

Effects of Stretching on Creatine Kinase (CK) Level: A Systematic Literature Review

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Abstract

Exercise-induced muscle damage (EIMD) commonly occurs following strenuous or unaccustomed exercise, particularly eccentric exercise, and may result in delayed-onset muscle soreness, reduced muscle strength, stiffness, and impaired physical performance. Creatine kinase (CK) is widely used as an indirect biomarker of EIMD. This systematic literature review aimed to evaluate the effects of stretching on CK levels following EIMD. A systematic search was conducted in PubMed, Cochrane, and ScienceDirect from database inception to August 2026. Eligible studies included human randomized controlled trials or comparative experimental studies evaluating stretching, reporting CK levels, and including a control or comparative group. Of 202 records identified, 11 duplicates were removed. Following screening and full-text assessment, six studies were included. All six studies evaluated static stretching. Three studies reported lower CK levels at specific post-exercise time points compared with passive or no-intervention controls, although these findings were not consistently supported by overall group-by-time analyses. High-intensity stretching showed an unfavorable CK response, while static stretching was less effective than active recovery and cold-water immersion. No significant difference was observed between static stretching and foam rolling. Overall, stretching, particularly static stretching, did not consistently reduce CK levels following EIMD. The available evidence is limited by small sample sizes and heterogeneity in stretching protocols, exercise stimuli, and CK measurement methods. Therefore, static

stretching cannot currently be considered a reliable strategy for reducing CK following EIMD.

Keywords: creatine kinase, exercise-induced muscle damage, muscle recovery, stretching

Introduction

Exercise-induced muscle damage (EIMD) is a transient physiological response that most commonly occurs following strenuous or unaccustomed exercise, particularly eccentric exercise in which an active muscle is lengthened under load. Unaccustomed eccentric exercise imposes nonuniform mechanical strain on muscle fibers and sarcomeres, initiating disturbances in sarcomeric, cytoskeletal, sarcoplasmic-reticulum, and membrane systems. These initial mechanical disturbances can subsequently interact with calcium dysregulation, metabolic stress, oxidative processes, and immune responses, producing a cascade that contributes to muscle dysfunction and subsequent tissue repair¹.

Clinically, EIMD is characterized by delayed-onset muscle soreness, reduced voluntary force, muscle stiffness, reduced range of motion, and impaired capacity for high-intensity exercise, with the severity and duration of these effects varying according to exercise modality, training status, and individual characteristics². Although EIMD is generally considered a reversible stress-and-repair response, it can temporarily impair physical performance and may have important consequences for subsequent exercise sessions³.

Creatine kinase (CK) is a practical biochemical marker commonly used to assess the response to strenuous exercise, particularly unfamiliar eccentric exercise. CK is an enzyme involved in the rapid regeneration of adenosine triphosphate (ATP), and skeletal muscle contains high concentrations of the CK-MM isoenzyme. Following exercise-induced muscle damage, mechanical and metabolic stress can increase muscle membrane permeability, allowing intracellular CK to enter the circulation⁴. Therefore, an increase in circulating CK can reflect muscle stress and membrane disruption associated with EIMD, making it a useful outcome for evaluating the response to different recovery interventions⁵. However, CK is an indirect marker rather than a direct

measure of structural muscle damage, and its magnitude and timing are highly variable between individuals and exercise protocols⁶. CK concentrations typically peak several days after the initial exercise insult, with the timing influenced by factors such as exercise intensity and volume, muscle group, training status, and previous eccentric exposure⁴. Consequently, serial CK measurements interpreted alongside functional and symptomatic outcomes are more informative than a single post-exercise measurement when assessing EIMD and recovery⁶.

Despite the widespread use of stretching as a post-exercise recovery strategy, its effect on biochemical markers of exercise-induced muscle damage remains uncertain. Previous evidence has primarily focused on outcomes such as delayed-onset muscle soreness, strength, range of motion, and performance, while evidence regarding CK is more limited and has produced inconsistent findings. Differences in stretching intensity, duration, frequency, exercise protocols, and CK measurement may contribute to these inconsistencies. Therefore, a systematic review is needed to synthesize the available evidence regarding the effects of stretching on CK levels following exercise-induced muscle damage. This systematic literature review aims to evaluate the effects of stretching on CK levels following exercise-induced muscle damage in human participants.

