Creatine supplementation enhances corticomotor excitability and cognitive performance during oxygen deprivation

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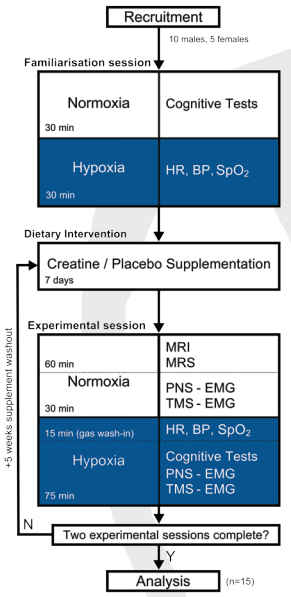
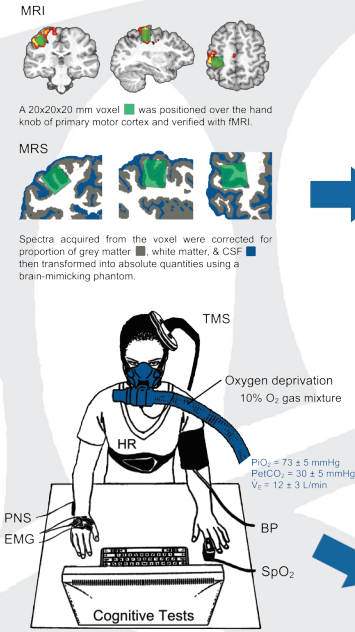
Background

Creatine is a naturally occurring compound involved in the buffering, transport and regulation of cellular energy. Creatine has been shown to be neuroprotective *in vitro* against anoxic/hypoxic damage[1-3]. The diet can be safely supplemented with creatine, but the utility of creatine to protect against energetic insult remains to be investigated in humans.

Aim: To assess the influence of dietary creatine monohydrate supplementation on brain function during oxygen deprivation.

Method





15 healthy volunteers completed a week-long, placebo controlled creatine monohydrate supplementation protocol within a double-blind, crossover design. Creatine concentration in the sensorimotor cortex was assessed using magnetic resonance spectroscopy. Cognition and corticomotor excitability were assessed using neurocognitive tests[4] and transcranial magnetic stimulation (TMS) at baseline and during a 90 min hypoxic gas breathing protocol.

Creatine concentratioin in the sensorimotor cortex increased by 10% with Creatine *vs*. Placebo supplementation

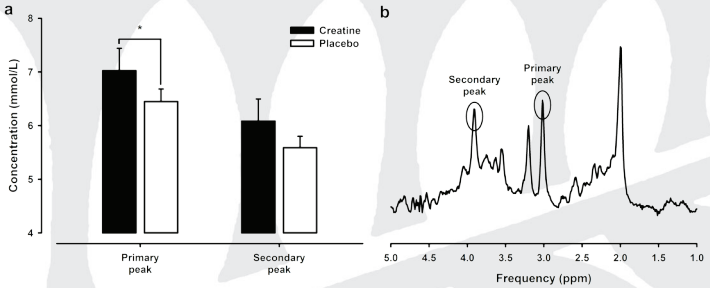


Figure 1. Neural creatine concentration after supplementation. a) Mean creatine concentration in the sensorimotor cortex after one week of Creatine and Placebo supplementation. * $p < 0.05$. b) An example spectra acquired at 3T from the sensorimotor cortex. Resonances at 3.069 ppm and 3.960 ppm represent the primary and secondary Cr + PCr peaks.

The oxygen deprivation protocol caused hypoxemia, reducing peripheral oxygen saturation by 19%

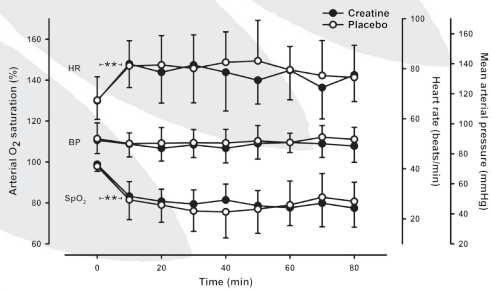


Figure 2. Cardiovascular responses to hypoxia. Mean arterial oxygen saturation (SpO_2), heart rate (HR) and blood pressure (BP) responses to hypoxia for Creatine and Placebo treatments. ** $p < 0.01$ for 0 min *vs*. > 0 min.

