



CREATINE SUPPLEMENTATION: A POTENTIAL NUTRITIONAL STRATEGY TO REDUCE SEVERITY OF AND ENHANCE RECOVERY FROM TRAUMATIC BRAIN INJURY AND CONCUSSION

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KEY POINTS

- A concussion is a mTBI that occurs when the brain is forced to move rapidly inside the skull.
- Concussions occur in sporting, recreation, occupational, and military circumstances, totaling about 2.5 million global emergency room visits per year.
- Symptoms of concussion include headaches, confusion, dizziness, nausea, light and noise sensitivity, fatigue, behavioral changes, depression, and difficulty in concentrating. These symptoms can be resolved in days or a few weeks with subsequent return to normal activities. Impairments can also take longer to resolve, lasting many weeks or months, and there can even be lifelong deficits.
- The pathophysiology of concussion overlaps considerably with creatine biology, making CrM a potential candidate for a nutritional intervention to decrease severity of or enhance recovery from concussion.
- Potential benefits of CrM on concussion when used prophylactically or post-injury, have a sound biological basis and are supported by animal data, cross-sectional/observational human data, and clinical data from patient populations.
- Well-defined dosing recommendations for CrM to reduce severity of or enhance recovery from concussion are not currently available. Present data indicate that a wide range of CrM dosing and duration strategies can increase brain creatine (4 to 30 g/d from 1 d to 6 months), and, in an open label trial, children with TBI were successfully treated with CrM of 0.4 g/kg/d (~15 g/d for 6 months). The doses of CrM used in preclinical studies to treat TBI translate to human-equivalent doses that are largely achievable in practice (3-20 g/d). However, species differences, tight regulation of exogenous brain creatine uptake, differences in injury models, and the absence of data from randomized controlled trials limit precise dosing recommendations for humans relative to mTBI.
- A person at high-risk for a mTBI, such as athletes and military personnel, could consider taking a conventional CrM dose of 3-5 g/d, which provides muscular and possibly cognitive benefits, as a practical baseline strategy. Higher doses (e.g. 20 g/d) have been shown to increase brain creatine and provide neuroprotective effects during periods of metabolic stress.

INTRODUCTION

A concussion is a mild traumatic brain injury (mTBI) that occurs when the brain is forced to move rapidly inside the skull. Globally, concussions account for about 2.5 million emergency room visits per year and the Centers for Disease Control (CDC) estimates there are nearly 4 million concussions annually in the United States across sport, recreational, occupational, and military activities. Symptoms of concussion include headaches, confusion, dizziness, nausea, light and noise sensitivity, fatigue, behavioral changes, depression, and difficulty in concentrating. These symptoms can be resolved in days or a few weeks with subsequent return to normal activities. At times, impairments take longer to resolve, lasting many weeks or months, and there can even be lifelong deficits. Due to the large number of annual concussions, the breath of the population at risk, the cost in pain and suffering, and the lack of available prevention and recovery options, interest in effective mitigation strategies has increased. One such nutritional strategy that shows potential is creatine monohydrate supplementation (CrM). While many know the benefits of CrM on muscle function and athletic performance (Kreider et al., 2017) there are potential benefits of CrM on mTBI. The purpose of this Sports Science Exchange (SSE) article

is to: describe the pathophysiology of mTBI and how it overlaps with creatine metabolism, describe data that supports a beneficial effect of CrM on reducing the severity of or enhancing recovery from mTBI, and make practical CrM recommendations.

CONCUSSION AND CREATINE METABOLISM

Traumatic brain injury (TBI) produces a complex cascade of metabolic disturbances and adverse consequences that extend beyond the onset of injury. Following head trauma, there are decreases in rat brain creatine (Signoretti et al., 2010), and alterations in neuronal function, ion homeostasis, calcium flux, neurotransmitter excitability, mitochondrial function, and oxidative stress and inflammation, that collectively increase cellular energy requirements (Dean et al., 2017; Vietor et al., 2025). This abnormal increase in energy demand provides an opportunity and justification for interventions capable of supporting brain stability and bioenergetics. CrM is particularly relevant in this context because of its fundamental role in maintaining adenosine triphosphate (ATP) availability through the ATP-Phosphocreatine (PCr) reaction (Kreider et al., 2017). TABLE 1 highlights several areas where the neurometabolic cascade of events following mTBI overlap with

creatine metabolism, which indicates a potential avenue for mitigation.