Method

This systematic review was conducted based on Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). Eligibility Criteria: studies were considered eligible if they were original research articles or research reports involving human participants and employing a randomized controlled trial design. Studies were excluded if they were narrative reviews, systematic reviews, meta-analyses, non-comparative studies, *in silico*, *in vitro*, or *in vivo* studies, technical reports, editor responses, scientific posters, study protocols, or conference abstracts. Articles with unavailable full texts, publications in languages other than English, and studies unrelated to the research topic were also excluded.

Search Strategy: this study acquired studies by searching databases such as PubMed, Cochrane, and ScienceDirect. The search was conducted from the inception of each database until August 2026. The search strategy used combinations of keywords

and Boolean operators, including “stretching,” “stretching exercise,” “stretch exercise,” “muscle stretching,” “creatine kinase,” “creatine phosphokinase,” and “CPK.” The search terms were adapted as necessary to the specific requirements of each database. In addition to database searching, the reference lists of relevant articles and included studies were manually screened to identify additional potentially eligible studies that may not have been retrieved through the electronic database search. The retrieved studies were stored and managed using reference management software.

Study Criteria include inclusion and exclusion criteria. *Inclusion Criteria:* In this systematic literature review, the author determined the study criteria. The inclusion criteria were: (1) studies involving human participants, (2) studies evaluating stretching as an intervention, (3) studies reporting creatine kinase (CK) levels as an outcome, (4) randomized controlled trials or comparative experimental studies, (5) studies with a control or comparator group, and (6) full-text articles published in English or Bahasa Indonesia. The exclusion criteria in this systematic literature review were: (1) animal or non-human studies, (2) review articles, systematic reviews, meta-analyses, case reports, case series, editorials, letters, study protocols, and conference abstracts, (3) studies that did not evaluate stretching or CK levels, (4) studies without a control or comparator group, (5) articles with unavailable full text, and (6) duplicate publications.

Results

Study Selection

A total of 202 records were identified through database searching. Before screening, 11 duplicate records were removed, with no records removed by automation tools or for other reasons, leaving 191 records for screening. During title and abstract screening, 177 records were excluded, and 14 reports were sought for retrieval. Of these, 4 reports could not be retrieved, leaving 10 reports assessed for eligibility. Following full-text assessment, 4 reports were excluded, consisting of 1 study with an inappropriate study method and 3 studies with an inappropriate intervention. Ultimately, 6 studies were included in the systematic literature review.

