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Does Collagen Supplementation Support Musculoskeletal Recovery? A Narrative Review of Tendon, Ligament and Muscle Adaptation

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Abstract

Introduction and aim: Collagen accounts for ~1/3 of body protein and forms the structural core of tendons, ligaments, and skeletal muscle extracellular matrix (ECM). Oral hydrolyzed collagen or collagen peptide (CP) supplementation has gained popularity, yet clinical evidence lags marketing claims. Results vary, several trials report null effects, and key questions on dosing, tissue specificity, and cofactors such as vitamin C remain open. This review critically appraises CP effects on tendon morphology and mechanics, ligament remodeling, and post-exercise muscle recovery.

Material and method: Searches (April 2026) covered PubMed, Scopus, Web of Science, and Google Scholar. Terms included: collagen peptides, hydrolyzed collagen, tendon remodeling, ligament, exercise-induced muscle damage, DOMS, ECM, and musculoskeletal health. English-language RCTs, systematic reviews, and meta-analyses were prioritized.

Results: For tendons, trials report increased cross-sectional area (CSA) and stiffness with 15–30 g/day CP combined with resistance or plyometric training. However, at least two RCTs found no effect on mechanical properties and certainty of evidence is low-to-moderate. Human

ligament data are lacking as the field relies on in vitro and biomarker studies. Muscle recovery data are contradictory: some trials show attenuated creatine kinase release and improved neuromuscular function while stable isotope tracer studies found no rise in intramuscular connective protein synthesis after CP ingestion, undermining the proposed mechanism.

Conclusions: Evidence for collagen supplementation in musculoskeletal health is weaker than its commercial prominence suggests. A modest signal for tendon CSA exists, but mechanical effects are inconsistent. For ligament repair and muscle recovery, evidence is indirect or negative. Broad recommendations are premature. Future RCTs need longer interventions, female populations, active protein controls, and clinically meaningful endpoints.

Keywords: *collagen supplementation; musculoskeletal health; tendons; ligaments; muscle recovery; exercise-induced muscle damage; connective tissue; extracellular matrix; collagen peptides*

Introduction

Collagen makes up a substantial proportion of total body protein (estimated at around 30%) and is a major structural component. At least 28 genetically distinct collagen types have been already described, but types I, II, and III are the most important to the musculoskeletal system [1,2]. Type I collagen, produced most importantly by fibroblasts and tenocytes, is the most common in tendons, ligaments, bone, and the intramuscular connective tissue (IMCT). Its repeating Gly-X-Y triple-helical motif, with around 30% glycine and 15-20% proline or hydroxyproline, gives it unique tensile strength [3,4].

Tendons transfer force from muscle to bone, while ligaments stabilize joints by resisting nonphysiological movement. Tendons and ligaments are relatively poorly vascularized and have limited regenerative capacity following injury [5]. The skeletal muscle ECM is equally rich in collagen and turns over at around 0.5-2% per day [6,7]. Apart from structural support, the IMCT also contributes to the lateral transmission of contractile force from myofibrils to tendinous insertions [8].

Oral supplementation with hydrolyzed collagen or CP delivers low molecular weight peptides that are detectable in blood within 60 minutes of ingestion. In cell culture experiments, collagen-derived dipeptides such as prolyl-hydroxyproline (Pro-Hyp) stimulate fibroblast collagen synthesis in a dose-dependent way [9,10]. A study by Shaw et al. [11] showed that 15

g of gelatin enriched with vitamin C, taken one hour before exercise, significantly elevated serum P1NP (procollagen type I N-terminal propeptide, a validated collagen synthesis marker) compared to 5 g and placebo.

The purpose of adding vitamin C comes from its biochemistry. Prolyl hydroxylase and lysyl hydroxylase, both essential for collagen cross-linking, are vitamin C-dependent [3,4]. Conventional high-quality proteins (whey, casein) contain about 6% proline and 2% glycine, whereas hydrolyzed collagen contains around 20% proline/hydroxyproline and 33% glycine [9,12,13]. This compositional gap is central to the hypothesis that collagen, unlike standard protein supplements, can selectively raise the availability of precursors for connective tissue synthesis.

