm 1.6\text{ min}$, $3.4\pm 0.1\text{m}\cdot\text{s}^{-1}$ and 17 ± 0 strokes-per-minute, respectively. The corresponding mean ergometer distance, duration, power output, heart rate, RPE and BLa were: $19,700\pm 50\text{m}$, $76.7\pm 2.7\text{ min}$, $228\pm 37\text{W}$, $151\pm 7\text{ bpm}$, 4 ± 0 and $1.9\pm 0.8\text{mmol}\cdot\text{L}^{-1}$, respectively. Whilst the training sessions were submaximal in their prescription, the ergometer sessions achieved a higher intensity being undertaken at 1800m, compared with 915m on-water. Total training time (inclusive of on-water and cross-training) was $\sim 26\text{ h}\cdot\text{week}^{-1}$, consisting of 12-15 individual sessions.

Weekly TSS for the group for the weeks prior to, during and following the camp is presented in Figure 2. Descriptive data from the wellbeing questionnaires for three out of the four athletes is presented in Figure 3.

Discussion

The main finding of the present study was that twelve days of hypoxic exposure at 1800m did not increase RMR in four elite rowers preparing for the 2016 Olympic Games. In contrast, small-to-moderate decreases in absolute and relative RMR and body composition variables were observed. The changes in RMR are close to the boundary of the technical error of the measurement, however, and so might not be meaningful.

The present findings are in contrast with our previous work reporting an increased RMR at 2200m in highly-trained distance runners (Woods et al., 2016b). Such disparities may relate not only to the differences in camp duration and elevation, but also the nature of the training (and the greater metabolic demands of rowing compared with running), the load imposed, and possibly, greater chemoreflex-mediated sympathoexcitation at higher elevations (Hansen and Sander, 2003). In the present study, despite each athlete completing the prescribed training without incident, increased fatigue and decreased wellbeing were reported. The athletes experienced a substantial increase in training load undertaken while at altitude, which was almost double the average for the five weeks prior to the altitude camp. RMR may thus have been subtly decreased as a safeguard mechanism to conserve energy and ensure basic physiological function could be maintained.

Hypoxia-induced variations in RMR are proposed to relate to cold exposure, endocrine regulation, changes in body composition, and ventilation rate (Hamad and Travis, 2006, Huang et al., 1984). In the present study, we were unable to investigate

endocrine markers but mean environmental temperature was 20.9°C, and ventilation remained stable between tests so it is unlikely these were influencing factors. Regarding body composition, the small-to-moderate decreases in body mass and fat mass may indicate negative energy balance. It is also plausible that, since RMR is principally dependent on lean mass, the observed decrease in both absolute and relative RMR was due to an energy-conservation mechanism to safeguard against any further reductions in lean mass, which would be problematic in this elite group of rowers due to its correlation with 2000m velocity (Cosgrove et al., 1999). Notably, the athletes reported an anecdotal loss in appetite and a decreased desire to eat post-training, but this was not explicitly measured. High-altitude exposure has been reported to influence prominent hormones associated with appetite regulation and energy intake (Hamad and Travis, 2006), the notion of which was apparent in the runners at 2200m (Woods et al., 2016b), but there is little literature in athletes at lower elevations. It is probable that in the present cohort an insufficient energy intake, coupled with an increased training load, led to an (likely) acute reduction in EA, and subsequent reductions in RMR and body composition. Further investigation of the potential interplay between appetite, hormones and energy balance as a result of different hypoxic exposures is therefore warranted in future research.

Limitations

Projects of an applied nature are affected by the logistics of a high performance sport environment, and critically have to accommodate coach and athlete training plans. Therefore whilst care was taken to ensure appropriate scientific rigour in the present case study, we acknowledge there remain some limitations. Firstly, it was not possible to obtain a larger sample size or pair-match for a control group, primarily due to the

logistics of transporting the equipment and the number of boats required to accommodate individual hypoxic responses. It is reasonable to suggest that factors including a 'training camp-effect' were the primary cause for the present results, however data under review from our laboratory (Woods et al., 2017), coupled with anecdotal evidence from the Australian Rowing Team, demonstrate similar reductions in absolute (-4.8%) and relative RMR (-5.7%), body mass (-2.0%) and fat mass (-18.3%) after a minimum of 4 weeks intensified training. We are thus confident that the hypoxic-stimulus was primarily responsible for the present changes within the short timeframe. Finally, future investigations of this nature would benefit from detailed food diaries, appetite, haematological and performance data to elucidate the underlying mechanisms of physiological change.

