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CRITICAL LITERATURE REVIEW

Creatine monohydrate: what the evidence actually says

A critical review of the literature for training professionals

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EXECUTIVE SUMMARY

Creatine monohydrate is the most heavily researched ergogenic supplement in sport science. The evidence supports a **real but modest** effect on fat-free mass and on performance in short, high-intensity efforts.

Three nuances that commercial messaging tends to leave out:

1. The effect size for hypertrophy, when measured with direct imaging, is **very small** ($g \approx 0.11$).
2. The effect is **physiologically reversible**: it does not permanently modify muscle.
3. There is **considerable interindividual variability**, largely determined by baseline intramuscular stores.

Practical conclusion: it is a **training amplifier**, not a requirement. Its absence from a well-built diet is not a deficiency.

1. Physiological basis

Creatine is an amino-acid derivative synthesised endogenously in the liver, kidneys and pancreas from glycine, arginine and methionine, and obtained through the diet mainly from meat and fish. Roughly 95% of the body pool is stored in skeletal muscle, in an approximate ratio of two thirds as phosphocreatine (PCr) and one third as free creatine.

The phosphagen system acts as an energy buffer: creatine kinase catalyses the transfer of a phosphate group from PCr to ADP, regenerating ATP within fractions of a second. This system is the limiting factor in maximal efforts of roughly 0-10 seconds and in recovery between sets.

Supplementation raises intramuscular creatine content by 20% to 40% in individuals with low baseline stores, up to a physiological ceiling of approximately 150-160 mmol/kg dry mass.

Key implication: the effect depends on the distance between baseline level and that ceiling. Anyone already close to saturation has little room to improve.

IN PLAIN LANGUAGE

Muscle keeps a fast-start battery. Creatine fills that battery. If it is already nearly full because you eat meat and fish, adding more does little: the tank has a lid.

2. Effect on muscle mass

2.1 Pooled estimate

A dose-response meta-analysis of 61 controlled trials ($n \approx 1,457$) found an increase in fat-free mass of **1.39 kg** (95% CI: 1.07-1.70; $p < 0.001$) and in body mass of **0.89 kg** (95% CI: 0.76-1.01) versus control, with no significant effects on fat mass, BMI or body-fat percentage.

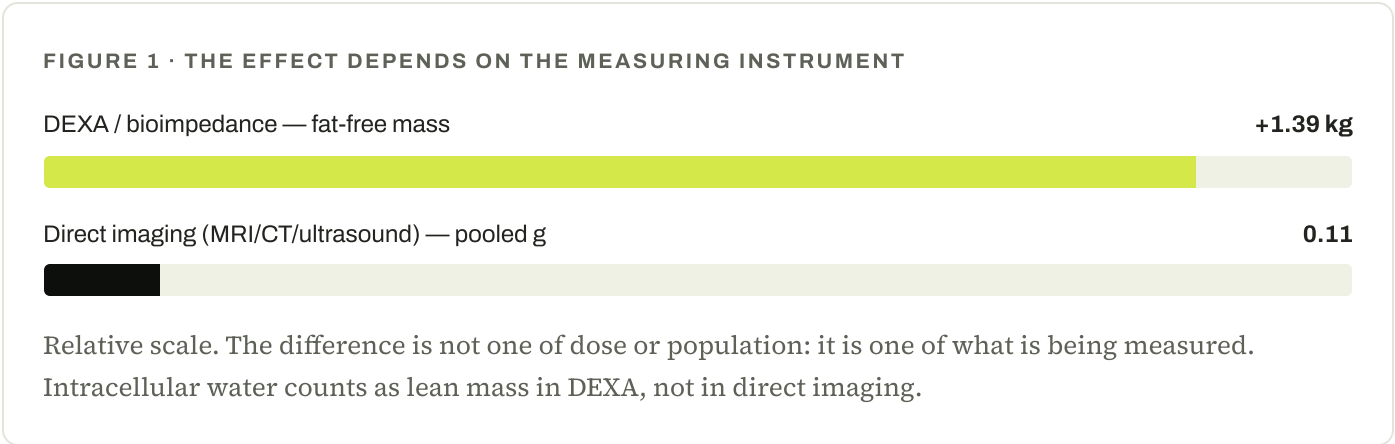
AN IMPORTANT, COUNTERINTUITIVE NOTE

In this analysis, **trained** subjects gained *more* fat-free mass than novices (1.82 kg vs. 1.23 kg), although the difference between groups **did not reach statistical significance**. That qualifies the widespread idea that creatine "stops working" in the veteran athlete. The honest conclusion is that the available evidence **does not demonstrate** superiority in either group.

