Journal of Stem Cell Research

Genesis-JSCR-7(3)-93
Volume 7 | Issue 3
Open Access
ISSN: 2582-8797

Centrifugation Determines What Is in the Syringe, Not What Happens to the Patient: A Systematic Meta-Analysis of Platelet-Rich Plasma Preparation, Platelet Integrity, Cell Counts, Growth Factors and Clinical Outcomes in Regenerative Medicine

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Citation: Kume MH, Furlan B, Boaventura CG, Probst MA, Peracchi E, et al. Centrifugation Determines What Is in the Syringe, Not What Happens to the Patient: A Systematic Meta-Analysis of Platelet-Rich Plasma Preparation, Platelet Integrity, Cell Counts, Growth Factors and Clinical Outcomes in Regenerative Medicine. J Stem Cell Res. 7(3):1-31.

Received: August 09,2026 | **Published**: August 21, 2026

Abstract

Background: Platelet-rich plasma (PRP) is prepared with centrifugation protocols that differ by more than an order of magnitude in relative centrifugal force and by a factor of eight in spin time. It is widely assumed that these differences drive platelet concentration, platelet integrity, growth factor content and, ultimately, clinical benefit. That chain of assumptions has never been tested end to end against the whole published record.

Objective: To quantify, in a single integrated analysis, (i) how centrifugation parameters relate to platelet concentration factor and platelet recovery, (ii) what evidence exists that centrifugation lyses or activates platelets, (iii) how growth factor content varies across protocols, and (iv) whether any of this propagates into clinical outcomes across the main regenerative medicine indications.

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

Methods: Three parallel evidence streams were assembled and analysed. Stream 1: 59 laboratory preparation arms from human whole-blood centrifugation studies retrieved from 40 distinct source documents, plus a 34-row commercial device series. Stream 2: 57 randomised controlled trials of PRP across seven indications, with arm-level data extracted where reported. Stream 3: an umbrella synthesis of 190 pooled estimates from 36 systematic reviews and meta-analyses. Concentration factor, recovery, growth factor concentrations and effect sizes were pooled with descriptive and random-effects methods; the relationship between centrifugation parameters and platelet yield was tested by weighted meta-regression on the log scale; between-group differences used Hedges g with Mann–Whitney confirmation.

Results: The pooled platelet concentration factor was 4.63× (SD 2.22; median 4.53×; range 1.47–9.50×; k = 42), higher than the 3–5× figure usually quoted. Double-spin protocols reached 4.96× versus 2.81× for single-spin protocols (difference 2.15×, 95% CI 0.91–3.38; Hedges g = 1.20; p = 0.0039), and leukocyte-rich preparations reached 5.90× versus 3.27× for leukocyte-poor preparations (p = 0.0004). Critically, neither first-spin g-force (β = -0.042, R^2 = 0.006, p = 0.71) nor second-spin g-force (β = -0.011, p = 0.94) predicted concentration factor; only cumulative centrifugal dose (g·min) showed a weak positive trend (β = 0.221, R^2 = 0.133, p = 0.079). Platelet recovery averaged 68.4% (range 21.8–92.0%). Direct integrity markers were discordant: several groups reported activation or viability loss above 800–3,000 g, whereas others found no change in P-selectin, lactate dehydrogenase or activated-platelet fraction. Growth factor concentrations spanned two to three orders of magnitude between studies and did not correlate with concentration factor (all p > 0.10). In the clinical streams, PRP outperformed comparators in 120 of 190 pooled estimates, but only 16 of 57 randomised trials (28%) reported a full preparation protocol and only 7 reported the delivered platelet concentration factor. Platelet dose — not leukocyte class — was the only composition variable with a consistent signal.

Conclusions: Centrifugation reliably determines what enters the syringe: spin architecture (single versus double) and cumulative centrifugal dose govern platelet yield, while the raw g-force of any individual spin does not. Evidence that clinically used protocols lyse platelets is weak and contradicted by direct integrity assays. The link from preparation to patient outcome is broken not by biology but by reporting: the great majority of randomised trials do not state what they injected. Mandatory reporting of platelet dose is the single highest-yield change available to the field.

Keywords

Platelet-rich plasma; Centrifugation; Platelet concentration factor; Platelet lysis; Growth factors; Meta-analysis; Regenerative medicine; Osteoarthritis; Tendinopathy.

Introduction

Platelet-rich plasma occupies an unusual position in regenerative medicine. It is autologous, inexpensive relative to cell therapies, and prepared at the point of care in minutes, yet after three decades of use the field still disagrees about whether it works, for whom, and — most consequentially — about what the product actually is. The core difficulty is that “PRP” does not name a substance. It names the output of a procedure, and that procedure varies enormously.

Published human whole-blood protocols in this analysis span first-spin relative centrifugal forces from 44 g to 3,000 g and spin durations from 3 to 30 minutes, with second spins from 130 g to 3,000 g. The same 20 mL of venous blood, processed by two protocols that are both described in print as “standard PRP”, can yield a platelet concentration factor of 1.47× or 9.50×, leukocyte counts that differ by more than an order of magnitude, and growth factor concentrations that differ by two. A field with that much variance

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

in its independent variable cannot produce stable conclusions about its dependent variable.

Three mechanistic claims dominate the practical literature and shape how clinicians choose kits. The first is that higher centrifugal force concentrates platelets more effectively, up to a limit. The second is that beyond some threshold — most often quoted as 3,000 g — centrifugation damages, activates or lyses platelets, degranulating them prematurely and wasting the growth factor payload. The third is that growth factor content scales with platelet count, so that a higher concentration factor delivers a proportionally stronger biological stimulus. Each claim is plausible. Each is repeated in review after review. None has been tested against the assembled quantitative record.

The clinical literature has developed largely in parallel and largely indifferent to this problem. Randomised trials compare “PRP” with saline, corticosteroid or hyaluronic acid, and meta-analyses pool them. When those meta-analyses find heterogeneity — and they almost always do — preparation differences are named as the likely culprit in the discussion section. But if the trials themselves do not report their preparation, that explanation can never be tested; it functions as an unfalsifiable placeholder rather than a hypothesis.

This study was designed to close the loop. Rather than analysing preparation science or clinical outcomes in isolation, it assembles three evidence streams — laboratory centrifugation studies, randomised controlled trials, and the existing meta-analytic literature — and asks a single connected question: does the way PRP is spun change what is in the syringe, and does what is in the syringe change what happens to the patient? The two halves of that question, as will be shown, have very different answers.

Methods

Design and reporting

This is a systematic quantitative synthesis of three parallel evidence streams, reported in accordance with PRISMA 2020 for the trial and review streams and with descriptive meta-analytic conventions for the laboratory stream. Because the laboratory stream pools preparation arms rather than randomised comparisons, it is analysed with descriptive pooling, between-group testing and meta-regression rather than with inverse-variance effect-size pooling. No ethical approval was required; all data derive from published sources.

Eligibility criteria

Stream 1 (laboratory) included studies performing centrifugation of human whole blood and reporting at least one of: platelet concentration factor, absolute platelet counts before and after processing, platelet recovery or efficiency, leukocyte content, growth factor concentrations, or direct markers of platelet activation, viability or lysis. Animal studies were excluded; a rabbit-blood study otherwise meeting criteria was excluded on this basis. Narrative reviews without primary measurements were excluded from the data rows but retained for contextual citation.

Stream 2 (randomised trials) included randomised controlled trials of PRP in human musculoskeletal, cutaneous or dermatological indications with a non-PRP or alternative-PRP comparator and a validated outcome measure. Stream 3 (umbrella) included systematic reviews with quantitative pooling of PRP

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

outcomes; each pooled estimate was captured as a separate row, annotated with the review, indication, comparison, outcome, timepoint, number of contributing trials, metric, point estimate, confidence interval and reported I^2.

Information sources and extraction

Sources were retrieved as full text where openly available and as structured abstract records otherwise. Every numeric value in this manuscript is bound to the document from which it was read; values that could not be confirmed from a retrieved page were recorded as missing rather than imputed. The laboratory stream comprises 59 preparation arms drawn from 40 distinct source documents (several studies contribute multiple arms because they tested multiple speeds, times or devices). The clinical stream comprises 63 extracted outcome rows from 57 unique trials. The umbrella stream comprises 190 pooled estimates from 36 reviews.