Although theoretically correct, the clinical literature is far from convincing. Several RCTs have found no between-group difference in tendon mechanical properties [14,15], and stable isotope studies report no acute increase in intramuscular collagen synthesis following oral collagen intake [16]. The theoretical rationale for the supplementation hypothesis has not translated reliably into measurable tissue-level benefits in humans. Differences between trial results likely reflect variation in dosing, supplement form, training protocols, population characteristics, and outcome measurement, but also, possibly, publication bias in favour of positive findings.

Material and Methods

This narrative review is based on evidence from randomized trials, systematic reviews and mechanistic studies identified through PubMed, Scopus, Web of Science and Google Scholar. Keyword combinations included: "collagen supplementation", "collagen peptides", "hydrolyzed collagen", "tendon CSA", "tendon stiffness", "ligament remodeling", "anterior cruciate ligament," "EIMD", "DOMS", "extracellular matrix", "musculoskeletal recovery", "vitamin C collagen", “collagen supplementation safety”.

Randomized controlled trials (RCTs), systematic reviews, and meta-analyses published in English were the primary target. Animal and in vitro studies were admitted only where human data were unavailable, particularly for ligament-specific outcomes.

Results and Discussion

1. Collagen Structure, Biosynthesis, and the Case for Supplementation

Collagen is synthesized in several steps, starting in the rough endoplasmic reticulum, where individual pro-alpha chains are assembled. A key step is the hydroxylation of proline and lysine residues, both requiring vitamin C, which enables triple-helix formation (procollagen). After secretion, procollagen peptidases cleave the propeptide regions to yield tropocollagen, which self-assembles into fibrils stabilized by lysyl oxidase-mediated cross-links [1,3,4].

From a supplementation perspective it's worth taking into account the fact that vitamin C availability directly limits cross-link formation. Supplementing collagen without adequate ascorbic acid may deliver amino acid precursors that cannot be properly incorporated into mature fibrils. Second, the high glycine and proline content of collagen hydrolysate (~33% and ~20% respectively [9,12,17]) distinguishes it from whey or casein, which are rich in leucine and branched-chain amino acids but provide only around 2–6% of these connective tissue-specific residues.

Pharmacokinetically, Pro-Hyp and Hyp-Gly dipeptides appear in blood within 60 minutes of ingesting 15 g gelatin, peak at around 90 minutes, and persist for several hours [10,11]. Their concentration in serum from a 15 g gelatin condition, when applied to engineered ligament constructs in vitro, significantly increased collagen content compared with serum from a 5 g dose or placebo. This finding forms the basis of the pre-exercise supplementation strategy [11].

2. Tendon Morphology and Mechanical Properties

Tendons adapt to mechanical loading by increasing CSA, stiffness, and collagen fibril density. High-intensity resistance training (≥70% 1RM) is the well-known stimulus. Collagen supplementation has been proposed to amplify this response. Most studies have focused on the patellar and Achilles tendons.

Simon Jerger et al. contributed two RCTs. In the first [18], 40 healthy males performed 14 weeks of resistance training while receiving either 5 g specific collagen peptides (SCP) or placebo daily. Achilles tendon (AT) CSA increased significantly more in the SCP group (+11.0% vs. +4.7%, $p = 0.002$); muscle thickness followed a similar pattern (+7.3% vs. +2.7%,

p =0.014). However, tendon stiffness did not differ between groups, they both increased similarly with training. A follow-up trial with the same protocol targeting the patellar tendon [19] found greater patellar tendon (PT) CSA hypertrophy in the SCP group, again with no between-group difference in stiffness. Lee et al. [20] took a different approach, studying elite female soccer players over a 10-week in-season training period. Here, CSA did not change in either group, but the collagen group showed significantly greater increases in PT stiffness (+18.0% vs. +5.1%) and Young's modulus (+17.3% vs. +4.8%; $p < 0.05$ for both). These differences between studies, CSA gains without stiffness differences versus stiffness gains without CSA changes, are difficult to explain and possibly reflect differences in training intensity, population, and tendon condition at the start.

Not all studies have reported positive effects. Balshaw et al. reported these findings in companion papers from a single 15-week RCT: a tendon-focused analysis [14] and a muscle/function-focused analysis [15]. They both presented no effect of CP on the respective tendon mechanical properties or on tendinous CSA, despite using high-resolution MRI. Those two trials are a serious challenge to the supplementation hypothesis.

