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Beyond the Muscle: Assessing the Efficacy of Oral Creatine Supplementation in Modulating Brain Bioenergetics and Cognitive Performance

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Abstract

Background. The human brain is a metabolically demanding organ, accounting for nearly 20% of the body's total resting energy expenditure. While creatine has historically been viewed primarily as a supplement for muscle hypertrophy, the discovery of brain-type creatine kinase (BB-CK) has shifted scientific consensus, highlighting its critical role as a neuroprotective metabolite and potential nootropic agent during transient "energy gaps."

Aim. This review aims to synthesize current scientific evidence on how oral creatine supplementation can modulate brain bioenergetics and affect overall cognitive performance.

Material and methods. This narrative literature review synthesized data from PubMed, Scopus, and the Cochrane Library. The search focused on keywords including “creatine”, “cognitive function”, and “brain bioenergetics”, incorporating systematic reviews, meta-analyses, and randomized controlled trials published in English or Polish, without time restrictions.

Results. Current evidence suggests that creatine facilitates the efficient recycling of ATP, which is essential for maintaining neuronal membrane potentials and signal transduction. Supplementation appears to fortify brain bioenergetics, particularly during periods of intense cognitive activity, sleep deprivation, or mental fatigue, helping to bridge the gap when ATP consumption exceeds aerobic synthesis.

Conclusions. Oral creatine supplementation emerges as a promising public health strategy to support brain bioenergetics and mitigate cognitive decline. It serves as an effective intervention to enhance cognitive performance under metabolic stress, though further research is warranted to optimize dosing protocols specifically for neurological health.

Keywords: creatine, brain bioenergetics, cognitive performance, neuroprotection, phosphocreatine

1. Introduction

The human brain is a metabolically "gluttonous" organ, characterized by an exceptionally high energy demand that remains relatively constant despite its structural stability [1]. While accounting for a small fraction of total body weight, it is responsible for nearly 20% of the body's total resting energy expenditure [2]. This energy is predominantly consumed by the Na+/K+-ATPase pumps required to maintain neuronal membrane potentials and facilitate signal transduction [3]. During periods of intense cognitive activity, local energy demands can fluctuate rapidly, creating a transient "energy gap" where the rate of ATP consumption exceeds its aerobic synthesis [2].

Worthy of attention is the fact that any neuroanatomical or metabolic fluctuations within this complex system can significantly modify cognitive functions [4]. For decades, creatine was viewed through a narrow lens as a supplement reserved for muscle hypertrophy and power in strength athletes [5]. However, the identification of brain-type creatine kinase (BB-CK) and the discovery of inborn errors of creatine metabolism leading to severe neurological deficits have shifted the scientific consensus[6]. Creatine is now recognized as a critical neuroprotective metabolite and a potential nootropic agent [7]. As the population ages and the prevalence of cognitive decline increases, understanding the capacity of oral supplementation to fortify brain bioenergetics has become a public health priority [8]. This review aims to synthesize current evidence on how oral creatine can modulate brain bioenergetics and cognitive performance.

2. Methodology

This study was conducted as a narrative literature review. A literature search was performed in the PubMed, Scopus, and Cochrane Library databases. Systematic reviews, meta-analyses, randomized controlled trials, and original research articles were included. The search was conducted using a combination of keywords including: “creatine”, “creatine monohydrate”, “cognitive function”, “memory”, “brain bioenergetics”, “neuro-nutrition”, “magnetic resonance spectroscopy”, and “blood-brain barrier”. The literature screening and selection were performed independently by the authors. Studies published in languages other than English and Polish were excluded. No time restrictions were applied to the literature search.

3. Literature review

3.1 Brain Bioenergetics

At the molecular level, the brain's energy homeostasis relies on the efficient recycling of ATP (adenosine triphosphate). Creatine facilitates this process via the phosphocreatine (PCr) shuttle system[9]. Within the cytosol and mitochondria, the enzyme creatine kinase (CK) catalyzes the reversible transfer of a phosphoryl group between ATP and creatine, forming PCr and ADP [10]. PCr serves as a mobile energy reservoir, capable of diffusing from mitochondrial sites of production to distal cytosolic sites of high energy demand, where it rapidly regenerates ATP [6].

Despite this elegant system, the brain's ability to augment its creatine stores through oral supplementation is constrained by physiological barriers [11]. The blood-brain barrier (BBB) restricts the entry of systemic creatine [2]. The primary mechanism for entry is the SLC6A8 transporter, located on the capillary endothelial cells and astrocytes [12]. However, the density of SLC6A8 in the brain is significantly lower than in skeletal muscle, making brain uptake a slower and less efficient process [13]. Consequently, while muscle creatine can be saturated within 5–7 days of loading, the brain may require higher dosages (e.g., 20g/day) and longer durations (4 weeks or more) to show significant increases in tissue concentration [2].