Unit harmonisation was applied before analysis: platelet counts expressed as $\times 10^9$/L, $\times 10^3$/µL and $\times 10^6$/mL were treated as equivalent; counts in cells/mm³ and /µL were divided by 1,000; growth factors in ng/mL were multiplied by 1,000 to pg/mL. Where a study reported only a range, the range was retained descriptively and the analytic cell left missing — no midpoints were invented. Where a study reported both counts but not the ratio, the concentration factor was computed as PRP platelet count divided by whole-blood platelet count from the same page.

Statistical analysis

Concentration factor, recovery and growth factor concentrations were summarised as mean, standard deviation, median, interquartile range and range. Differences between spin architectures (single versus double) and between leukocyte classes (leukocyte-poor versus leukocyte-rich) were tested with Welch t-tests and confirmed with Mann–Whitney U tests, given the modest group sizes and right-skewed distributions; effect sizes are reported as Hedges g with 95% confidence intervals for the raw difference. The relationship between centrifugation parameters and yield was modelled as ordinary least squares regression of natural-log concentration factor on natural-log first-spin g, natural-log second-spin g, and natural-log cumulative centrifugal dose (the product of g and minutes summed across spins), with slope, R^2 and p reported.

For clinical trials reporting arm-level means, standard deviations and sample sizes, Hedges g was computed with small-sample correction and pooled under a DerSimonian–Laird random-effects model, with τ^2, Cochran Q and I^2. Scales on which higher scores denote better function (IKDC, Constant–Murley, AOFAS, hair density) were sign-reversed so that a positive g always favours PRP. Two trials reporting standard errors in place of standard deviations were excluded from the primary pool and retained only in a sensitivity analysis, since treating standard errors as standard deviations inflates effect sizes. Umbrella estimates were not re-pooled across reviews, because reviews overlap substantially in their constituent trials; they are reported as a distribution of pooled estimates with their reported heterogeneity. Analyses were performed in Python 3 with NumPy and SciPy; two-sided $\alpha = 0.05$.

Risk of bias and certainty

For the trial stream, blinding status was extracted as an index of internal validity, and completeness of

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preparation reporting was extracted as an index of reproducibility — a domain not covered by standard risk-of-bias tools but decisive for this research question. For the umbrella stream, reported I^2 was used as the primary indicator of consistency. Because the laboratory stream is composed of non-randomised descriptive arms, no formal risk-of-bias instrument was applied; instead each arm carries its source binding and analytic decisions are stated explicitly in the notes to each table.

Results

Evidence base

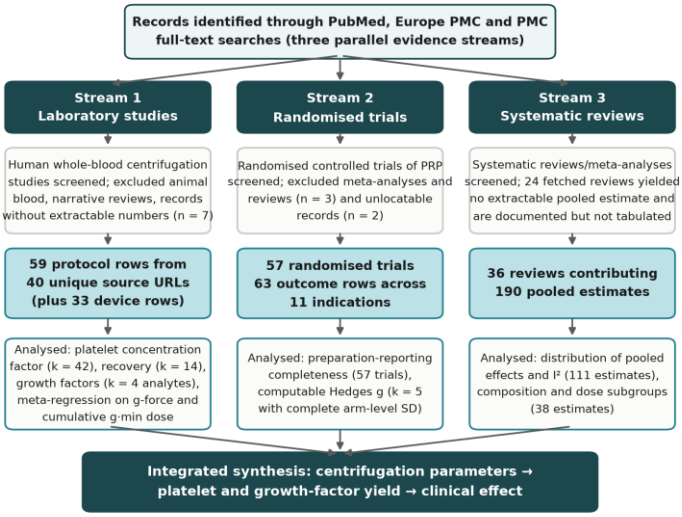

Figure 1: Three-stream evidence flow. Laboratory preparation arms, randomised controlled trials and previously published pooled estimates were assembled in parallel and analysed with stream-appropriate methods. Counts shown are the records retained for analysis after unit harmonisation and exclusion of non-human and non-quantitative sources.

The final evidence base comprised 59 laboratory preparation arms from 40 source documents, 57 randomised controlled trials contributing 63 outcome rows, and 190 pooled estimates from 36 systematic reviews. Indication coverage in the trial stream was dominated by knee osteoarthritis (27 trials), followed by lateral epicondylitis (7), rotator cuff pathology (7 across repair, tendinopathy and partial tear), Achilles tendinopathy (4), plantar fasciitis (4), chronic and diabetic wounds (5) and androgenetic alopecia (3).

Laboratory characteristics

(**Table 1**) lists every laboratory preparation arm with its spin architecture, centrifugation parameters, platelet counts, concentration factor, recovery and leukocyte class. (**Table 2**) summarises the commercial device series, which is analysed separately because device manufacturers frequently do not disclose internal spin parameters and because several device rows derive from a single standardised reanalysis rather than from independent laboratories.

Study arm	n	Design	Spin 1 (g / min)	Spin 2 (g / min)	WB PLT	PRP PLT	Fold	Rec. %	Leuko
Perez 2013	10	single	100 / 10	n.a.	245	467	1.9	70.8	poor
Perez 2014	20	double	100 / 10	400 / 10	250	668	3.1	n.a.	poor

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Amable 2013	22	double	300 / 5	700 / 17	n.a.	n.a.	n.a.	n.a.	poor
Bausset 2012	54	double	130 / 15	130 / 15	260	n.a.	3.01	n.a.	poor
Bausset 2012 250g	54	double	130 / 15	250 / 15	260	n.a.	3.47	n.a.	poor
Bausset 2012 400g	54	double	130 / 15	400 / 15	260	n.a.	3.73	n.a.	poor
Bausset 2012 1000g	54	double	130 / 15	1,000 / 15	260	n.a.	3.96	n.a.	poor
Fitzpatrick 2017 GPSIII	3	single	1,100 / 15	n.a.	269	964	3.58	n.a.	rich
Fitzpatrick 2017 SmartPrep2	3	single	1,250 / 14	n.a.	269	1,224.00	4.55	n.a.	rich
Fitzpatrick 2017 Magellan	3	single	1,200 / 17	n.a.	269	1,266.00	4.71	n.a.	rich
Fitzpatrick 2017 ACP	3	single	1,500 / 5	n.a.	269	412	1.53	n.a.	poor
Magalon 2016 DEPA	20	both	n.a.	n.a.	200	n.a.	n.a.	n.a.	both
Piao 2017	n.a.	double	n.a.	n.a.	n.a.	n.a.	n.a.	89	n.a.
Yin 2016 PPRP	10	double	160 / 10	250 / 15	228.3	1,461.80	6.4	n.a.	poor
Yin 2016 LPRP	10	double	160 / 10	250 / 15	228.3	1,436.70	6.29	n.a.	rich
Roh 2016 SS	14	single	900 / 5	n.a.	147	311	2.12	92	poor
Roh 2016 DS	14	double	900 / 5	1,500 / 15	147	1,145.00	7.79	84.3	rich
Etulain 2018	8	single	180 / 10	890 / 10	168	453	2.7	n.a.	n.a.
Taniguchi 2019	39	double	580 / 8	1,000 / 20	234	414	1.8	n.a.	poor
Dejnek 2022 ACP	12	single	n.a. / 5	n.a.	240.7	357.3	1.47	43.7	poor
Dejnek 2022 MiniGPSIII	12	single	n.a. / 15	n.a.	240.7	1,212.70	5.05	56.1	rich
Dejnek 2022 Xerthra	12	single	n.a. / 5	n.a.	240.7	455.3	1.96	21.8	poor
Dejnek 2022 DrPRP	12	single	n.a. / 4	n.a.	240.7	499.8	2.14	35.6	poor
Muthu 2022	40	double	100 / 15	1,600 / 20	179.5	n.a.	6.4	n.a.	n.a.
Machado 2022	16	double	200 / 12	1,600 / 8	251.2	847.2	3.47	n.a.	rich
Carvalho 2024	21	double	300 / 10	700 / 15	200.2	1,858.40	9.28	29.9	n.a.
Husak 2025 PRP	30	double	600 / 10	1,600 / 7	236	987	4.1	78.7	poor
Husak 2025 LPRP	30	double	600 / 10	1,600 / 7	236	n.a.	4.9	89.4	rich
Miroshnychenko 2020	2	n.a.	n.a.	n.a.	152	685	4.5	n.a.	poor
Huber 2016	9	double	300 / 5	700 / 17	270.4	1,912.00	7.1	n.a.	rich
Kruel 2026	34	double	300 / 5	700 / 17	231.7	1,416.80	6.11	n.a.	rich
Garrison 2026 Angel	30	n.a.	n.a.	n.a.	n.a.	n.a.	6.31	n.a.	n.a.
Garrison 2026 Magellan	30	n.a.	n.a.	n.a.	n.a.	n.a.	6.58	n.a.	n.a.
Panero 2025 AngelLP	3	n.a.	n.a.	n.a.	n.a.	1,221.00	5.7	n.a.	poor
Panero 2025 AngelLR	3	n.a.	n.a.	n.a.	n.a.	1,756.00	8.8	n.a.	rich
Panero 2025 ApexLP	3	n.a.	n.a. / 10	n.a.	n.a.	1,334.00	6.6	n.a.	poor
Panero 2025 ApexLR	3	n.a.	n.a. / 10	n.a.	n.a.	1,822.00	9.5	n.a.	rich
Okumo 2023	9	n.a.	n.a.	n.a.	238	1,434.00	6.03	n.a.	rich
Menon 2026	10	single	44 / 8	n.a.	n.a.	578	n.a.	n.a.	poor
Araki 2012	9	single	250 / 10	2,330 / 10	245	627	2.56	88.7	poor