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Conclusion

Athletes undertaking altitude training camps with concomitant increases in training load may suffer a reduction in RMR and body composition, and increased perceptions of fatigue, likely related to insufficient energy intake. Careful monitoring of RMR, training load, energy intake, appetite, body composition and wellbeing may safeguard against the risk of impaired physiological function associated with reduced EA.

The authors report no conflicts of interest associated with this manuscript.

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4. Acknowledgements

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6. Tables

Table 1: RMR and body composition PRE and POST altitude. Results are presented between PRE and POST as the difference in means (%) and Cohen's effect sizes (where a small effect is >0.2 , moderate >0.6 and large >1.2), with associated 90% confidence limits.

Outcome Measure	PRE	POST	Difference in Means (POST-PRE)		Effect Size (Cohen's d)	
			(%)	90% CL	d	90% CL
Absolute RMR (kJ.day ⁻¹)	9905 ± 2803	9355 ± 1753	-5.2	-8.6 to -1.7	-0.2	-0.3 to -0.1
Relative RMR (kJ.kg.FFM ⁻¹)	134.0 ± 13.1	127.7 ± 10.3	-4.6	-7.3 to -1.8	-0.4	-0.6 to -0.1
Relative RMR (cal.kg.FFM ⁻¹)	31.9 ± 3.1	30.4 ± 2.4	-4.6	-7.3 to -1.8	-0.4	-0.6 to -0.1
Body mass (kg)	85.6 ± 10.9	84.6 ± 10.5	-1.2	-2.4 to 0.0	-0.1	-0.2 to 0.0
Fat mass (kg)	11.68 ± 0.90	11.23 ± 1.20	-4.1	-7.9 to -0.1	-0.4	-0.8 to -0.01
Fat-free mass (kg)	73.36 ± 8.97	72.86 ± 8.71	-0.7	-2.5 to 1.2	-0.04	-0.2 to 0.1
Lean mass (kg)	69.89 ± 8.66	69.36 ± 8.41	-0.7	-2.7 to 1.2	-0.05	-0.2 to 0.1

7. Figure Captions

Figure 1: RMR and body composition responses to 12 days of altitude exposure at 1800m. Data are presented for A) absolute RMR, B) relative RMR, C) body mass, D) fat mass, E) fat-free mass and F) lean mass for each of n=4 athletes between PRE and POST. The closed circles indicate individual responses, with the group mean represented by the closed triangles.

Figure 2: Training Stress Score (TSS) for the five weeks prior, two weeks during and one week after the altitude camp, with mean TSS \pm SD for the athlete group per week.

Figure 3: Individual athlete wellbeing responses.. Data are presented for A) fatigue, B) muscle soreness, C) stress, D) sleep quality and E) external load, anchored from 1 (“Not at all”) to 5 (“Extremely”) for n=3 out of n=4 athletes between PRE and POST. The closed circles indicate individual responses, with the group mean represented by the closed triangles.