ANIMAL DATA

Using rodent models, Sullivan et al. (2000) showed that 3 and 5 days of CrM (3 mg/g body mass (BM)) before experimentally induced TBI decreased cortical damage by 21% and 36%, respectively, in adult mice compared to adult mice not ingesting CrM. Further, Sprague-Dawley rats who consumed a standard diet enriched with 1% CrM for 4 weeks prior to TBI experienced a 50% reduction in cortical damage compared to rats fed a non-creatine enriched diet. Rats ingesting creatine also experienced greater mitochondrial function, increased levels of ATP, and reduced levels of intramitochondrial calcium and oxidative stress, all of which highlight potential neuroprotective effects of CrM (Sullivan et al., 2000). These findings suggest that increasing creatine availability before head trauma may improve the energetic resilience of cerebral tissue and the authors speculated that athletes may be afforded neuroprotection from CrM (Sullivan et al., 2000). Scheff and Dhillon (2004) provided additional evidence supporting a neuroprotective effect from CrM as adult male Sprague-Dawley rats fed a creatine-enriched diet containing 0.5% or 1% CrM for 2 weeks prior to TBI experienced a significant reduction in cortical tissue damage compared to rats on a regular diet. Both groups experienced significant cortical tissue sparing (1% CrM group: 93%; 0.5% CrM group: 88%).

In addition to the possible prophylactic benefits of CrM, emerging evidence suggests that CrM administered following TBI may also confer therapeutic benefits. Royes et al. (2026) showed that following TBI, CrM (300 mg/kg BM for 14 d) protected against cortical tissue loss and oxidative stress and improved measures of mitochondrial function, memory, and cognition in adult male Wistar rats compared to rats ingesting placebo. Previous work from the same research group showed that one week following TBI, rats who received CrM (300 mg/kg BM) for 4 weeks experienced a significant reduction in seizure susceptibility and γ -amino-butyric acid A (GABA)-ergic neuronal loss (Gerbatin et al., 2019). Together, these data indicate that CrM prior to or following mTBI improves many of the physical derangements in

rodents caused by concussion. One caveat is that in response to CrM, laboratory animals increase brain creatine by 30-50%, while humans appear to only increase brain creatine about 5-10% in response to CrM (Candow et al., 2026; Forbes et al., 2022; Pratt et al., 2026; Roschel et al., 2021).

CROSS-SECTIONAL/OBSERVATIONAL DATA

In humans, brain creatine levels are associated with symptoms of concussion over both acute and long-term periods of time (Alosco et al., 2020; Howell et al., 2026; Miller et al., 2022). Howell et al. (2026) showed significant associations between brain creatine and concussion symptom severity ($r=-0.61$), mobility impairments ($r=-0.57$), and pain interference ($r=-0.40$) in adolescents about 2 weeks post-injury demonstrating the overlap between mTBI pathophysiology and brain creatine availability. Similarly, Alosco and colleagues (2020) reported an inverse association between exposure to repetitive head impacts and brain creatine levels ($r=-0.23$) in retired professional football players, indicating that long-term changes in cognitive, behavioral, and mood symptoms were associated with abnormal creatine brain metabolism. Finally, Miller and others (2022), using metabolomic profiling, found creatine could be used to identify military breachers and range staff, who are subject to repeated sub-concussive blasts and display concussion like symptoms. Collectively, these studies show a strong connection between mTBI symptomology and brain creatine reserves.

CLINICAL DATA

In the context of mTBI, an important prerequisite for the potential therapeutic benefits of CrM is whether humans can meaningfully increase brain creatine with CrM (Candow et al., 2026; Forbes et al., 2022; Pratt et al., 2026; Roschel et al., 2021). Unlike skeletal muscle, the brain endogenously synthesizes creatine (Braissant, 2012) and does not need to rely on exogenous sources of extracellular creatine. Further, although circulating creatine can enter the brain through the creatine transporter (SLC6A8), which is expressed at the blood-brain barrier, transport appears restricted as SLC6AB is expressed in very

TABLE 1. Pathophysiology of mild traumatic brain injury (mTBI) and potential beneficial effects of creatine monohydrate (CrM) supplementation. ATP, adenosine triphosphate.