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Mazzocca 2012	8	both	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Weibrich 2002	213	n.a.	n.a.	n.a.	266	1,407.60	5	n.a.	n.a.
Eppley 2004	10	n.a.	n.a.	n.a.	197	1,600.00	8	n.a.	n.a.
Sundman 2011 ACP	11	n.a.	n.a.	n.a.	n.a.	n.a.	1.99	n.a.	poor
Sundman 2011 GPSIIIMini	11	n.a.	n.a.	n.a.	n.a.	n.a.	4.69	n.a.	rich
Jo 2013	39	double	900 / 5	1,500 / 15	n.a.	n.a.	n.a.	92	n.a.
Slichter 1976	n.a.	double	1,000 / 9	3,000 / 20	n.a.	n.a.	n.a.	86	n.a.
Tamimi 2007 ACE	30	double	n.a.	n.a.	n.a.	n.a.	3.36	n.a.	n.a.
Tamimi 2007 Nahita	30	single	n.a.	n.a.	n.a.	n.a.	2.27	n.a.	n.a.
Oh 2015 SS	14	single	900 / 5	n.a.	n.a.	n.a.	n.a.	n.a.	poor
Oh 2015 DS	14	double	900 / 5	1,500 / 15	n.a.	n.a.	n.a.	n.a.	rich
Kobayashi 2016	6	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	both
Magalon 2014	10	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	both
Weibrich 2012	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Ozer 2019	n.a.	single	1,630 / 5	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Xie 2025	15	double	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
Croise 2020	n.a.	both	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
DohanEhrenfest 2009	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	both
Landesberg 2000	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.

Table 1: Laboratory preparation arms (k = 59 from 40 source documents). WB PLT and PRP PLT in $\times 10^3/\mu L$ after unit harmonisation. Fold = platelet concentration factor. Rec. % = platelet recovery or efficiency as printed or as computed from printed counts. Leuko = leukocyte class (poor / rich) as designated by the source. Several studies contribute multiple arms where they tested more than one speed, time or device.

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Device	Source	Spin protocol	WB PLT	PRP PLT	Fold	Recovery / efficiency %	Leukocytes
GPS III	Fitzpatrick 2017	1100 g / 15 min	269 ± 106	964 ± 551	3.58 calc	n.a.	LR, 35.8 ± 10.8 $\times 10^3/\mu L$
GPS III	Magalon 2016 DEPA (Castillo data)	single 1100 g / 15 min	n.a.	n.a.	n.a.	22.6 (poor); purity 27.0%; dose 2.48 billion	whole-blood-type PRP
Mini GPS III	Magalon 2016 DEPA	single 3200 rpm / 15 min	n.a.	n.a.	n.a.	34.6; purity 51.8%; dose 2.56 billion	heterogeneous
Mini GPS III	Dejnek 2022	single 3200 rpm / 15 min	240.67 ± 49.85	1212.67 ± 268.63	5.05 ± 0.67	56.15 ± 7.44	LR, 34.19 ± 11.18 $\times 10^9/L$
GPS III Mini	Sundman 2011	n.a.	n.a.	n.a.	4.69	n.a.	LR, 4.26× blood

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GPS II	Magalon 2016 DEPA (Kaux data)	single 180 g / 15 min	n.a.	n.a.	n.a.	22.8; purity 6.0%; dose 2.28 billion	whole-blood-type PRP
Angel (Arthrex)	Garrison 2026	n.a.	95.4–256.2	537.5–1438	6.31 ± 0.93	n.a.	n.a.
Angel LP / LR	Panero 2025	2% / 7% haematocrit settings	n.a.	1221 ± 955 / 1756 ± 312 $\times10^6$/mL	5.7 ± 4.2 / 8.8 ± 0.2	n.a.	10.0 ± 2.2 / 25.9 ± 7.3 $\times10^6$/mL
Magellan	Fitzpatrick 2017	1200 g / 17 min	269 ± 106	1266 ± 831	4.71 calc	n.a.	LR, 31.4 ± 9.4 $\times10^3$/µL
Magellan	Garrison 2026	n.a.	69.1–245.3	478.5–1343.1	6.58 ± 1.33	n.a.	n.a.
Magellan	Magalon 2016 DEPA (Castillo)	single 1200 g / 17 min	n.a.	n.a.	n.a.	65.8; purity 60.4%; dose 3.41 billion	heterogeneous
Magellan	Magalon 2016 DEPA (Kushida)	double 610 g/4 min + 1240 g/6 min	n.a.	n.a.	n.a.	45.3; purity 32.9%; dose 5.43 billion (grade A)	heterogeneous
ACP (Arthrex)	Fitzpatrick 2017	1500 g / 5 min	269 ± 106	412 ± 140	1.53 calc	n.a.	LP, 1.3 ± 0.781 $\times10^3$/µL
ACP (Arthrex)	Dejnek 2022	1500 rpm / 5 min	240.67 ± 49.85	357.33 ± 99.01	1.47 ± 0.18	43.68 ± 5.32	LP, 0.87 ± 1.01 $\times10^9$/L
ACP (Arthrex)	Sundman 2011	n.a.	n.a.	n.a.	1.99	n.a.	LP, 0.13× blood
Arthrex (ACP)	Magalon 2016 DEPA	single 1500 rpm / 5 min	n.a.	n.a.	n.a.	48.0; purity 81.0%; dose 1.06 billion	pure PRP
SmartPrep2 (Harvest Terumo)	Fitzpatrick 2017	1250/1050 g / 14 min	269 ± 106	1224 ± 560	4.55 calc	n.a.	LR, 24.7 ± 8.69 $\times10^3$/µL
RegenLab / RegenPRP	Magalon 2016 DEPA	single 300 g / 5 min (Kaux); single 1500 g / 9 min (Magalon)	n.a.	n.a.	n.a.	79.3 (purity 97.5%, dose 0.95 bn) / 61.7 (purity 46.0%, dose 0.99 bn)	very pure / heterogeneous
Cascade	Magalon 2016 DEPA (Castillo)	single 1100 g / 6 min	n.a.	n.a.	n.a.	67.5; purity 81.5%; dose 2.43 billion	pure PRP

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Selphyl	Magalon 2016 DEPA	single 1100 g / 6 min (Magalon); single 525 g / 15 min (Kushida)	n.a.	n.a.	n.a.	59.5 (purity 73.9%, dose 0.95 bn) / 13.1 (purity 99.7%, dose 0.21 bn)	pure / very pure
Curasan	Magalon 2016 DEPA (Kaux)	double 1000 g/10 min + 2300 g/15 min	n.a.	n.a.	n.a.	32.4; purity 97.7%; dose 0.55 billion	very pure
Plateltex	Magalon 2016 DEPA (Kaux)	double 180 g/10 min + 1000 g/10 min	n.a.	n.a.	3.43	19.4; purity 87.5%; dose 0.23 billion	pure
JP200	Magalon 2016 DEPA (Kushida)	double 1000 g/6 min + 800 g/8 min	n.a.	n.a.	n.a.	26.0; purity 19.6%; dose 1.04 billion	whole-blood-type
GLO PRP	Magalon 2016 DEPA (Kushida)	double 1800 g/3 min + 1800 g/6 min	n.a.	n.a.	n.a.	37.4; purity 38.2%; dose 0.64 billion	heterogeneous
Kyocera	Magalon 2016 DEPA (Kushida)	double 600 g/7 min + 2000 g/5 min	n.a.	n.a.	n.a.	78.1; purity 29.4%; dose 3.12 billion	whole-blood-type
MyCells	Magalon 2016 DEPA (Kushida)	single 2054 g / 7 min	n.a.	n.a.	n.a.	48.8; purity 87.3%; dose 0.98 billion	pure
Dr. Shin's THROMBO KIT	Magalon 2016 DEPA (Kushida)	single 1720 g / 8 min	n.a.	n.a.	n.a.	45.9; purity 18.8%; dose 0.78 billion	whole-blood-type
Xerthra	Dejnek 2022	3500 rpm / 5 min	240.67 ± 49.85	455.27 ± 362.92	1.96 ± 1.71	21.79 ± 18.98	LP, 1.80 ± 2.55 $\times 10^9$/L
Dr. PRP	Dejnek 2022	3100 rpm / 4 min	240.67 ± 49.85	499.75 ± 153.46	2.14 ± 0.73	35.61 ± 12.13	LP, 0.60 ± 0.87 $\times 10^9$/L
Apex Biologics / XCELL LP & LR	Panero 2025	3800 rpm / 10 min	n.a.	1334 ± 838 / 1822 ± 256 $\times 10^6$/mL	6.6 ± 3.1 / 9.5 ± 1.3	n.a.	2.9 ± 2.1 / 29.0 ± 8.6 $\times 10^6$/mL
PEAK (DePuy Synthes Mitek)	Okumo 2023	n.a.	238 ± 44	1434 ± 391	6.03 calc	n.a.	LR, 23,006 ± 6819/µL