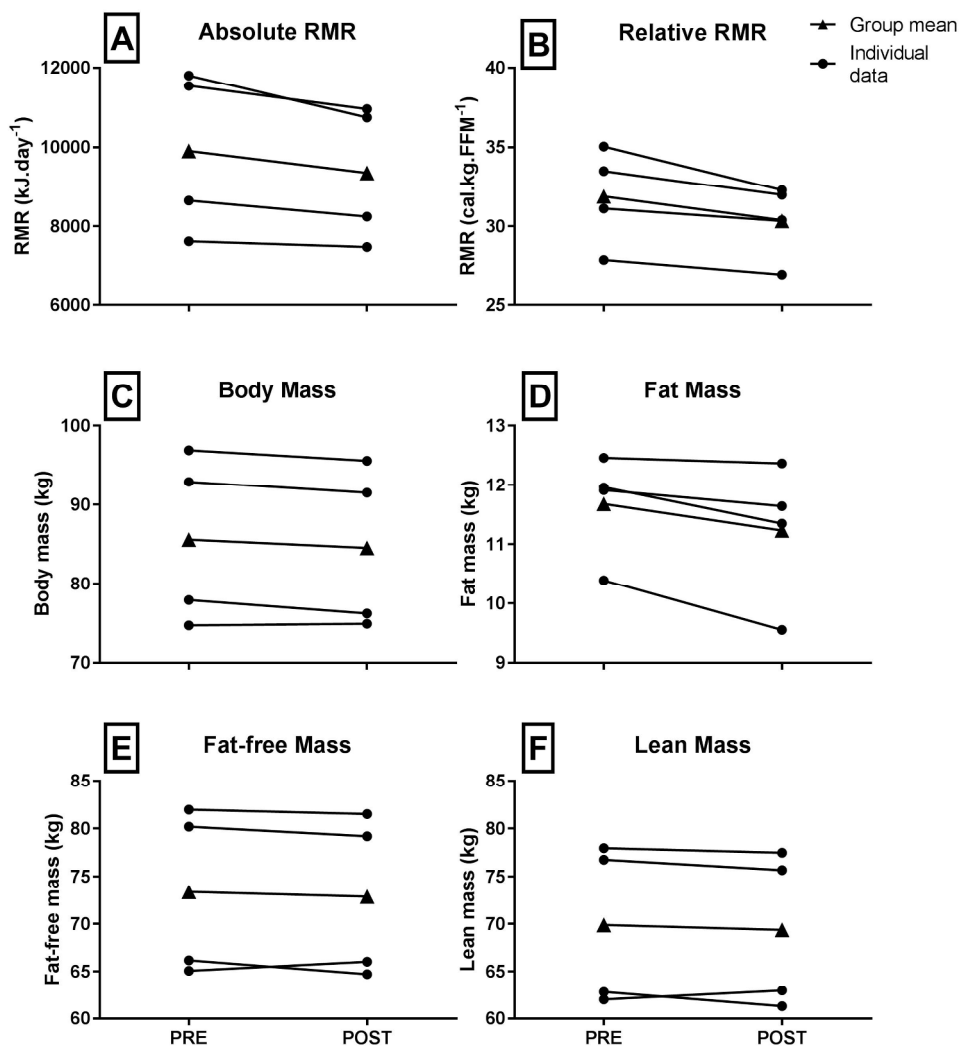


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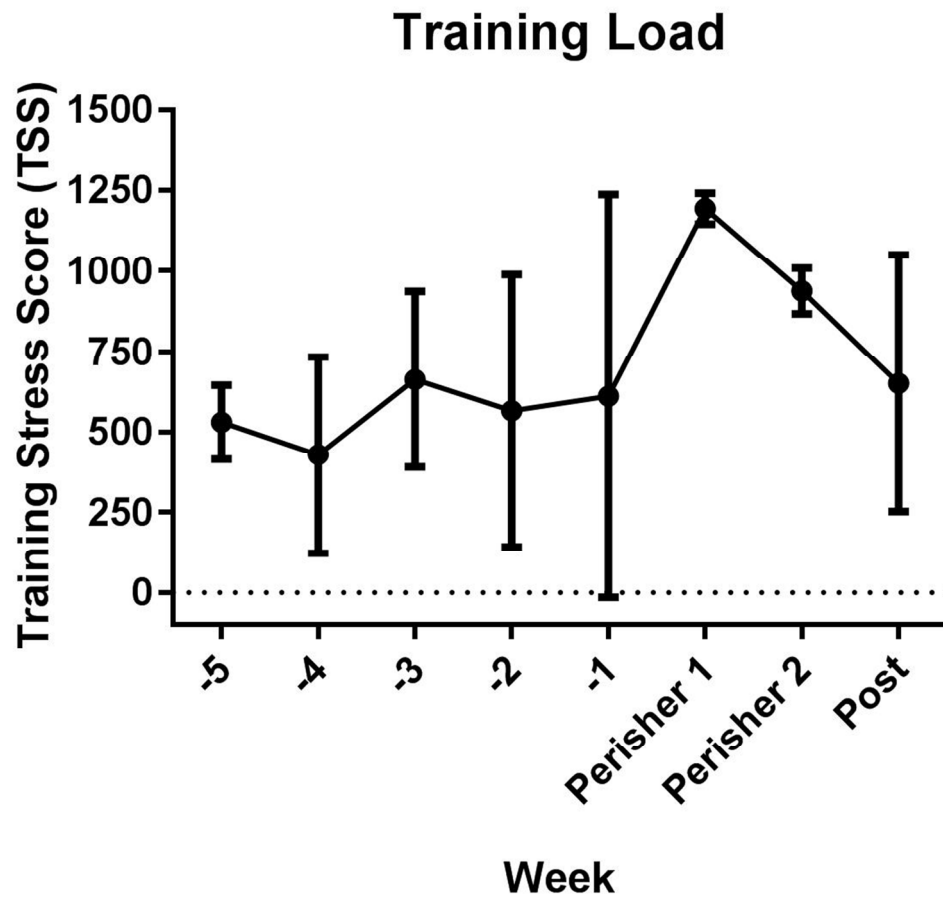