The 2026 systematic review by Buchalski et al. [21], covering 8 RCTs, applied GRADE criteria and concluded: Grade A evidence for increased tendon CSA and stiffness with 15-30 g/day CP combined with resistance training, and Grade A evidence against an additive effect on muscle strength. The GRADE rating reflects consistency and effect size across the included trials, but it does not override the fact that trials using lower doses or employing more rigorous imaging consistently failed to show effects. The 2024 meta-analysis by Bischof et al. [22], pooling 19 RCTs ($n = 768$), found a statistically significant overall effect on tendon morphology, but rated the certainty as very low. The effect on tendon mechanical properties was non-significant ($SMD = 0.05$, $p = 0.82$), meaning that even a pooled analysis of nearly 800 participants cannot demonstrate that collagen supplementation makes tendons mechanically stronger or stiffer.

Overall, the tendon data present a mixed and an unclear picture. CSA increases are reported in some positive trials, but CSA alone, a morphological surrogate, does not guarantee improved tendon function or injury resilience. Effects on mechanical properties, which are the outcomes that actually matter clinically, are inconsistent across studies, and two RCTs found no effect at all. The field would benefit considerably from pre-registered trials with adequately

powered samples and imaging methods sensitive enough to detect small but real effects if such effects exist.

3. Ligaments: Biological Plausibility Without Direct Human RCT Evidence

Ligaments share most structural features with tendons, especially type I collagen, fibroblast-maintained matrix, low vascularity. However, they differ in their loading profile, joint-stabilizing function, and the content of type III collagen [5]. Healing after acute ligament injury is notoriously incomplete. Scar tissue, rich in type III collagen, has inferior mechanical properties compared to native ligament [23], which is why chronic joint instability is common after rupture, particularly in the case of ACL injuries [24].

There is currently no published RCT that has used oral collagen supplementation as a sole intervention and measured outcomes in human ligament tissue directly. The evidence rests primarily on three lines of indirect argument. First, the Shaw et al. [11] engineered ligament experiment in which blood serum collected 1 hour after 15 g gelatin ingestion, when applied to human-engineered ligament constructs in culture, increased collagen content and mechanical properties compared to serum from 5 g or placebo conditions. This is a cell culture model, not an in vivo study, and the relevance to intact human ligament repair is uncertain. Second, Nyland et al. review [25] proposed that glycine-rich collagen peptides may help maintain ACL collagen integrity under repetitive loading by supporting ECM turnover. Third, a comparative cohort study of patients after ACL reconstruction found better functional outcomes in a group receiving a multi-ingredient combination including collagen versus PRP alone [26]. This result cannot be attributed to collagen specifically.

The theoretical argument is strengthened by isotope turnover data showing that ligament tissue shows protein synthesis rates comparable to or slightly exceeding those of tendon (~1.5-2% per day in some compartments) [27]. It suggests responsiveness to nutritional inputs. Vitamin C co-administration would be mechanistically essential for the same reasons as in tendon since lysyl hydroxylase and prolyl hydroxylase are both ascorbate-dependent [3,4].

The conclusion here is that the biological case has appeal, but clinical evidence is essentially absent. Promoting collagen supplementation for ligament repair or ACL rehabilitation on the basis of a cell culture experiment, a narrative review, and a confounded retrospective analysis is a weak ground for clinical recommendation. The gap between mechanistic plausibility and demonstrated clinical benefit is important, considering how

common ACL injuries are. Until adequately powered RCTs with direct ligament tissue outcomes are published, recommending collagen supplementation in this context is speculative at best.

4. Muscle Recovery After Exercise-Induced Damage (EID)

Evidence on muscle recovery represents perhaps the most contested area in the collagen supplementation literature and the area where mechanistic findings most directly contradict clinical claims.

Holwerda and van Loon [9] reviewed evidence showing that myofibrillar protein synthesis is robustly stimulated by conventional high-quality proteins, while intramuscular connective tissue (IMCT) protein synthesis is not. Likely because whey and casein are low in glycine and proline. This led to the hypothesis that collagen supplementation might preferentially stimulate IMCT synthesis. The hypothesis was attractive, but it has not held up. The stable isotope tracer RCT by Aussieker et al. [16], the most direct test of the mechanism to date, found that collagen protein ingestion did not increase muscle connective protein synthesis rates above whey or placebo in resistance-exercised young athletes. If collagen supplementation does not increase intramuscular collagen synthesis, the rationale for recommending it for muscle recovery loses its main mechanistic argument.