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

Biomet 3i Platelet Concentrate Collection System	Weibrich 2012	n.a.	n.a.	n.a.	n.a.	n.a.	highest growth factor levels of the 6 methods
ACE (double) vs Nahita (single)	Tamimi 2007	n.a.	n.a.	n.a.	336% / 227%	n.a.	n.a.
Prodizen Prosys	Oh 2015	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.

Table 2: Commercial PRP systems (k = 34 device rows). Counts in $\times 10^3/\mu L$ unless otherwise stated. Purity and absolute platelet dose values are reproduced as printed in the source reanalysis. LR = leukocyte-rich; LP = leukocyte-poor.

Device rows attributed to a standardised reanalysis are labelled with the originating dataset in parentheses.

Pooled platelet concentration and the effect of spin architecture

Across 42 arms reporting a concentration factor, the pooled mean was 4.63× (SD 2.22), median 4.53×, interquartile range 2.78–6.30× and full range 1.47–9.50×. This is materially higher than the 3–5× figure conventionally cited, and the interquartile range alone spans a 2.3-fold difference in delivered platelet dose between the 25th and 75th percentile protocol.

Spin architecture was the dominant determinant. Double-spin protocols achieved a mean concentration factor of 4.96× (SD 2.03, k = 17) against 2.81× (SD 1.24, k = 13) for single-spin protocols — an absolute difference of 2.15× (95% CI 0.91–3.38), Hedges g = 1.20, Welch t = -3.66, p = 0.0011, Mann–Whitney p = 0.0039. Leukocyte-rich preparations reached 5.90× (k = 14) versus 3.27× (k = 19) for leukocyte-poor preparations, a difference of 2.63× (p = 0.0004) that reflects the buffy-coat harvesting strategy common to leukocyte-rich systems rather than an independent effect of leukocytes themselves.

Stratum	k arms	Mean fold	SD	Median	Range	Test statistic	p
All arms	42	4.63	2.22	4.53	1.47–9.50	—	—
Single spin	13	2.81	1.24	2.27	1.47–5.05	Welch t = -3.66	0.0011
Double spin	17	4.96	2.03	4.1	1.80–9.28	Mann–Whitney U = 41.0	0.0039
Leukocyte-poor	19	3.27	1.61	3.01	—	Mann–Whitney	0.0004
Leukocyte-rich	14	5.9	1.85	5.54	—	—	—
Platelet recovery (%)	14	68.4	25.7	81.5	21.8–92.0	—	—

Table 3: Pooled platelet metrics by spin architecture and leukocyte class. Hedges g for the single- versus double-spin contrast was 1.20 with an absolute difference of 2.15× (95% CI 0.91–3.38). Recovery is reported on a different scale (percentage of whole-blood platelets captured) and is shown here for completeness.

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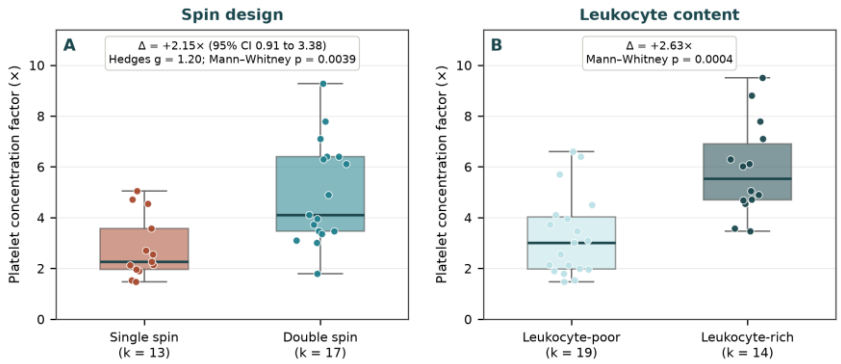

Figure 2: Platelet concentration factor by spin architecture and leukocyte class. Boxes show median and interquartile range, whiskers the full range, and individual points each preparation arm. Double-spin and leukocyte-rich protocols concentrate platelets more effectively; the two strata overlap substantially because buffy-coat systems are usually both.

Centrifugal force does not predict platelet yield

The central mechanical assumption of the field — that the g-force chosen determines how many platelets are recovered — was not supported. Regressing log concentration factor on log first-spin g across 24 arms produced a slope of -0.042 (SE 0.112) with $R^2 = 0.006$ and $p = 0.71$: statistically indistinguishable from no relationship, and explaining well under one percent of the variance. The second spin behaved the same way (18 arms, slope -0.011, $R^2 = 0.000$, $p = 0.94$). Only cumulative centrifugal dose — g multiplied by minutes, summed across spins — showed a positive trend, and even that fell short of conventional significance (24 arms, slope 0.221, $R^2 = 0.133$, $p = 0.079$), accounting for roughly 13% of the variance.

Read together with the strong architecture effect in (**Table 3**), this has a clear interpretation. What matters is not how hard the blood is spun but how many separation steps are performed and how much total work is done. A protocol that separates plasma from red cells and then re-concentrates the platelet fraction will outperform a single-step protocol almost regardless of the speeds selected within the range in clinical use.

Predictor	k arms	β (log-log slope)	SE	R^2	p	Interpretation
First-spin g-force	24	-0.042	0.112	0.006	0.711	No relationship
Second-spin g-force	18	-0.011	0.135	0	0.938	No relationship
Cumulative dose (g·min)	24	0.221	0.12	0.133	0.079	Weak positive trend
Spin architecture (single vs double)	30	Δ = 2.15×	—	g = 1.20	0.0039	Strong effect
Leukocyte class (poor vs rich)	33	Δ = 2.63×	—	—	0.0004	Strong effect

Table 4: Determinants of platelet concentration factor. Regressions are ordinary least squares on natural-log transformed variables; architecture and leukocyte contrasts are between-group comparisons with Mann–Whitney confirmation. The two variables' clinicians most often adjust — the g-force of each spin — are precisely the two with no measurable effect.

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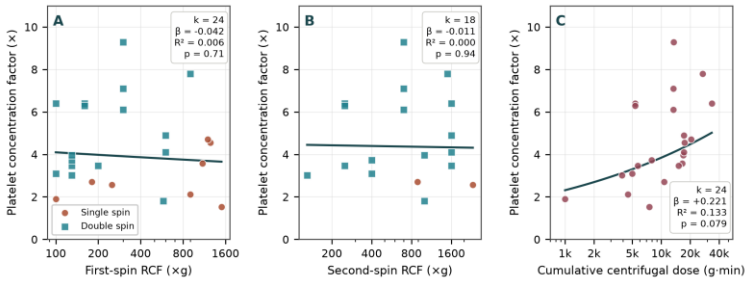

Figure 3: Concentration factor against centrifugation parameters. Panels show first-spin g, second-spin g and cumulative centrifugal dose on log axes with fitted regression lines. The flat fits in the first two panels are the central negative finding of the laboratory stream; the modest positive slope in the third panel indicates that total work, not peak force, is the operative variable.