2.2 The measurement problem

This is the core of the methodological debate. Creatine is osmotically active: its intracellular accumulation pulls water in. DEXA and bioimpedance **cannot distinguish** new contractile tissue from intracellular water. Both are counted as "lean mass".

When **direct imaging** is used (MRI, CT, ultrasound), the effect shrinks drastically. The meta-analysis by Burke et al. (2023), with 44 outcomes extracted from 10 RCTs of at least six weeks, produced a pooled estimate of **0.11** (95% credible interval: -0.02 to 0.25) — a **very small** effect favouring creatine, with an interval that crosses zero.



2.3 The trial that separated water from muscle

Desai et al. (2025), UNSW Sydney, designed a 13-week RCT specifically to resolve this confound. 63 participants (34 women, 29 men; 31 ± 8 years) were randomised to 5 g/day of monohydrate or control, with three DEXA measurements:

PHASE	DURATION	RESULT
T1 → T2: <i>wash-in</i> , no training	7 days	Creatine group gained ~0.5 kg more lean mass
T2 → T3: supervised training	12 weeks	No difference between groups
Total	13 weeks	Both groups: ~2 kg of lean mass

The design logic is elegant: by capturing water retention *before* introducing the training stimulus, everything gained afterwards is attributable to training. And there, creatine contributed nothing measurable.

The authors note that 5 g/day may be insufficient for a hypertrophy goal, and that 12 weeks may be a short window in which to detect a cumulative effect. These are legitimate limitations, not excuses.

3. On reversibility: "are the effects temporary?"

Yes, the direct effect is. There is no mechanism by which creatine permanently modifies muscle tissue.

On discontinuing supplementation:

- PCr stores decline progressively towards baseline, with a typical window of **4 to 6 weeks**. Some biopsy studies have reported values still elevated at 30 days, which indicates the return is slower than usually assumed.
- The associated intracellular water is lost, with the corresponding drop in weight (usually 1-2 kg).
- Performance in repeated high-intensity efforts returns to baseline.

The nuance that does matter: what is not reversed is the **accumulated work**. If for months supplementation allowed one or two extra repetitions per set, that extra volume produced structural and neural adaptation that persists. Creatine does not build muscle; it lets some people train marginally more.

Hence the most accurate characterisation is a **stimulus-dependent amplifier**: without training it produces no hypertrophy, and with an already-optimised stimulus the margin it can amplify is small.

IN PLAIN LANGUAGE

If you stop taking creatine, you lose the water and the small performance edge within a few weeks. You do not lose the muscle you built while training with it: that is yours.

4. Variability of response

Not everyone responds the same way. The documented determinants:

Baseline stores. The dominant factor. Vegetarians and vegans, with essentially no dietary intake, have stores 20-30% lower and show the largest absolute increases.

Fibre composition. A higher proportion of type II fibre is associated with greater uptake and greater functional benefit, since the phosphagen system is proportionally more relevant in those fibres.

Habitual dietary intake. Roughly 1-2 g/day in an omnivore who regularly eats red meat and fish, which brings the person close to the saturation ceiling through food alone.

Total muscle mass. Determines absolute storage capacity.

An estimated 20% to 30% of the population are "non-responders" in terms of increased intramuscular PCr. This is not a protocol failure: there was simply no deficit to correct.

5. Safety

5.1 Kidney function

Controlled research in healthy individuals has not demonstrated impairment of kidney function at conventional doses (3-5 g/day maintenance), even over years of follow-up.

RELEVANT CLINICAL WARNING

Creatine raises **serum creatinine** through non-enzymatic conversion. This can produce an abnormal lab value with no kidney dysfunction whatsoever. It is essential to tell the physician before a blood test, or to stop 3-4 weeks beforehand, to avoid mistaken diagnoses of kidney disease.

In pre-existing kidney disease, the decision belongs to the treating physician, not the trainer.

5.2 Hair loss

The myth comes from a single study in rugby players (2009) that reported an increase in DHT after a loading phase, without ever measuring hair.

Lak et al. (2025) ran the first RCT to assess the hair follicle directly: 45 strength-trained men (18-40 years), 5 g/day of monohydrate vs. 5 g of maltodextrin, 12 weeks, measuring total and free testosterone, DHT, hair density, follicular-unit count and hair thickness.