Results

Corticomotor excitability increased during hypoxia with Creatine supplementation, but not with Placebo

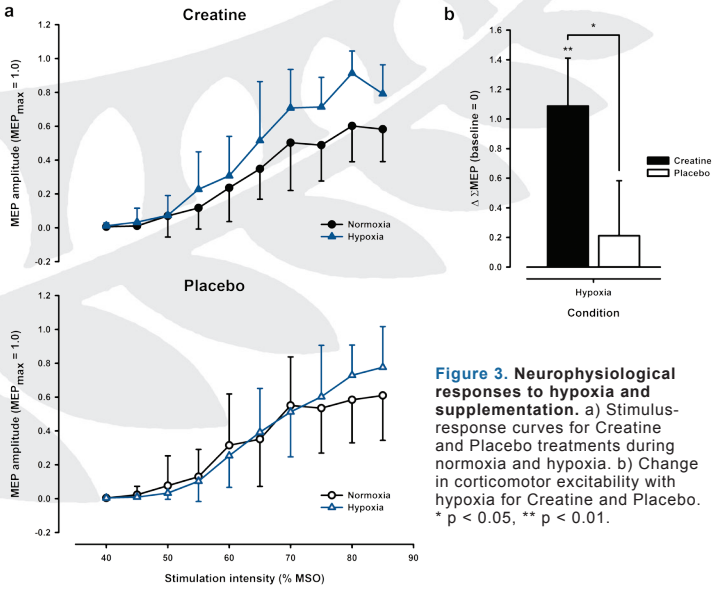
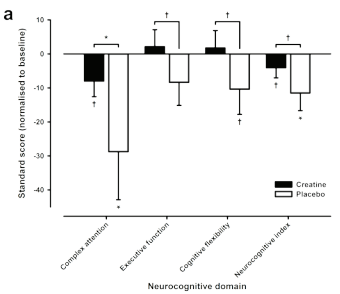


Figure 3. Neurophysiological responses to hypoxia and supplementation. a) Stimulus-response curves for Creatine and Placebo treatments during normoxia and hypoxia. b) Change in corticomotor excitability with hypoxia for Creatine and Placebo. * $p < 0.05$, ** $p < 0.01$.

Creatine supplementation offset the decline in cognition that occured with Placebo during hypoxia



b

Correlations between Δ corticomotor excitability and Δ cognition (R^2)

	Creatine	Placebo
Composite memory	0.14, p = 0.22	**0.50, p < 0.01**
Verbal memory	0.08, p = 0.36	0.28, p = 0.06
Visual memory	0.15, p = 0.19	**0.38, p = 0.02**
Processing speed	**0.33, p = 0.04**	**0.40, p = 0.02**
Executive function	0.03, p = 0.56	**0.57, p < 0.01**
Psychomotor speed	0.20, p = 0.19	**0.68, p < 0.01**
Reaction time	0.01, p = 0.73	0.29, p = 0.06
Complex attention	0.12, p = 0.25	**0.59, p < 0.01**
Cognitive flexibility	0.04, p = 0.51	**0.59, p < 0.01**
Neurocognitive index	0.02, p = 0.63	**0.59, p < 0.01**

Figure 4. Neuropsychological responses to hypoxia and supplementation. a) Changes in cognitive domain scores during hypoxia for Creatine and Placebo. * $p < 0.05$, † $p \geq 0.05$ and < 0.1. b) Bold typeface highlights significant correlations with Figure 3b data.

Conclusion

Creatine has potential utility as a neuroprotective supplement when cellular energy provision is compromised.

References **1.** Carter, A. J. et al. *J Neurochem* 64, 2691-2699 (1995). **2.** Balestrino, M. et al. *Brain Res* 816, 124-130 (1999). **3.** Shen, H. et al. *Neurobiol Dis* 47, 184-193 (2012). **4.** Gualtieri, C. T. et al. *Arch Clin Neuropsychol* 21 (2006).

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