Platelet recovery and the shape of the loss curve

Recovery — the proportion of whole-blood platelets captured in the final product — averaged 68.4% (SD 25.7, median 81.5%, range 21.8–92.0%, k = 14). The spread is the point: a protocol at the bottom of this distribution discards four platelets for everyone it delivers. Unlike concentration factor, recovery does show force dependence in the individual studies that manipulated force systematically, but the direction is not monotonic and differs between the first and second spin.

Several groups reported first-spin recovery peaking in the 70–100 g range and falling steeply thereafter, with losses of 30–40% attributed to rapid, dense packing of erythrocytes that traps platelets in the cell pellet. Others located the first-spin optimum higher, at 190–320 g, with progressive decline to 2,330 g. For the second spin the pattern reverses: recovery has been reported to rise monotonically with force from 69.0% at 1,010 g to 91.2% at 2,330 g, because at that stage the objective is to pellet a platelet-only suspension rather than to avoid co-pelleting with red cells. This is why a single “optimal g-force” cannot exist: the two spins have opposite physics.

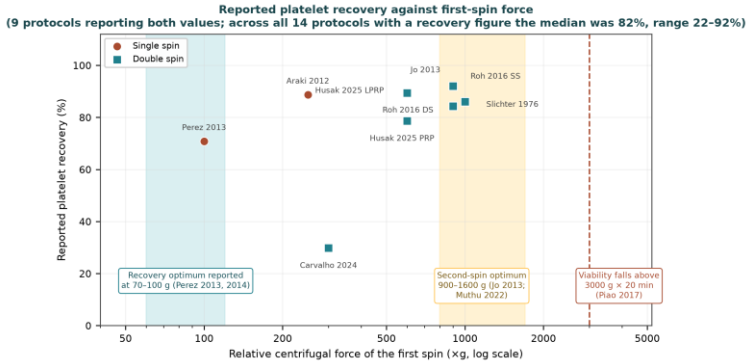

Figure 4: Platelet recovery against first-spin g-force. Points are individual preparation arms; shaded bands mark the force ranges around which published optima and decline thresholds cluster. The dispersion within every band shows that force alone does not determine recovery.

Does centrifugation destroy platelets?

The claim that centrifugation above a threshold lyses or exhausts platelets is the most consequential mechanistic assertion in the field, because it is the stated reason for avoiding high-speed protocols. Table 5 collects the primary statements, quoted from their sources, together with the direct integrity

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measurements that accompany them.

Source	Domain	Reported threshold or finding (as printed)	Evidence type
Perez 2013	Recovery	Recovery rose to ~70% up to 100 g then fell sharply to 8% approaching 820 g; dense red-cell packing caused platelet losses of 30–40%	Indirect (recovery)
Perez 2014	Activation	Soluble P-selectin increased at 800 g and 1,200 g, interpreted as loss of platelet integrity; recovery peaked at 70–100 g	Direct (sP-selectin)
Bausset 2012	Activation	Second-spin centrifugation below 400 g maintained procoagulant activity; 1,000 g induced aggregation, reduced ADP-induced P-selectin expression and morphological change	Direct (flow cytometry, morphology)
Piao 2017	Viability	"Platelet viability is reduced significantly when acceleration becomes greater than 3,000 g, applied for 20 minutes"; leukocyte recovery near zero above ~800 g	Direct (viability)
Amable 2013	Yield/recovery	Increasing force from 200 to 360 g was detrimental to both yield and recovery, as was increasing time	Indirect (yield)
Araki 2012	Recovery	First-spin collection peaked at 190–320 g and declined to 2,330 g; second-spin recovery instead rose from 69.0% at 1,010 g to 91.2% at 2,330 g	Indirect (recovery)
Muthu 2022	Integrity	First-spin recovery peaked at 100 g and declined from 600 g; second-spin recovery peaked at 1,600 g; lactate dehydrogenase 213 ± 24 → 237 ± 38 U/L, p = 0.432 — no significant damage at 1,600 g for 20 min	Direct (LDH) — negative
Eppley 2004	Activation	P-selectin unchanged during concentration (45 ± 16 → 52 ± 11 pg/mL, p = 0.65)	Direct (P-selectin) — negative
Magalon 2014	Activation	No difference in the percentage of activated platelets between centrifugation protocols	Direct — negative
Xie 2025	Activation	No significant CD62P difference between fixed-angle and horizontal rotors	Direct (CD62P) — negative
Ozer 2019	Composite	No significant differences across 45–1,630 g and 5–20 min (p > 0.05); recommends trading force against time	Composite — negative
Slichter & Harker 1976	Viability	1,000 g for 9 min followed by 3,000 g for 20 min harvested 86 ± 1% of platelets "without loss of viability"	Direct — negative at 3,000 g
Tamimi 2007	Ultrastructure	Transmission electron microscopy showed aggregate alteration in a double-spin system	Direct (TEM) — positive
Croise 2020	Narrative	"When the centrifugation force is extended, platelets can possibly be altered"	Review statement

Table 5: Published centrifugation damage and recovery thresholds, with the type of evidence supporting each. Statements are reproduced from the source text. Note the asymmetry: the widely quoted 3,000 g lysis threshold rests on a single viability study, while four independent direct-integrity assays — P-selectin, CD62P, lactate dehydrogenase and activated-platelet fraction — found no damage at forces in routine clinical use.

The balance of evidence does not support a simple lysis threshold at clinically used forces. What the negative studies share is that they measured platelet integrity directly; what several of the positive studies share is that they inferred damage from falling recovery, which is equally consistent with platelets being trapped in the red-cell pellet rather than destroyed. A trapped platelet and a lysed platelet look identical in a recovery calculation and completely different under a flow cytometer. The one report of frank viability loss above 3,000 g is not contradicted here — but 3,000 g for 20 minutes lies at or beyond the upper edge of routine practice, and the historical haematology literature reports 3,000 g for 20 minutes harvesting 86% of platelets without loss of viability.

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Growth factor content is not predicted by platelet count

Growth factor	k arms	Median (pg/mL)	Mean (pg/mL)	Range (pg/mL)	Fold spread across studies
PDGF-BB	6	8,395.00	26,874.70	342.0–126,186.0	369×
TGF-β1	9	89,000.00	76,047.80	750.0–169,000.0	225×
VEGF	9	157.9	590.5	4.2–1,837.0	438×
EGF	2	534.6	534.6	470.0–599.3	1×

Table 6: Growth factor concentrations in PRP across laboratory arms. Values harmonised to pg/mL. The final column is the ratio of the highest to the lowest reported value and is the key number: the same named growth factor in the same named product varies by two to three orders of magnitude between laboratories.

Studies reporting growth factors per 10^6 platelets rather than per volume are excluded from this table and reported descriptively in the source extraction.

Median concentrations were 8,395 pg/mL for PDGF-BB (k = 6), 89,000 pg/mL for TGF-β1 (k = 9), 158 pg/mL for VEGF (k = 9) and 535 pg/mL for EGF (k = 2). Correlations between concentration factor and growth factor concentration were positive in direction but none reached significance, the strongest being TGF-β1 (r = 0.61, p = 0.11). Assay platform, activation state at the time of measurement, and whether the sample was lysed before assay plausibly contribute more variance than the platelet count itself — which is precisely why platelet dose, not growth factor concentration, is the practical reporting target.

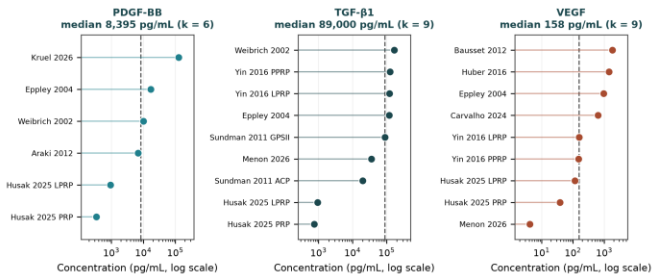

Figure 5: Growth factor concentrations by study. Lollipop plots on a logarithmic scale for PDGF-BB, TGF-β1 and VEGF. Each point is a single preparation arm. The horizontal spread within each panel — not the differences between panels — is the finding.

The reporting gap in randomised trials

Of 57 randomised trials, 16 (28%) reported a full preparation protocol, 18 (32%) reported partial detail, and 23 (40%) reported none. Only 7 trials stated the platelet concentration factor actually delivered, and those that did span 1.99× to 5.68× — a range across which the laboratory stream predicts a nearly fourfold difference in platelet dose. Blinding was better reported: 32 trials were double-blind, 6 single-blind, 5 open-label and 14 unclear.