On the clinical side, two RCTs provide some supporting data. Bischof et al. [28] assigned 55 sedentary males to 15 g SCP or placebo alongside 12 weeks of concurrent training. After the training period, EID was induced by 150 drop jumps. The SCP group showed significantly better preservation of Maximal Voluntary Contraction (MVC) ($p = 0.02$) and rate of force development in the post-exercise recovery period. A subsequent study by Bischof et al. [29] found significantly lower CK elevation at 24 hours post-exercise in the SCP group.

The study by Robberechts et al. [30] is the most practically relevant comparison yet published. Rather than contrasting CP against a non-protein placebo, the design used in most positive trials, which conflates the effects of any protein intake with those of collagen specifically, it directly compared whey protein against a mixture of whey and collagen peptides during a 3-week eccentric training protocol in fit males. Partly replacing whey with CP produced no benefit for muscle damage indices or recovery of functional capacity. For athletes who already consume adequate total protein, this result suggests that the supposed advantages of collagen over conventional protein sources may not materialise in practice.

The umbrella review by Ravindran et al. [31], synthesizing 16 systematic reviews (113 RCTs, 7,983 participants), found no statistically significant effect on acute strength recovery at 24 or 48 hours, and no effect on muscle soreness at any time point. With nearly 8,000 participants, this is the largest body of evidence available and its inability to detect an acute ergogenic effect cannot be explained by insufficient sample size. The authors' suggestion that collagen effects are structural and chronic rather than acutely ergogenic may be correct, but this argument is hard to test empirically without much longer trials.

On body composition, some results appear more consistent, but require careful interpretation. Zdzieblik et al. [32] showed that 15 g/day CP combined with 12 weeks of resistance training significantly increased fat-free mass and muscle strength while reducing fat mass in elderly sarcopenic men. Kirmse et al. [33] found similar results in young recreationally active. However, biometrical impedance analysis (BIA)-derived and DXA-measured fat-free mass cannot distinguish contractile muscle hypertrophy from increases in connective tissue, extracellular water, or other non-contractile lean tissues. Because all of these components contribute to the lean mass signal, apparent increases in 'muscle mass' may partly reflect limitations of the measurement technique rather than true myofibrillar hypertrophy. [34,35]. Balshaw et al. [14] used high-resolution MRI and found greater quadriceps and vastus medialis volume in the CP group which is more compelling.

5. Dosing, Timing, and Practical Considerations

The dose of 15 g/day is the most consistently studied in positive trials and dose-response data suggest this threshold matters. Studies using 5 g/day often show CSA hypertrophy without mechanical property effects, while 15–30 g/day more reliably show stiffness improvements in tendons [21]. Balshaw et al. [14,15] used a patented CP formulation at a higher dose without benefit, so the picture is not simply "more is better."

Pre-exercise timing (30–60 minutes before mechanical loading) is recommended based on pharmacokinetics. This puts peak plasma collagen peptide concentrations within the window of exercise-induced upregulation of tendon collagen synthesis [11]. Post-exercise supplementation is less studied but may also have merit for recovery purposes. Co-administration with ≥50 mg vitamin C is mechanistically justified and incorporated in most study designs.

Exercise as a co-stimulus is not optional. This point deserves emphasis. Multiple reviews and meta-analyses [21,22,31] confirm that the connective tissue effects of collagen supplementation are observed almost exclusively in studies that include structured resistance or plyometric loading. Supplementation alone appears insufficient to drive meaningful tendon remodeling. While proponents frame this as an important interaction to exploit, a more frugal interpretation is that the loading stimulus alone may account for the observed effect, and the marginal contribution of collagen above that of any adequate protein source remains unproven.

Regarding supplement form, hydrolyzed collagen and CP appear bioavailable and broadly equivalent in terms of published outcomes. Specific patented formulations (FORTIGEL, TENDOFORTE) have been used in several trials but have not been shown to be superior to generic hydrolysates in head-to-head comparisons. Marine collagen from fish skin is increasingly available as an alternative for individuals avoiding land-animal products, with preliminary evidence of comparable bioavailability [12].