3.2 Cognitive Efficacy Across the Lifespan and Demographics

The efficacy of creatine as a nootropic is highly context-dependent, often showing greater benefits in individuals with lower baseline levels or during metabolic stress [14]. Research has identified several distinct populations that respond differently to supplementation protocols [3].

3.2.1 Older Adults and Aging Populations

Aging is associated with a natural decline in brain PCr concentrations and mitochondrial efficiency, which contributes to age-related memory loss [15]. Meta-analyses of RCTs have demonstrated that creatine supplementation significantly improves memory performance in individuals aged 66–76 years. These gains are often observed in tasks requiring working memory and executive control [1]. Supplementation may act by stabilizing the ATP/ADP ratio, thereby preserving neuronal integrity and reducing cognitive fatigue [16].

3.2.2 Vegetarians and Vegans

Dietary creatine is found exclusively in animal products. Individuals following a vegetarian or vegan diet typically have lower systemic and brain creatine levels than omnivores. Early landmark studies showed that a 6-week protocol of 5g/day led to significant improvements in intelligence and working memory in vegetarians [3]. More recent evidence suggests that while the effects are robust, they may vary based on the specific cognitive domains tested [14].

3.2.3 Healthy Young Omnivores

In contrast, healthy young omnivores who consume meat often have baseline creatine levels near saturation [17]. In these individuals, the cognitive benefits of supplementation under "normal" (non-stressed) conditions are frequently non-significant or marginal [18]. This "ceiling effect" suggest that additional creatine provides little benefit when the metabolic demand is already met by endogenous synthesis and diet [11].

Table 1. Comparative effect of creatine supplementation on cognitive performance in various populations

Author (Year)	Population	Dose/Duration	Key Cognitive Outcome
Rae et al. (2003)	Vegetarians (n=45)	5g/day for 6 weeks	Significant increase in intelligence (Raven's Matrices) and memory (Digit Span).
McMorris et al. (2007)	Elderly (n=32)	20g/day for 7 days	Improved performance in forward spatial recall and long-term memory.

Sandkühler et al. (2023)	Young Adults (n=120)	20g/day for 7 days	No significant effect on Raven's Matrices in healthy, rested individuals.
Prokopidis et al. (2023)	Aging Meta-analysis	Various	Strong evidence for memory enhancement in older adults.

Source: Author's own elaboration

3.3 Performance Under Metabolic Stress and Neuroprotection

The true potential of creatine as a nootropic emerges when the brain is subjected to acute physiological or environmental stressors [9]. Under these conditions, the ATP demand increases, and the compensatory capacity of the PCr system becomes vital [2].

3.3.1 Sleep Deprivation and Mental Fatigue

Sleep deprivation causes a significant drop in brain ATP and an increase in mental fatigue. Research indicates that creatine supplementation can mitigate the negative effects of sleep loss on mood and complex executive tasks [19]. In athletes, creatine may help maintain visuomotor skills and decision-making accuracy during prolonged mental exertion [20]. By buffering energy levels, creatine prevents the cognitive "crash" often associated with intense mental fatigue [10].

3.3.2 Hypoxia and Oxygen Deprivation

In conditions of hypoxia (e.g., high altitude or clinical stroke models), oxidative phosphorylation is impaired. Creatine supplementation has been shown to preserve reaction time and psychomotor performance during acute oxygen deprivation [9]. This neuroprotective effect is likely due to the enhanced reliance on anaerobic ATP regeneration via the PCr system, delaying neuronal dysfunction[13].

3.3.3 Traumatic Brain Injury (TBI) and Concussion

Post-concussion, the brain enters a state of "metabolic crisis" where energy demand is high but glucose metabolism is compromised [19]. Emerging clinical data suggest that creatine can

reduce the severity of post-traumatic symptoms and decrease cortical tissue loss [20]. Prophylactic use in high-risk populations, such as football or rugby players, is being advocated to build neural resilience before injury occurs [5]. Pre-loading the brain with creatine may limit the metabolic shock that follows a mechanical impact [21].