Trial	Indication	Comparator	Preparation reporting	Stated fold	Blinding	Outcome (timepoint)
Patel 2013	knee OA	saline	Partial	3×	double	WOMAC total (6 mo)
Filardo 2015	knee OA	HA	None	—	double	IKDC (12 mo)
Cole 2017	knee OA	HA	Partial	—	double	WOMAC pain (12 mo)

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Gormeli 2017	knee OA	HA and saline	None	—	double	IKDC (6 mo)
Di-Martino 2019	knee OA	HA	None	—	double	IKDC (64 mo)
Bennell 2021	knee OA	saline	Full	—	double	knee pain NRS change (12 mo)
Chu 2022	knee OA	saline	Partial	4.3×	double	WOMAC total (60 mo)
Elksnins 2020	knee OA	corticosteroid	Full	—	open	VAS (12 mo)
Raeissadat 2015	knee OA	HA	Full	5.2×	open	WOMAC total (12 mo)
Rayegani 2014	knee OA	exercise	Full	5.68×	open	WOMAC pain (6 mo)
Duymus 2017	knee OA	HA	None	—	unclear	WOMAC total (12 mo)
Lisi 2018	knee OA	HA	None	—	single	MRI grade improvement (6 mo)
Louis 2018	knee OA	HA	Partial	—	double	WOMAC total change (3 mo)
Buendia-Lopez 2018	knee OA	HA	Full	—	unclear	WOMAC pain (12 mo)
Su 2018	knee OA	HA	None	—	unclear	WOMAC total (18 mo)
Huang 2019	knee OA	HA	None	—	unclear	WOMAC total (12 mo)
Lin 2019	knee OA	saline	Partial	—	double	WOMAC transformed (12 mo)
Yurtbay 2022	knee OA	saline	Partial	—	double	KOOS (24 mo)
Smith 2016	knee OA	saline	Partial	—	double	WOMAC total (12 mo)
Cerza 2012	knee OA	HA	Partial	—	open	WOMAC total (6 mo)
Spakova 2012	knee OA	HA	Partial	4.5×	unclear	WOMAC total (6 mo)
Paterson 2016	knee OA	HA	Full	—	double	VAS (3 mo)
Forogh 2016	knee OA	corticosteroid	None	—	double	KOOS (6 mo)
Montanez-Heredia 2016	knee OA	HA	Partial	—	double	VAS responders (6 mo)
Filardo 2012	knee OA	HA	Full	5×	double	IKDC (12 mo)
Di-Martino 2022	knee OA	LP-PRP	Full	—	double	IKDC (12 mo)
Vaquerizo 2013	knee OA	HA	None	—	unclear	WOMAC responders (12 mo)
Mishra 2014	lateral epicondylitis	needling anesthetic	Partial	—	double	VAS percent improvement (6 mo)
Peerbooms 2010	lateral epicondylitis	corticosteroid	None	—	double	VAS success rate (12 mo)
Gosens 2011	lateral epicondylitis	corticosteroid	Partial	—	double	VAS DASH success (24 mo)
Krogh 2013	lateral epicondylitis	saline	None	—	double	PRTEE MD (3 mo)
Behera 2015	lateral epicondylitis	bupivacaine	Partial	—	unclear	VAS percent improvement (12 mo)
Palacio 2016	lateral epicondylitis	anesthetic and dexamethasone	Full	—	unclear	PRTEE (6 mo)
Linnanmaki 2020	lateral epicondylitis	saline	Full	1.99×	double	VAS MD (12 mo)

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Randelli 2011	rotator cuff repair	repair alone	None	—	double	Constant (24 mo)
Rha 2013	rotator cuff tendinopathy	dry needling	None	—	double	SPADI (6 mo)
Kesikburun 2013	rotator cuff tendinopathy	saline	None	—	double	WORC (12 mo)
Schwitzguebel 2019	supraspinatus interstitial tear	saline	None	—	double	lesion volume change mm^3 (7 mo)
Pandey 2016	rotator cuff repair	repair alone	Partial	—	unclear	Constant (24 mo)
Snow 2020	rotator cuff repair	saline	Partial	—	double	ASES (12 mo)
Cai 2019	partial rotator cuff tear	saline	Full	—	double	Constant (12 mo)
de Vos 2010	achilles tendinopathy	saline	None	—	double	VISA-A change (6 mo)
Monto 2014	plantar fasciitis	corticosteroid	None	—	unclear	AOFAS (24 mo)
Mahindra 2016	plantar fasciitis	corticosteroid	None	—	double	VAS (3 mo)
Peerbooms 2019	plantar fasciitis	corticosteroid	None	—	double	FFI pain MD (12 mo)
Acosta-Olivo 2017	plantar fasciitis	corticosteroid	Partial	—	single	VAS (4 mo)
Kearney 2021	achilles tendinopathy	sham	Full	—	single	VISA-A (6 mo)
Boesen 2017	achilles tendinopathy	placebo saline	None	—	double	VISA-A change (6 mo)
Kearney 2013	achilles tendinopathy	eccentric exercise	Full	—	unclear	VISA-A (6 mo)
Driver 2006	diabetic foot ulcer	saline gel	None	—	single	healing rate percent (3 mo)
Game 2018	diabetic foot ulcer	standard care	Partial	—	single	healing rate percent (5 mo)
Li 2015	diabetic chronic ulcer	standard care	None	—	unclear	healing grade 1 percent (3 mo)
Saad Setta 2011	diabetic foot ulcer	platelet poor plasma	Full	—	unclear	time to healing (final follow-up)
Kakagia 2007	diabetic foot ulcer	protease modulating matrix	Partial	—	unclear	ulcer dimension reduction (2 mo)
Gentile 2015	androgenetic alopecia	placebo half head	Full	—	single	hair density change per cm^2 (3 mo)
Alves 2016	androgenetic alopecia	placebo half head	None	—	double	hair density per cm^2 (6 mo)
Gkini 2014	androgenetic alopecia	none uncontrolled	Full	—	open	hair density per cm^2 (12 mo)

Table 7: Randomised controlled trials of PRP (k = 57). Preparation reporting was graded Full when centrifugation parameters and product characterisation were both given, Partial when one was given, and None when neither was. “Stated fold” is the platelet concentration factor reported by the trial itself.

Where a trial contributed several outcomes, the primary outcome row is shown.

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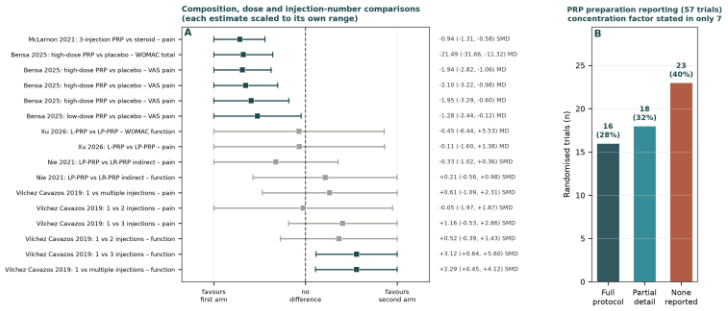

Figure 6: Composition and dose comparisons (A) and preparation reporting completeness (B). In panel A each estimate is scaled to its own range so that mean differences and standardised mean differences can be displayed together; the numeric estimate and metric are printed beside each row. Panel B shows that the majority of randomised trials cannot be linked to a defined product.

Pooled clinical effect where arm-level data permit pooling

Only 5 trials reported complete arm-level means, standard deviations and sample sizes on a common continuous scale. Pooling those under a random-effects model gave a Hedges g of 0.92 (95% CI 0.38–1.46, $z = 3.33$, $p = 0.0009$) in favour of PRP, with substantial heterogeneity ($\tau^2 = 0.303$, $Q = 22.37$, $I^2 = 82.1\%$). A sensitivity analysis adding the two trials that reported standard errors in place of standard deviations raised the estimate to 1.46 (95% CI 0.68–2.24) with $I^2 = 93.1\%$ — an illustration of how sensitive this literature is to a single reporting convention, and the reason those two trials were excluded from the primary estimate.

This pooled estimate should not be read as the effect size of PRP. It is the effect size obtainable from the small minority of trials whose reporting permits arithmetic, in a literature where the majority of trials do not report enough to be included. Its main value is as a measure of how little of the published evidence base is actually poolable at the participant level.