Figure 2: Training Stress Score (TSS) for the five weeks prior, two weeks during and one week after the altitude camp, with mean TSS \pm SD for the athlete group per week.

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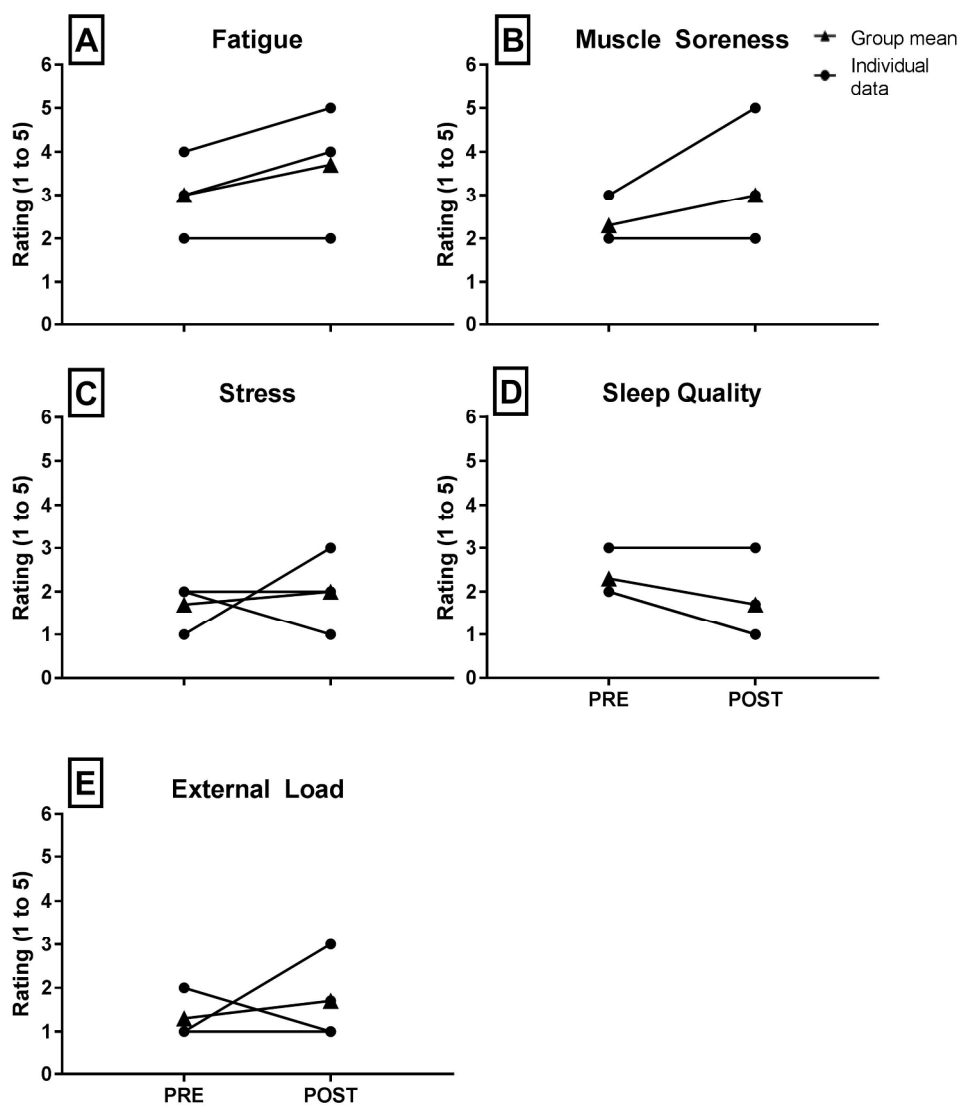


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