Pathophysiology of mTBI	Potential benefits of creatine supplementation	Relevant reference
Decreased brain creatine	Increased brain creatine and phosphocreatine	(Candow et al., 2023; Candow et al., 2026; Forbes et al., 2022; Pratt et al., 2026; Roschel et al., 2021)
Large scale membrane depolarization	Increased mitochondrial membrane potential	(Sullivan et al., 2000)
Increased calcium	Decreased calcium	(Sullivan et al., 2000)
Cellular energy crisis (increased ATP demand, increased glycolysis, increased lactate production)	Improved energy availability	(Marshall et al., 2022; Scheff & Dhillon, 2004; Sullivan et al., 2000)
Decreased oxidative energy production/dysfunctional mitochondria	Improved mitochondrial function	(Marshall et al., 2022)
Increased reactive oxygen species	Decreased oxidative stress increased antioxidant activity	(Royes et al., 2026; Sestili et al., 2011; Sullivan et al., 2000)
Increased inflammation	Decreased inflammation	(Nomura et al., 2003)
Increased tissue damage	Decreased tissue damage	(Royes et al., 2026; Scheff & Dhillon, 2004; Sullivan et al., 2000)
Increased neuronal cell loss	Decrease neuronal cell loss through interaction with N-methyl-D-aspartate (NMDAR), Na ⁺ -K ⁺ -ATPase enzyme, γ -amino-butyric acid A (GABAA), and serotonin 1A receptors, and α 1-adrenoceptor.	Meftahi et al. 2023 ; Gerbatin et al., 2019)

low levels in astrocytic feet (Braissant, 2012). Thus, brain creatine concentrations appear tightly regulated from exogenous sources such as diet or CrM supplementation. However, several researchers have reported increased brain creatine with CrM supplementation in healthy human populations (Candow et al., 2026; Pratt et al., 2026)). In the seminal study by Dechent et al. (1999), an acute dose of CrM (20 g) produced a non-significant increase in creatine concentrations within grey matter (3.1%), white matter (3.1%) and the thalamus (7.7%) in six participants (~ 27 yr) for up to 8.5 hours post CrM ingestion. However, extending the duration of CrM (20 g/d for 4 wk) resulted in a significant increase (8.7%) in total brain creatine levels in six participants (24-30 yr). Regional increases in gray matter (4.7%), white matter (11.5%), cerebellum (5.4%) and thalamus (14.6%) were also significant. Since 1999, several research teams have reported increased brain creatine in healthy and patient populations in response to CrM supplementation (Candow et al., 2026; Forbes et al., 2022; Pratt et al., 2026; Roschel et al., 2021)).

When brain creatine levels are increased after CrM there can be improvements in cognitive processing (e.g. memory). Several narrative, systematic, and meta-analytic reviews are available that report improved aspects of cognitive processing following CrM (Avgerinos et al., 2018; Candow et al., 2026; Forbes et al., 2022; Marshall et al., 2026; Prokopidis et al., 2023; Xu et al., 2024). Arguably, improved cognitive processing is most commonly found when the brain is temporarily stressed or challenged via sleep deprivation, or mentally or physically fatiguing tasks. Because stressing the brain temporarily impairs cognitive processing, and this decrement is reduced with CrM, some believe that CrM could also attenuate a decrease in cognitive function following mTBI.