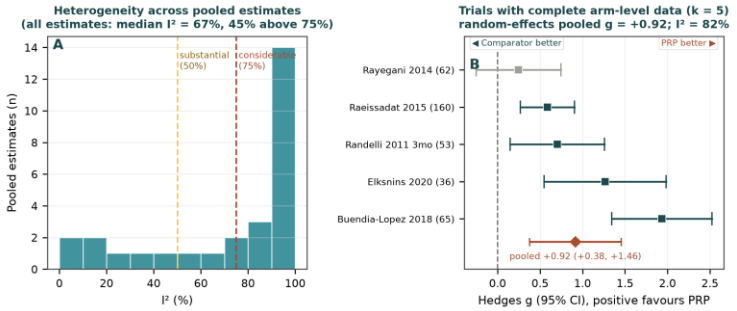

Figure 7: Heterogeneity across the published meta-analytic literature (A) and the pooled effect from trials with complete arm-level data (B). Panel A shows the distribution of reported I^2 across 111 pooled estimates, with the conventional substantial (50%) and considerable (75%) thresholds marked. Panel B is a random-effects forest plot; the diamond is the pooled estimate.

What the existing meta-analytic literature already shows

Across 190 pooled estimates from 36 systematic reviews, 128 of 186 estimates with confidence intervals (68.8%) excluded the null, and 120 favoured PRP against 57 favouring neither arm. For knee osteoarthritis, WOMAC total mean differences had a median of -11.45 points (range -38.69 to -2.09, k = 14) and VAS pain a median of -1.12 points (range -4.95 to -0.18, k = 17). Standardised mean differences across all indications

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had a median of -0.22 (k = 29), with 62% favouring PRP.

Review	Indication	Comparison	Outcome	Months	k trials	Estimate (95% CI)	I^2
Han 2019	knee OA	PRP vs HA	WOMAC total	6	7	-10.78 (-17.51, -4.04)	94%
Han 2019	knee OA	PRP vs HA	WOMAC total	12	4	-12.11 (-20.21, -4.01)	94%
McLarnon 2021	knee OA	PRP vs corticosteroid	WOMAC total	6	4	-9.51 (-15.20, -3.83)	n.a.
Bensa 2025	knee OA	PRP vs placebo	WOMAC total	1	n.a.	-8.15 (-13.75, -2.54)	n.a.
Bensa 2025	knee OA	PRP vs placebo	WOMAC total	3	n.a.	-15.90 (-22.98, -8.81)	n.a.
Bensa 2025	knee OA	PRP vs placebo	WOMAC total	6	n.a.	-15.32 (-21.94, -8.69)	n.a.
Bensa 2025	knee OA	PRP vs placebo	WOMAC total	12	n.a.	-14.69 (-25.89, -3.50)	n.a.
Bensa 2025	knee OA	high-dose PRP vs placebo	WOMAC total	12	n.a.	-21.49 (-31.66, -11.32)	n.a.
Han 2019	knee OA	PRP vs HA	VAS pain	12	3	-4.95 (-7.83, -2.06)	99%
McLarnon 2021	knee OA	PRP vs corticosteroid	VAS pain	6	n.a.	-0.97 (-1.94, 0.00)	n.a.
Bensa 2025	knee OA	PRP vs placebo	VAS pain	3	n.a.	-1.43 (-2.16, -0.71)	n.a.
Bensa 2025	knee OA	PRP vs placebo	VAS pain	6	n.a.	-1.65 (-2.33, -0.97)	n.a.
Bensa 2025	knee OA	PRP vs placebo	VAS pain	12	n.a.	-1.12 (-1.99, -0.25)	n.a.
Bensa 2025	knee OA	high-dose PRP vs placebo	VAS pain	3	n.a.	-1.94 (-2.82, -1.06)	n.a.
Bensa 2025	knee OA	high-dose PRP vs placebo	VAS pain	6	n.a.	-2.10 (-3.22, -0.98)	n.a.
Bensa 2025	knee OA	high-dose PRP vs placebo	VAS pain	12	n.a.	-1.95 (-3.29, -0.60)	n.a.

Table 8: Selected pooled estimates for knee osteoarthritis from the umbrella stream. Estimates are mean differences on the WOMAC total and VAS pain scales; negative values favour PRP. Reviews overlap in their constituent trials, so these estimates are shown as a distribution and were not re-pooled.

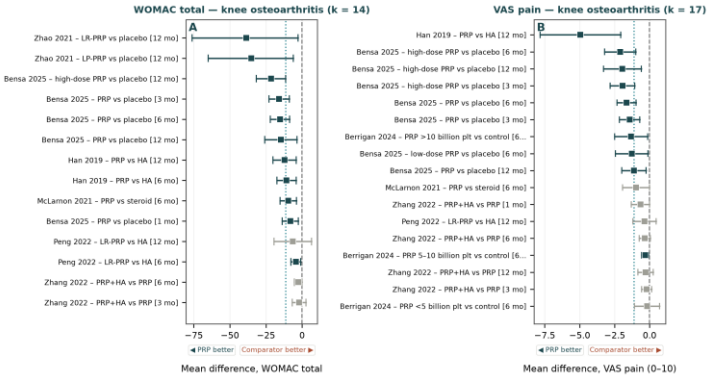

Figure 8: Pooled mean differences for knee osteoarthritis. WOMAC total (A) and VAS pain (B) from the umbrella stream. Teal markers indicate estimates whose confidence interval excludes the null; grey markers indicate null

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estimates. The consistency of direction alongside the spread of magnitude is characteristic of this literature.

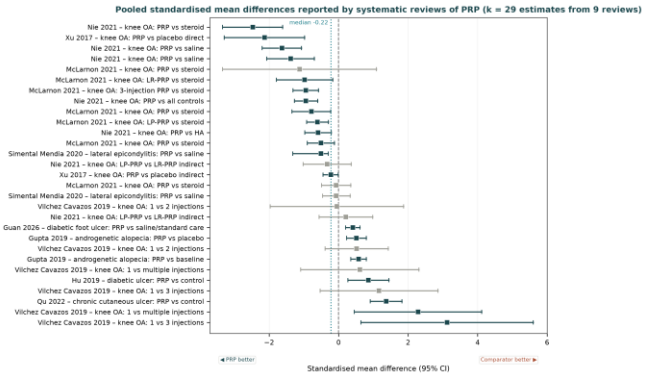

Figure 9: Pooled standardised mean differences across all indications. All 29 SMD estimates from the umbrella stream, ordered by magnitude. Negative values favour PRP for pain and symptom outcomes; positive values favour PRP where the source review coded improvement positively (wounds, alopecia, and injection-number comparisons).

Composition and dose: what actually moves the outcome

Review	Indication	Comparison	Outcome	Months	Estimate (95% CI)	Favours
McLarnon 2021	knee OA	LP-PRP vs steroid	pain	6	-0.61 (-0.92, -0.29) SMD	PRP
McLarnon 2021	knee OA	LR-PRP vs steroid	pain	6	-0.98 (-1.79, -0.17) SMD	PRP
McLarnon 2021	knee OA	single injection PRP vs steroid	pain	6	-0.52 (n.a., n.a.) SMD	neither
McLarnon 2021	knee OA	triple injection PRP vs steroid	pain	6	-0.94 (-1.31, -0.58) SMD	PRP
Bensa 2025	knee OA	high-dose PRP vs placebo	WOMAC total	12	-21.49 (-31.66, -11.32) MD	PRP
Bensa 2025	knee OA	high-dose PRP vs placebo	VAS pain	3	-1.94 (-2.82, -1.06) MD	PRP
Bensa 2025	knee OA	high-dose PRP vs placebo	VAS pain	6	-2.10 (-3.22, -0.98) MD	PRP
Bensa 2025	knee OA	high-dose PRP vs placebo	VAS pain	12	-1.95 (-3.29, -0.60) MD	PRP
Bensa 2025	knee OA	low-dose PRP vs placebo	VAS pain	6	-1.28 (-2.44, -0.12) MD	PRP
Xu 2026	knee OA	LP-PRP vs placebo	WOMAC function	6	-15.26 (-21.91, -8.61) MD	PRP
Xu 2026	knee OA	L-PRP vs LP-PRP	WOMAC function	6	-0.45 (-6.44, 5.53) MD	neither
Xu 2026	knee OA	LP-PRP vs placebo	pain	6	-11.07 (-19.56, -2.57) MD	PRP
Xu 2026	knee OA	L-PRP vs LP-PRP	pain	6	-0.11 (-1.60, 1.38) MD	neither

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

Xu 2026	knee OA	LP-PRP vs placebo	WOMAC function	12	7.32 (-5.17, 19.80) MD	neither
Nie 2021	knee OA	LP-PRP vs LR-PRP indirect	pain	n.a.	-0.33 (-1.02, 0.36) SMD	neither
Nie 2021	knee OA	LP-PRP vs LR-PRP indirect	function	n.a.	0.21 (-0.56, 0.98) SMD	neither
Peng 2022	knee OA	LR-PRP vs HA	WOMAC total	6	-4.29 (-7.66, -0.91) MD	PRP
Peng 2022	knee OA	LR-PRP vs HA	WOMAC total	12	-6.45 (-19.37, 6.47) MD	neither

Table 9: Composition, dose and injection-number comparisons from the umbrella stream. Leukocyte-rich versus leukocyte-poor comparisons are consistently null; platelet dose comparisons consistently favour the higher-dose arm in knee osteoarthritis. Injection-number comparisons favour repeated injection but with wide intervals.