3.4 Synergistic Effects and the Muscle-Brain Axis

Recent advancements in sports science have identified a "muscle-brain axis," where physical activity and nutritional interventions interact to enhance systemic health [13]. Resistance training has been shown to improve cognitive function through the release of myokines like Irisin and the upregulation of Brain-Derived Neurotrophic Factor (BDNF) [22]. Creatine supplementation appears to amplify these benefits, especially in the elderly [13]. Studies combining creatine with structured exercise programs show superior outcomes in both muscle mass and cognitive resilience compared to exercise alone [22]. This synergy suggests that creatine is not just an energy buffer but also a modulator of neurotrophic pathways [23].

3.5 The Bioavailability and Dosing Conundrum

The primary challenge in utilizing creatine monohydrate as a nootropic agent lies in its restricted bioavailability within the central nervous system [2]. Unlike skeletal muscle tissue, which readily sequesters systemic creatine, the human brain demonstrates a pronounced physiological resistance to the uptake of this exogenous compound [11]. Specifically, brain creatine uptake relies heavily on the sodium- and chloride-dependent creatine transporter, known as SLC6A8, which is expressed at the blood-brain barrier (BBB) [6]. However, this transporter lacks ubiquitous distribution across all neuroglial cell types, particularly astrocytes [24]. Because astrocytes are intimately involved in both the maintenance of the BBB and the complex metabolic coupling with adjacent neurons, this absence of widespread SLC6A8 expression creates a significant physiological bottleneck for the transport of exogenous creatine into the central nervous system [2]. Consequently, achieving meaningful elevations in cerebral phosphocreatine stores necessitates substantially higher and more prolonged dosing strategies than those conventionally employed for athletic purposes [18].

Standard loading protocols of five grams per day are generally insufficient to induce significant bioenergetic alterations within neuronal networks [25]. Current neurocognitive research indicates that prolonged supplementation protocols, often requiring 20 grams per day for four weeks or longer, are necessary to yield a modest five to ten percent increase in total brain

creatine concentrations [21]. The chronic administration of such high-dose regimens, particularly those exceeding 0.1 grams per kilogram of body weight per day, inevitably raises questions regarding long-term clinical safety [26]. Extensive biochemical monitoring in longitudinal studies has consistently demonstrated that these elevated dosages do not precipitate renal impairment, hepatic dysfunction, or deleterious alterations in the glomerular filtration rate in healthy adult cohorts [8]. Despite this robust systemic safety profile, the most frequently reported adverse effect associated with high-dose loading is mild gastrointestinal distress [10]. To mitigate symptoms such as nausea or diarrhoea, clinicians frequently recommend fractionating the total daily dosage into smaller, more frequent administrations throughout the day [8]. In clinical populations enduring severe metabolic distress, the neuroprotective advantages of high-dose creatine supplementation substantially outweigh the minimal risks of these transient gastrointestinal perturbations [19].

Furthermore, the efficacy of creatine as a cognitive enhancer is highly modulated by inter-individual variability, resulting in a distinct dichotomy between clinical responders and non-responders [27]. This status is fundamentally dictated by the individual's baseline neurochemical profile, particularly their preexisting cerebral phosphocreatine reserves [14]. Subjects adhering to vegan or vegetarian diets typically exhibit lower baseline brain creatine concentrations, rendering them highly responsive to exogenous supplementation [3]. Conversely, healthy omnivores often possess near-saturated neural creatine stores, which effectively blunts the nootropic potential of further dietary intake under basal conditions [11]. Beyond dietary habits, genetic polymorphisms influencing the enzymatic activity of arginine: glycine amidinotransferase (AGAT) and guanidinoacetate N-methyltransferase (GAMT) can significantly alter endogenous synthesis rates and subsequent supplementation needs [12]. Similarly, inherent genetic variations in the SLC6A8 transporter gene may completely impede effective transmembrane transport, rendering some individuals entirely refractory to oral protocols [25]. Finally, acute physiological stressors, such as severe sleep deprivation or systemic hypoxia, can transiently deplete neural ATP levels, temporarily shifting a phenotypic non-responder into a state where exogenous creatine provides substantial cognitive salvage [28].

Ultimately, a nuanced understanding of these inter-individual biochemical differences is imperative for the development of precise, personalized dosing algorithms in contemporary neuro-nutrition [7].

3.6 Insights from Magnetic Resonance Spectroscopy (MRS)

Proton Magnetic Resonance Spectroscopy and Phosphorus Magnetic Resonance Spectroscopy serve as the primary non-invasive modalities for the *in vivo* quantification of cerebral metabolites, effectively acting as a window into the energy reserves of the central nervous system [2]. While standard neuroimaging provides structural data, Spectroscopy allows for the direct measurement of total creatine—the sum of free creatine and phosphocreatine—within specific intracranial regions [29]. Research utilizing these techniques has demonstrated that the human brain exhibits significant regional heterogeneity in its baseline creatine concentrations, typically showing higher levels in gray matter compared to white matter [30].