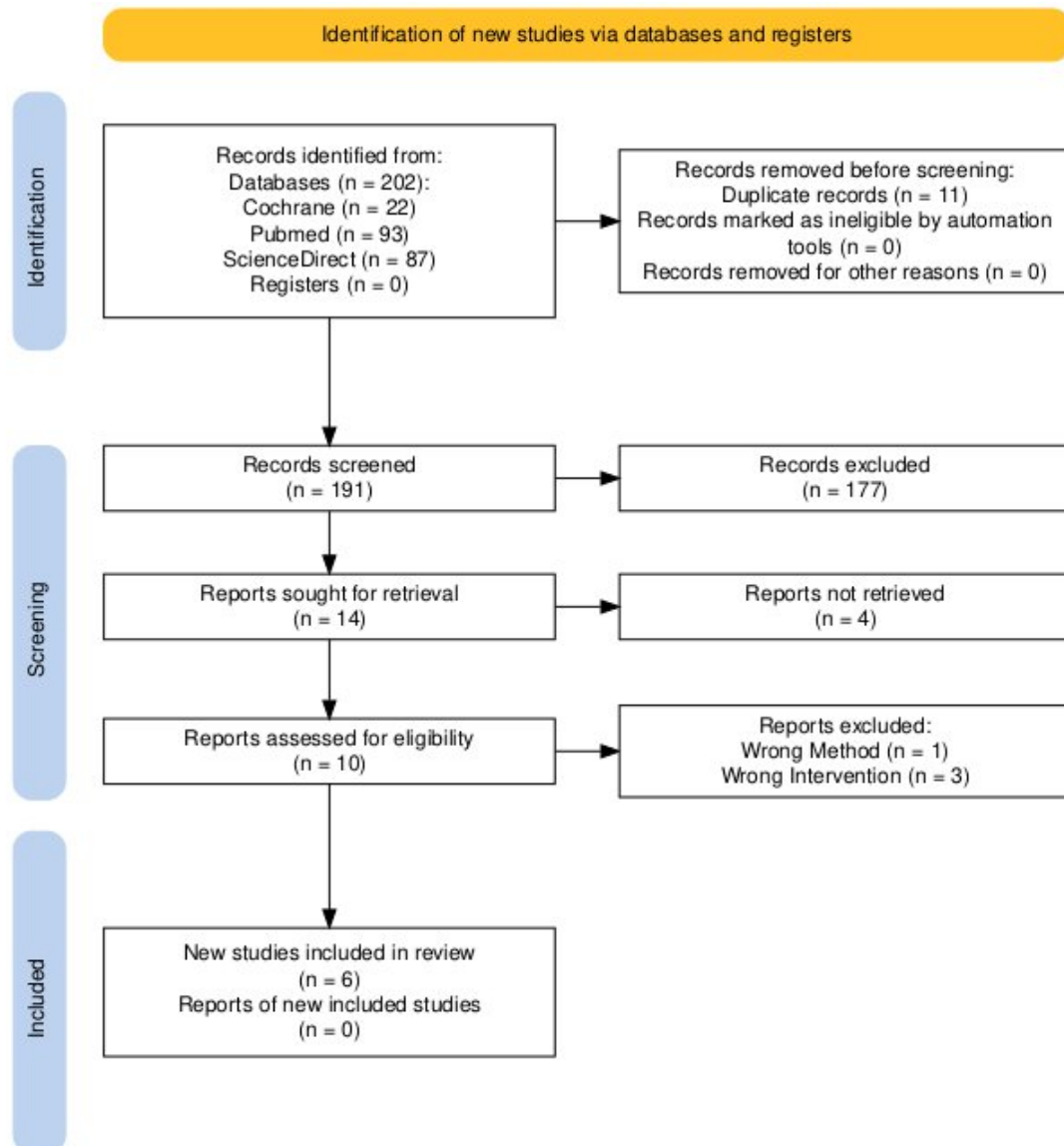


Figure 1. PRISMA Diagram

Characteristics of Included Studies

Six studies met the screening criteria and were included in the systematic literature review. All included studies evaluated static stretching as a post-exercise intervention following exercise protocols intended to induce muscle damage. Three studies used a crossover design, while the other three were parallel-group randomized controlled trials. The sample sizes were relatively small, ranging from 10 to 15 participants in the crossover studies and 5 participants per group in the parallel-group trials. The study populations consisted of healthy or physically active individuals, including experienced runners, recreational active men, healthy male university

students, and elite youth soccer players.

The exercise protocols used to induce muscle damage varied among studies. These included downhill treadmill running, competitive soccer matches, eccentric knee-extension exercise, incremental treadmill exercise, and loaded squat-jump exercise. Static stretching protocols also varied in duration, frequency, and intensity. The total stretching duration per muscle group ranged from 30 to 180 seconds, with interventions ranging from a single post-exercise session to three consecutive days of stretching. Comparators included passive or no-intervention control, active recovery, cold-water immersion, and foam rolling.

All six studies measured CK levels at multiple time points following the exercise protocol, although the measurement methods and reported units differed between studies. Two studies appeared to use the same data set, as they reported identical baseline and follow-up CK values. Therefore, these studies should be considered carefully when interpreting the overall body of evidence to avoid treating potentially overlapping data as independent findings.

Effects of Stretching on CK Levels

Across the six included studies, the effects of static stretching on creatine kinase (CK) levels following exercise-induced muscle damage were inconsistent. Two studies reported significantly lower CK levels following static stretching compared with no-intervention control, whereas other studies found no significant benefit or reported higher CK levels compared with other recovery interventions^{7,8,9,10,11}.

In the previous study, participants who performed static stretching demonstrated lower CK levels than the no-intervention control at 48 hours after exercise ($p < 0.05$), although the overall group-by-time interaction was not statistically significant ($p = 0.150$).¹² CK levels in the stretching group decreased from 278.51 ± 53.72 U/L immediately after exercise to 227.04 U/L, 184.47 U/L, and 159.59 U/L at 24, 48, and 72 hours, respectively, while the control group showed slower reductions during the same period¹³. A second previous study, which appears to involve the same dataset, reported significantly lower CK levels in the stretching group at 24 and 48 hours compared with control, but not at 72 hours¹⁴.