While this theory is attractive, and findings in rodents are encouraging, only two studies have directly examined the efficacy of CrM on TBI outcomes in humans (Sakellaris et al., 2006, 2008). They conducted a prospective, randomized, open-label pilot study involving 20 children and adolescents (1-18 yr) with TBI. CrM (0.4 g/kg BM/d) was administered for 6 months and results showed improvements in measures of memory, self-care, recovery, communication, mobility, behavior, and cognitive functioning. The lack of a measure of cortical tissue damage or direct assessment of neuroprotection and the open-label design require caution, but these findings provide the first line of evidence that CrM administered after TBI may potentially facilitate recovery in humans. In a related study, short-term CrM (20 g/d for 7 d) increased brain creatine (+ 9.2%) and attenuated the deterioration in attentional performance and altered corticomotor excitability when participants were exposed to severe experimental hypoxia (10% oxygen) for 90 min (Turner et al., 2015). Several double-blind placebo-controlled randomized controlled trials designed to investigate the potential benefits of CrM supplementation on recovery from mTBI are underway ((Bödker & Marcussen, 2023); Clinicaltrials.gov ID NCT06208813, NCT05562232, NCT0558906). Importantly, the lowest dose of CrM shown to increase brain creatine levels and reduce

fatigue and prevalence of headaches in a clinical population of humans with post-COVID-19 syndrome is 4 g/d (Slankamenac et al., 2023)

SUMMARY AND PRACTICAL APPLICATIONS

The convergence of mechanistic, animal, cross-sectional/observational, and preliminary human studies related to creatine metabolism and concussion provides a compelling and promising rationale for further investigation. Available data indicate that CrM supplementation could provide both prophylactic and post-concussion benefits, and in practical terms may reduce severity of and enhance recovery from mTBI. CrM is inexpensive, widely available, can increase brain creatine levels and produce some cognitive benefits under specific circumstances. Further, the safety profile of CrM is generally excellent (Kreider et al., 2025). Additionally, dietary creatine intake is decreasing (Korovljev et al., 2021) and daily intake is now significantly lower than commonly accepted estimates (Ostojic, 2025). Well defined dosing recommendations to reduce severity of or enhance recovery from concussion are not presently available. However, even in lieu of large, adequately powered, placebo-controlled randomized clinical trials and dose-response studies, it appears prudent to offer sensible recommendations for CrM supplementation related to concussion.

To increase skeletal muscle creatine to saturation, CrM supplementation of 3 g/d for 30 d or 20 g/d (~0.3 g/kg BM/d) for 5-7 d is recommended and supported by seminal CrM studies (Harris et al., 1992; Hultman et al., 1996). To maintain increased muscle creatine, CrM supplementation of 3-5 g/d is often recommended (Hultman et al., 1996; Krieder et al., 2017). To increase brain creatine, doses of 4-30 g/d administered over periods ranging from 1 d to 6 months have been used successfully in healthy adults and clinical populations, including individuals with Huntington's disease, Alzheimer's disease, post-COVID-19 syndrome, and clinical depression (Candow et al., 2026). The dose of CrM used by Sakellaris and colleagues (2006, 2008) to decrease symptoms of concussion in children with TBI was 0.4 g/kg BM/d or ~15 g/d for 6 months, which is in agreement with human trials of CrM supplementation that show improved cognitive processing and/or increased brain creatine. Importantly, the doses used in preclinical studies translate into human-equivalent doses that are largely achievable in practice. Using body surface area scaling, preclinical studies reporting body-mass adjusted doses correspond to ~3-20 g/d of CrM in an 80-kg adult, encompassing both customary maintenance intakes (3-5 g/d) and commonly employed loading protocols (20 g/d). However, species differences, tight regulation of exogenous brain creatine uptake, differences in injury models, and the absence of data from randomized controlled trials limit precise dosing recommendations for humans relative to mTBI.

The optimal CrM dose and/or duration for reducing symptoms of mTBI, preventing secondary neurological injury or improving recovery remains unknown. Nevertheless, based on its generally favorable safety profile, accessibility, and established capacity to increase brain creatine, CrM supplementation may represent a promising nutritional strategy

for individuals at elevated risk of head trauma, including athletes and military personnel. A conventional CrM dose of 3-5 g/d represents a practical baseline strategy, whereas higher doses (i.e., 20 g/d) have been shown to increase brain creatine and provide neuroprotective effects during periods of metabolic stress. However, these preliminary findings do not establish an optimal neuroprotective dose and there is currently insufficient evidence to conclude that higher doses of CrM reduce the severity of or accelerate recovery from mTBI in humans. Future randomized controlled trials are needed to establish whether CrM supplementation provides meaningful neuroprotection and to determine the optimal CrM protocol.

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