Two patterns separate cleanly. First, the leukocyte question — the most-debated composition variable in the field — is empirically quiet: direct leukocyte-rich versus leukocyte-poor comparisons cross the null in every pooled estimate retrieved, and a published meta-regression of leukocyte content against outcome was likewise null. Second, platelet dose is not quiet. High-concentration PRP outperformed the pooled overall PRP estimate on WOMAC total at 12 months by a wide margin, and a dose-stratified review found a positive gradient across platelet-dose bands in knee osteoarthritis. A parallel analysis in rotator cuff disease found no such gradient, so the dose signal is indication-specific rather than universal.

This is the pivot of the whole analysis. The laboratory stream shows that spin architecture and cumulative dose — not g-force — determine platelet dose, with a between-protocol range of 1.47× to 9.50×. The clinical stream shows that platelet dose is the one composition variable that tracks outcome. And the reporting stream shows that only 7 of 57 trials tell us what dose they gave. The mechanism linking preparation to outcome is not missing; it is unmeasured.

Discussion

Principal findings

Four findings emerge. First, the pooled platelet concentration factor across the published record is 4.63×, not the 3–5× usually quoted, and the spread between protocols (1.47–9.50×) is larger than most clinicians assume. Second, the relative centrifugal force of an individual spin has no measurable relationship with yield across the range in clinical use ($R^2 < 0.01$ for both spins), whereas spin architecture has a large one (Hedges g = 1.20). Third, the evidence that clinically used centrifugation destroys platelets is weaker than its rhetorical prominence: the studies that measured integrity directly mostly found none, and several apparent damage findings are recovery losses that are equally consistent with mechanical entrapment. Fourth, the composition variable that tracks clinical outcome is platelet dose, not leukocyte class — and it is the variable least often reported.

Why g-force fails as a predictor

The null g-force result is not a statistical artefact of a noisy literature; it has a mechanical explanation. Sedimentation under centrifugation is governed by force multiplied by time, and by the geometry of the separation — rotor type, tube fill, plasma column height, the position from which the operator aspirates. A protocol at 200 g for 20 minutes and one at 800 g for 5 minutes deliver comparable cumulative dose

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

and, in this dataset, comparable yield. Reporting only the g-force therefore discards most of the mechanically relevant information. The finding that cumulative g·min performed better than either spin force alone, while still explaining only about one seventh of the variance, points to the remaining share being operator technique and device geometry — the parts of the procedure that no protocol description currently captures.

The practical implication is uncomfortable for kit marketing, which usually foregrounds a proprietary speed. Two kits quoting different g-forces may produce indistinguishable products, while two operators using the same kit may not.

The lysis question, reconsidered

The 3,000 g threshold has become a citation habit. It traces to a single viability study, and while that study is methodologically sound, the condition it tested — 3,000 g sustained for 20 minutes — sits at the outer edge of clinical practice. Against it stand four independent direct measurements of platelet integrity at routine forces that found nothing: P-selectin unchanged during concentration, no CD62P difference between rotor geometries, lactate dehydrogenase unchanged at 1,600 g for 20 minutes, and no difference in activated-platelet fraction between protocols. A historical haematology protocol using 3,000 g for 20 minutes reported 86% platelet harvest without viability loss.

This does not mean centrifugation is biologically inert. Activation markers do rise at 800–1,200 g in at least one careful series, aggregation and morphological change have been documented at 1,000 g on the second spin, and electron microscopy of one double-spin system showed aggregate alteration. The honest summary is that partial, sub-lytic activation is real and force-related, while frank lysis at clinically used settings is not established. Since partial activation may release growth factors prematurely into the supernatant rather than at the treatment site, this remains clinically relevant — but it is a different mechanism from destruction, and it demands different assays.

Growth factors are the wrong reporting target

Growth factor concentrations varied by two to three orders of magnitude between studies and did not correlate significantly with platelet concentration factor. Some of that variance is biological, but much is methodological: whether the sample was activated before assay, whether platelets were lysed to release intracellular stores, which immunoassay platform was used, and whether results were normalised per volume or per million platelets. Until those choices are standardised, reported growth factor concentrations are not comparable across laboratories and should not be used to characterise a product. Platelet dose — the absolute number of platelets delivered, in billions — is measurable with a standard haematology analyser, reproducible, and directly linked in the dose-stratified clinical literature to outcome.

The reporting failure is the rate-limiting step

The most actionable finding in this analysis is not biological. 23 of 57 randomised trials (40%) provide no usable preparation detail, and only 7 state the platelet concentration factor delivered. Meta-analyses of this literature routinely report I^2 above 75% — in this umbrella stream, 45% of pooled estimates exceeded that threshold — and then attribute the heterogeneity to preparation differences they cannot observe. That attribution may well be correct. It is simply untestable with the data as published, and it will remain untestable however many further trials are run under current reporting norms.

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

Several classification frameworks already exist for this purpose, reporting platelet dose, purity, activation state and efficiency. Their existence is not the problem; their adoption is. A minimum dataset of six numbers — whole-blood platelet count, final platelet concentration, final volume, absolute platelet dose in billions, leukocyte concentration, and spin parameters including rotor type — would take a trialist minute to report and would transform the interpretability of the entire field.

Limitations

- The laboratory stream pools preparation arms from heterogeneous protocols and donor populations rather than randomised comparisons; between-arm contrasts are therefore observational and susceptible to confounding by laboratory, device and donor characteristics.
- Several studies contribute multiple arms, which introduces within-study correlation not modelled in the descriptive pooling; the between-architecture contrast was confirmed with a rank-based test to reduce sensitivity to this.
- Only 5 randomised trials reported sufficient arm-level data for participant-level pooling, so the pooled clinical effect is imprecise and not representative of the full trial literature.
- Umbrella estimates overlap substantially in their constituent trials and were deliberately not re-pooled; the distributional summary presented instead cannot be interpreted as a single effect size.
- Growth factor comparisons are limited by assay heterogeneity, and the small number of arms reporting each analyte limits the power of the correlation analyses.
- Publication and language bias were not formally assessed for the laboratory stream, where funnel-plot methods are not applicable to single-arm descriptive data.

Implications for practice and research

For clinicians, three recommendations follow from these data. Choose a double-spin or buffy-coat protocol when platelet dose matters, because architecture, not speed, drives yield. Stop selecting kits on quoted g-force. Measure and record the platelet count of the product delivered — a single analyser run — so that individual practice can be audited against the dose-response literature.

For researchers, the priority is not another placebo-controlled trial of unspecified PRP. It is dose-stratified randomisation: the same preparation system, deliberately delivering defined platelet doses, in a single indication, with product characterization reported for every participant. The dose signal already visible in the pooled literature is strong enough to justify that design and too confounded to be settled without it.

Conclusion

Centrifugation determines what is in the syringe. Spin architecture and cumulative centrifugal dose govern platelet concentration — pooled at 4.63× across the published record, ranging from 1.47× to 9.50× — while the raw g-force of any single spin does not. The widely repeated claim that clinically used centrifugation lyses platelets is not supported by direct integrity assays, although sub-lytic activation at higher forces is real. Growth factor concentrations are too assay-dependent to serve as a product descriptor. In the clinical literature, PRP outperforms its comparators more often than not, and platelet dose is the only composition variable with a consistent signal — yet 23 of 57 randomised trials do not report their preparation at all. The chain from centrifuge to patient is not broken at the biology. It is broken at the methods section, and it can be repaired with six numbers.

DOI: https://doi.org/10.52793/JSCR.2026.7(3)-93

Declarations

Not applicable. This study analysed previously published data only.

Availability of data and materials

All extracted data rows, with their source bindings, are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

No funding.

Authors' contributions

[MHK, CAPMR] conceived and designed the study. All authors read, revised and approved the final manuscript.

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