Other research also found a significantly lower absolute CK level in the static

stretching group than in the passive recovery group at 48 hours ($p = 0.032$; $ES = 0.427$).¹⁵ However, the difference was no longer statistically significant when CK was expressed as percentage change from baseline, suggesting only a modest effect of stretching¹⁵. In contrast, previous study found no significant time-by-condition interaction ($p = 0.148$). Their analysis indicated that high-intensity static stretching was likely harmful for CK at 48 hours ($d = 0.49$) and 72 hours ($d = 0.37$) compared with control, while low-intensity stretching was possibly harmful at 48 hours ($d = 0.32$)¹⁶.

When static stretching was compared with other active recovery strategies, the results were less favorable. Other research was reported significantly higher CK levels 48 hours after exercise in the static stretching condition compared with both active recovery ($p = 0.020$; partial $\eta^2 = 0.468$) and cold-water immersion ($p = 0.031$; partial $\eta^2 = 0.430$). CK remained elevated above baseline after static stretching, whereas CK returned to baseline following cold-water immersion¹⁵. Similarly, previous study found no significant group-by-time interaction for CK between static stretching and either vibrating or non-vibrating foam rolling ($p = 0.50$), suggesting no stretching-specific effect on CK recovery¹⁷.

Overall, static stretching did not demonstrate a consistent effect on CK levels following exercise-induced muscle damage. Although some studies showed a reduction in CK at specific time points^{15,17}. These findings were inconsistent across analyses and comparators, while higher-intensity stretching and comparisons with active recovery modalities suggested no benefitor potentially unfavorable effects^{16,17}.

Table 1. Characteristics of Included Studies

Study	Design	Population / Sample	Stretching Intervention	Comparator
Wei et al., 2025	RCT	Healthy male university students; n = 30	Static; 3 × 30 s; 80% ROM	No intervention
Wei et al., 2025a	RCT	Healthy male sports-science students; n = 30	Static; 3 × 30 s; 80% ROM	No intervention
Pooley et al., 2017	Crossover	Elite male youth soccer players; n = 10	Static; 2 × 15 s/group	Passive recovery
Apostolopoulos et al., 2018	RCT	Recreationally active males; n = 30	Static; 3 × 60 s; low/high intensity	No stretching
Pooley et al., 2020	Crossover	Elite youth soccer players; n = 15	Static; 2 × 15 s/group	Active recovery + CWI
Wu et al., 2026	Crossover	Experienced adult runners; n = 11	Static; 4 × 30 s/group	Foam rolling

Table 2. Effects of Stretching on Creatine Kinase Levels Following Exercise-Induced Muscle Damage

Study	CK unit	Baseline CK: Stretching	Baseline CK: Comparator	Main CK finding	Statistical significance	Effect size	Direction of effect
Wei et al., 2025	U/L	138.75 ± 12.31	137.95 ± 14.03	CK was lower with static stretching than control at 48 h; no significant difference at other time points	48 h: p < 0.05 ; group × time interaction: p = 0.150	Partial η^2 = 0.242 for interaction	Numerically lower CK with stretching from 24–72 h; significant at 48 h
Wei et al., 2025a	U/L	138.75 ± 12.31	137.95 ± 14.03	CK was significantly different from control at 24 and 48 h, but not at 72 h	24 h: p < 0.05 ; 48 h: p < 0.05 ; 72 h: NS; interaction p > 0.05	Partial η^2 = 0.242 for interaction	Numerically lower CK with stretching at 24–72 h; significant at 24 and 48 h
Pooley et al., 2017	Not specified; fingertip whole-blood	2.8 (1.7)	2.9 (1.6)	CK was lower with static stretching than passive recovery at 48 h based on absolute values; percentage change from baseline was not significantly different	Absolute 48 h: p = 0.032 ; percentage change: p > 0.05	ES = 0.427	Small reduction favoring stretching
Apostolopoulos et al., 2018	U/L	Low: 316.5 ± 271.4; High: 239.4 ± 183.6	297.9 ± 187.1	No significant overall time × condition interaction; high-intensity stretching was likely harmful for CK at 48 and 72 h, while low-intensity stretching was possibly harmful at 48 h	Interaction: p = 0.148	Low vs control, 48 h: d = 0.32; High vs control, 48 h: d = 0.49; High vs control, 72 h: d = 0.37	Unfavorable effect, particularly with high-intensity stretching
Pooley et al., 2020	ng/mL	2.8 (1.7)	AR: 3.0 (1.0); CWI: 3.2 (1.2)	CK was significantly higher after static stretching than after active recovery and CWI at 48 h; CK remained elevated above baseline after stretching, whereas CWI returned to baseline	Stretching vs AR: p = 0.020 ; stretching vs CWI: p = 0.031	Partial η^2 = 0.468 (AR); 0.430 (CWI)	Stretching inferior to active recovery and CWI
Wu et al., 2026	Not reported	Not reported	Not reported for VR/NVR	No significant group × time interaction for CK between static stretching and foam rolling	Interaction: p = 0.50 ; time: p < 0.01 ; group: p < 0.001	Not reported	No stretching-specific effect