6. Safety

Clinical trials generally report that oral collagen supplementation is well tolerated, with no serious treatment-related adverse events and no clinically meaningful alterations in routine hepatic or renal safety markers during short-term supplementation in healthy individuals. Mild gastrointestinal symptoms, including bloating and fullness, have occasionally been reported at higher doses. Allergic reactions appear uncommon but may depend on collagen source, particularly in fish- or egg-allergic individuals using marine-derived or eggshell membrane products. Long-term safety beyond approximately 12 months remains insufficiently characterized [36,37]

The bigger issue is product quality rather than collagen itself. In the European Union, the United States, and most other major jurisdictions, dietary supplements are regulated as food rather than as medicinal products, which means that premarket evaluation of efficacy is not required. Batch testing is not routinely mandated and adverse event reporting is largely voluntary [38]. Heavy metal contamination is a recurring finding in retail-product surveillance. A 2025 analysis of 120 marine collagen samples reported mean arsenic concentrations of 0.59 mg/kg with up to fivefold variability between brands and lead detectable in nearly all fish-derived products. None of the samples exceeded EU regulatory limits at recommended daily doses, but the variability itself is informative about manufacturing inconsistency [39].

Augustsson et al. [40] reported broadly comparable findings across a wider category of food supplements. Bovine and porcine sources are not exempt. Environmental contamination of grazing land and feed can introduce lead and cadmium into the hide and bone material used as starting input. The European Union sets harmonised maximum levels for these contaminants in food and food supplements (Commission Regulation 2023/915), but enforcement relies primarily on official sampling rather than mandatory pre-release testing by manufacturers. Two products labelled identically as '15 g hydrolyzed collagen peptides' can in practice contain different amounts of protein and different levels of contaminants, with no requirement that the difference appear on the label.

Several populations remain notably understudied. Pregnant and lactating women have not been included in the published trials, and supplementation during these periods cannot be recommended on the available evidence. Pediatric data are likewise unavailable. In patients with chronic kidney disease at stages 4 to 5, in whom dietary protein is often clinically restricted, an additional 15 to 30 g/day of collagen peptides should not be regarded as a trivial addition to total intake and consultation with a nephrologist is appropriate before initiating use. At studied doses collagen peptides appear to have a favourable acute safety profile in healthy adults, but the limited representation of women and older participants outside specific sarcopenia studies, the absence of trials beyond a year, and the regulatory gap between supplement and pharmaceutical quality control together justify a more cautious framing than is typical of consumer-facing marketing.

Conclusions

The evidence for collagen supplementation as a nutritional adjunct to musculoskeletal health is insufficient. For tendon CSA, a modest and somewhat consistent signal exists in positive trials, but CSA is a surrogate marker and the GRADE A rating from Buchalski et al. [21] should be read alongside the fact that the pooled effect on tendon mechanical properties in the larger meta-analysis was statistically indistinguishable from zero [22]. Two RCTs using high-resolution MRI found no benefit at all. For ligament tissue, the case rests entirely on in vitro data and indirect inference. There is no human RCT evidence. For acute muscle recovery, both the best mechanistic study available [16] and the largest evidence synthesis [31] returned negative results. The narrative that collagen supplementation is a meaningful adjunct to connective tissue health is driven more by biological plausibility and current enthusiasm may exceed the certainty of supporting clinical evidence.

From a practical standpoint, the available evidence does not justify a blanket recommendation for collagen supplementation in athletic or clinical populations. If a trial is considered, for instance, in individuals with tendinopathy who have not responded to structured loading alone, or older adults with sarcopenia, then the protocol supported by the most positive trials (15–30 g/day hydrolyzed collagen or CP with vitamin C, 30–60 minutes before resistance or plyometric training, for at least 8–12 weeks) is at least plausible and appears safe. However, the expectation of benefit should be modest and the cost-effectiveness of this approach compared with simply ensuring adequate total protein intake and progressive mechanical loading has not been established.

Further progress will require a more rigorous standard of evidence than the field has produced so far. Specifically larger, adequately powered RCTs with pre-registered primary endpoints and active protein controls (rather than non-protein placebo, which systematically inflates apparent CP effects). Studies with direct ligament tissue outcomes in post-injury rehabilitation, long-term follow-up beyond the 12–15 weeks typical of current trials and inclusion of female participants, who are rarely included in published trials. Until such trials are available, the enthusiasm for collagen supplementation should be tempered by an honest appraisal of what the current evidence does actually show and what it doesn't.

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