Spectroscopy studies have played a pivotal role in validating the efficacy of oral supplementation, confirming that exogenous creatine can indeed cross the blood-brain barrier and augment neural stores[9]. Longitudinal monitoring has revealed that high-dose loading protocols—often involving 20 grams per day for several weeks—are typically required to achieve a measurable five to ten percent increase in total brain creatine [21]. This relatively modest increase, when compared to the twenty percent or greater expansion seen in skeletal muscle, highlights the brain's inherent resistance to uptake and its reliance on endogenous synthesis [11]. Interestingly, data show that this increase is not uniform across the brain, with some studies indicating more pronounced accumulation in the frontal cortex and cerebellum [3]. Furthermore, these methods have been instrumental in showing that augmented reserves correlate with improved recovery of energy levels following acute cognitive or metabolic challenges [30].

The diagnostic and prognostic utility of Spectroscopy extends to identifying states of metabolic depletion in various clinical conditions [19]. In cases of traumatic brain injury or concussion, imaging frequently reveals a significant reduction in the cerebral creatine pool, which correlates with the severity of the metabolic crisis and post-traumatic symptoms [31]. Similarly, aging is associated with a progressive decline in phosphocreatine levels in the prefrontal cortex, a change that can be detected long before structural atrophy becomes apparent [15]. By providing real-time data on the brain's energy reserves, these insights allow researchers to differentiate between individuals who respond to treatment and those who do not, based on how effectively their energy stores are replenished during a supplementation protocol [27]. Ultimately, these findings confirm that oral creatine acts as a strategic buffer, enhancing the brain's metabolic resilience against stressors that would otherwise compromise neuronal function [7].

4. Conclusions - Future Directions in Neuro-Nutrition

The integration of creatine monohydrate into the broader paradigm of neuro-nutrition represents a significant shift from its traditional perception as a purely athletic ergogenic aid [7]. Currently, the most robust clinical evidence supporting the nootropic efficacy of creatine highlights its capacity to mitigate cognitive deterioration during acute metabolic crises, such as severe sleep deprivation and systemic hypoxia [2]. Under these cognitively demanding conditions, exogenous supplementation effectively preserves executive functioning, psychomotor vigilance, and mood state by buffering the accelerated depletion of neural adenosine triphosphate [28]. Furthermore, comprehensive meta-analyses consistently demonstrate that creatine provides statistically significant improvements in both short-term and working memory, particularly among the elderly population aged 66 to 76 years [1]. These measurable cognitive benefits are heavily dictated by baseline neurochemical profiles, with the most profound enhancements observed in populations possessing inherently lower cerebral phosphocreatine reserves, such as vegetarians and vegans [14].

Despite these promising acute and medium-term findings, a critical knowledge gap persists regarding the neuroprotective efficacy of extended, multi-year supplementation protocols [11]. Specifically, the medical community currently lacks the rigorous, large-scale longitudinal trials necessary to definitively determine whether chronic creatine loading can alter the pathophysiological trajectory of age-related neurodegenerative conditions [32]. While preclinical models and pilot studies suggest potential benefits in mitigating the severity of neurological damage, the translational leap to preventing human cognitive decline requires robust epidemiological validation [2]. Additionally, further pharmacokinetic investigations are urgently required to establish standardized, brain-specific dosing guidelines that efficiently overcome the limited permeability of the blood-brain barrier without causing mild gastrointestinal distress [25]. Unraveling the complex inter-individual variability mediated by genetic polymorphisms in the SLC6A8 transporter remains a primary objective for the development of future personalized neuro-nutrition strategies [24].

Nevertheless, the extensive safety profile established over decades of clinical research confirms that even high-dose protocols do not induce renal or hepatic toxicity in healthy adults [26]. Emerging evidence also highlights a potent synergistic effect when creatine administration is combined with structured resistance training, offering a dual-action physiological defense against both sarcopenia and progressive cognitive decline [22]. Consequently, when objectively evaluating the current risk-to-benefit ratio, oral creatine monohydrate emerges as a highly viable, cost-effective, and safe non-pharmacological intervention for promoting healthy neuro-

aging [8]. As the global demographic shifts toward an older population, the routine inclusion of this metabolite in geriatric care and public health initiatives appears not only scientifically justified but practically essential [7]. Ultimately, creatine stands at the forefront of preventative neurology as an accessible tool for fortifying lifelong cognitive resilience and maintaining independent psychological functioning [2].

Disclosure:

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