Discussion

This systematic literature review included six studies evaluating the effects of static stretching on creatine kinase (CK) levels following exercise-induced muscle damage^{12,13,14,15,16,17}. Overall, the findings did not demonstrate a consistent beneficial effect of stretching on CK reduction. Three studies reported lower CK levels following stretching at specific post-exercise time points compared with passive or no-intervention controls. However, these findings were not consistently supported by measures of relative change or overall group-by-time interactions^{14,15,6,17}. In contrast, previous study found no significant time-by-condition interaction and reported that high-intensity static stretching was likely harmful for CK recovery at 48 and 72 hours, while low-intensity stretching was possibly harmful at 48 hours¹⁷. Furthermore, when compared with other recovery strategies, static stretching resulted in significantly higher CK levels than active recovery and cold-water immersion at 48 hours, while no significant difference was observed compared with foam rolling^{15,16}.

The inconsistent effects of stretching on CK levels across the included studies may be explained by differences in the intensity, dose, and timing of stretching, as well as differences in the exercise protocols used to induce muscle damage. The included studies applied substantially different stretching protocols, ranging from 2×15 seconds per muscle group to 3×60 seconds per muscle group over three consecutive days, with stretching intensity varying from mild discomfort to 70–80% of perceived maximum or approximately 80% of range of motion^{16,17}.

Among these factors, stretching intensity appears particularly relevant. Apostolopoulos et al. reported that high-intensity static stretching was likely harmful for CK at 48 and 72 hours compared with no stretching, whereas low-intensity stretching did not demonstrate a significant CK-lowering effect. The same study used three 60-second sets of stretching over three consecutive days, with intensity ranging from 3–4/10 to 7–8/10¹⁸. Evidence from other stretching studies similarly suggests that increasing the duration or frequency of acute stretching does not consistently reduce CK. A five-minute before exercise static stretching protocol did not alter CK, while single and repeated post-exercise stretching protocols also failed to demonstrate a significant reduction^{17,18,19}. Therefore, a greater stretching dose does not necessarily produce greater biochemical recovery, and excessive stretching intensity may

potentially produce an unfavorable response.

Differences in the exercise protocols used to induce muscle damage may also explain variation in CK outcomes. The included studies used several forms of exercise, including downhill running, competitive soccer matches, eccentric knee extension, treadmill exercise, and loaded squat-jump exercise^{19,20,21,22,23,24}. The magnitude and time course of CK elevation depend strongly on the characteristics of the damaging exercise. Unaccustomed, high-force eccentric exercise generally produces greater CK elevations, while exercise intensity, volume, range of motion, and the muscle group involved can further influence the response¹⁸. Previous exposure to eccentric exercise may also attenuate subsequent CK elevation through the repeated-bout effect². Consequently, differences in the exercise stimulus and participants' previous training exposure may have contributed to the heterogeneous CK responses observed across studies.