Result: no group × time interaction in any hormonal or hair variable ($p > 0.05$). No differences in DHT, DHT/testosterone ratio or hair-growth parameters.

Limitations worth acknowledging: 38 subjects completed the trial, there was no genetic screening for predisposition to androgenetic alopecia, and the trichogram is a debatable assessment tool. The evidence is reassuring, not definitive.

5.3 Other concerns

- **Cramps and dehydration:** controlled data do not support an increased incidence; several studies suggest the opposite in hot conditions.
- **Gastrointestinal discomfort:** possible with high single doses (>10 g). Resolved by splitting the dose.

- **Special populations:** the safety profile in adolescents, women and older adults is considered favourable in the available literature, although with less data than in young men.

6. Applications beyond muscle

This is where research is most active, and probably where the cost-benefit balance is most favourable.

Cognition

The meta-analysis by Xu et al. (2024), with 16 RCTs and 492 participants (20.8-76.4 years), found significant effects on **memory** (SMD = 0.31; 95% CI: 0.18-0.44), **attention time** (SMD = -0.31) and **processing speed** (SMD = -0.51), but **not** on global cognitive function or executive function.

Subanalyses point to a coherent pattern: the effect is greater in people with disease and in older adults — that is, **where cerebral energy availability is compromised**.

Sleep deprivation

Gordji-Nejad et al. (2024, *Scientific Reports*) gave a single high dose (0.35 g/kg) during 21 hours of enforced wakefulness, monitored by magnetic resonance spectroscopy (³¹P and ¹H). Creatine sustained cerebral phosphocreatine and ATP levels, and improved processing speed and working memory versus placebo. A later crossover RCT (2026) confirmed the mitigation of decline in logical and numerical tasks and in the psychomotor vigilance test.

This is young literature, with small samples and doses far above habitual use. Promising, not settled.

Sarcopenia and bone health

A line with accumulating evidence in older adults combining creatine with strength training. This is the population with the largest baseline deficit (lower meat intake, less muscle mass, lower mitochondrial efficiency) and therefore the greatest room to respond.

7. Note on conflicts of interest

A high proportion of the favourable literature comes from a relatively small group of researchers linked to the International Society of Sports Nutrition. Several of them declare research funding from creatine manufacturers, as well as advisory-board positions and speaking fees.

This **does not invalidate** their work — the declarations are transparent and the methodology is peer reviewed — but it does put in context the categorical tone of phrases such as "*the most effective ergogenic supplement available*".

It is worth noting that the same authors appear as co-authors on work with less favourable results (Candow is listed in Desai et al. 2025; Forbes and Antonio in Lak et al. 2025), which points to scientific integrity rather than deliberate bias.

8. Protocol, if you choose to use it

Form: monohydrate. No alternative form (HCl, chelate, ethyl ester, buffered) has demonstrated superiority in absorption, retention or outcomes, and all are considerably more expensive.

Dosing:

- *With loading:* 20-25 g/day split into 4-5 doses for 5-7 days, followed by 3-5 g/day maintenance. Saturates in roughly a week.
- *Without loading:* 3-5 g/day from the start. Saturates in 3-4 weeks. Less gastrointestinal discomfort and less acute water retention.

Timing: irrelevant. The mechanism is saturation, not acute action.

Continuity: continued use maintains saturation. There is no evidence supporting any need to cycle.

Quality: look for a recognised quality seal (Creapure, Informed Sport or equivalent), especially in athletes subject to anti-doping control.

9. Conclusions for professional practice

1. **It is not a necessary supplement.** It is an adjunct with a small effect, conditional on individual context.
2. **Training stimulus and energy balance explain the vast majority of the result.** Any discussion of creatine that precedes the discussion of volume, progression, sleep and protein intake is badly prioritised.
3. **The candidates most likely to respond** are those starting from low stores: vegetarians and vegans, people with low meat intake, older adults, and possibly women.

4. **The effects are reversible.** It is honest to tell the client: if they stop, they will lose the water weight and the performance margin, though they will keep the adaptation earned through the work done.
5. **The safety profile is solid** in healthy individuals at conventional doses, with the caveat of the analytical interference with serum creatinine.
6. **The non-sport applications** — cognition under metabolic stress, sarcopenia, bone health — may end up being the strongest argument in favour of the supplement, ahead of gym performance.
7. **Absence of supplementation is not a deficiency.** A professional with two decades of training and an omnivorous diet is predictably close to the saturation ceiling through food alone. Their lack of subjective response is an expected result, not an anomaly.

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