Finally, the variability may partly reflect the characteristics of CK itself as an outcome measure. CK is an indirect marker of exercise-induced muscle damage, and its circulating concentration reflects both enzyme release and clearance rather than structural muscle damage alone^{22,23}. CK elevation is also delayed and may peak several days after the damaging exercise, meaning that different sampling schedules can produce different apparent treatment effects^{23,24}. In addition, large inter-individual variation in CK responses has been reported even after standardized eccentric exercise. Thus, differences in participant characteristics, exercise protocols, and CK measurement time points may have contributed to the inconsistent findings across the included studies¹⁸.

Static stretching showed different results depending on the recovery strategy used as the comparator. When compared with passive or no-intervention recovery, some studies reported lower CK levels after stretching at specific time points, although these findings were not consistently supported by overall group-by-time analyses or changes from baseline^{16,19,20,23,24}. In particular, previous study found a small reduction in absolute CK at 48 hours with static stretching compared with passive recovery, but the difference was no longer significant when CK was analyzed as a percentage change from baseline²⁰. Similarly, previous controlled studies found that repeated passive stretching did not significantly alter CK compared with no stretching^{15,16}.

Compared with active recovery, static stretching appeared less favorable for CK reduction. In elite youth soccer players, CK levels at 48 hours were significantly higher

after static stretching than after active recovery ($p = 0.020$; partial $\eta^2 = 0.468$)²⁰. However, evidence supporting active recovery as a specific CK-lowering intervention remains limited. A systematic review of active recovery protocols reported improvements in outcomes such as soreness, strength loss, flexibility, and inflammatory markers, but did not establish a clear CK-specific advantage over passive recovery¹⁴.

A similar pattern was observed when stretching was compared with cold-water immersion (CWI). Static stretching resulted in significantly higher CK levels than CWI at 48 hours in elite youth soccer players ($p = 0.031$; partial $\eta^2 = 0.430$), while CK following CWI returned to baseline²⁰. Broader evidence also indicates that CWI has a more consistent effect on CK than static stretching. A systematic review and meta-analysis involving 52 studies found that CWI reduced CK at 24 and 48 hours after high-intensity exercise compared with passive recovery, although this effect was not consistently demonstrated following isolated eccentric exercise¹⁶. Therefore, the apparent advantage of CWI over stretching may depend partly on the type of exercise that produced the muscle damage.

When compared with foam rolling, static stretching did not demonstrate a significant difference in CK response. A study was reported no significant group-by-time interaction between static stretching and vibrating or non-vibrating foam rolling ($p = 0.50$), suggesting that CK changes were more consistent with natural recovery than a stretching-specific effect²². Other comparative evidence likewise remains insufficient to demonstrate a CK-lowering advantage of foam rolling over passive recovery or other recovery modalities^{9,12}.

Limitation

This systematic review has several limitations, including article differed substantially, reason the meta-analysis not conducted and database coverage. First, the included articles have differences in animal model type used, blood collected time and variation of exercise intervention time. Then, meta-analysis was not analyzed because p-value standard was used different among study and some research were not consistently reported using comparable conditions. Finally, database coverage access to full-text reports may also have influenced the review.

Conclusion

Stretching, particularly static stretching, does not consistently reduce creatine kinase (CK) levels following exercise-induced muscle damage. Although some studies reported lower CK at specific time points compared with passive or no-intervention controls, these findings were inconsistent, while higher-intensity stretching may produce an unfavorable CK response. Compared with other recovery strategies, stretching was less favorable than active recovery and cold-water immersion, while its effect was comparable to foam rolling. Overall, due to the small sample sizes and heterogeneity in stretching protocols, exercise stimuli, and CK measurement, current evidence does not support static stretching as a reliable strategy for reducing CK following exercise-induced muscle damage.

Ethical Considerations

This manuscript is a systematic review of previously digital reported studies and did not require direct recruitment of human participants or animal subjects. Ethical approval and informed consent were therefore not required for this systematic review.

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Conflict of Interest

The authors declare that there is no conflict of interest related to this manuscript and database.

Author contribution

Arwan Bin Laeto conceptualized the review topics, mapped the literature, prepared the initial manuscript draft, and approved the final manuscript. Catherine Dwi Augusthi & M. Iman Tarmizi Thaher developed the stretching and creatine kinase framework and interpreted physiology findings. Rara Inggarsih, Septi Purnamasari & Masayu Farah Diba contributed to the interpretation of the methods and creatine kinase outcomes.

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