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Review Article

Primary Dysmenorrhea at the Interface of Uterine Hypercontractility, Neuroimmune Signaling, and Pain Sensitization: An Integrative Review of Ayurvedic Therapeutic Rationale and Clinical Evidence

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ABSTRACT

Background

Primary dysmenorrhea is commonly explained through excess endometrial prostaglandin activity, uterine hypercontractility, and impaired perfusion. This account is clinically useful but does not fully characterize variation in pain, nonresponse to treatment, or symptoms extending beyond menstruation. Ayurvedic approaches offer additional therapeutic rationales, although conceptual correspondence and clinical efficacy require separate evaluation.

Objective

To integrate evidence on uterine, inflammatory, and pain-processing mechanisms with Ayurvedic descriptions and critically appraise their implications for treatment and research.

Methods

A critical integrative narrative review used targeted searches of PubMed-indexed literature, publisher records, Cochrane reviews, and an accessible scholarly edition of Charaka Samhita. Searches were conducted on 4 November 2026. Human mechanistic studies, treatment trials, evidence syntheses, and primary classical material were selected for relevance. No participant recruitment, original clinical dataset, or quantitative pooling was undertaken.

Results

Prostaglandin-mediated uterine activity remains the best-established peripheral explanation, while menstrual-effluent and immune-expression studies suggest additional inflammatory processes. Experimental pain findings support altered pain processing in some populations but do not establish universal central sensitization. Ayurvedic descriptions of Udavartini Yonivyapad provide a historical framework for painful menstruation without proving equivalence between Apana Vata and a molecular pathway. Botanical and procedure-based studies contain preliminary signals of benefit, but formulation variability, small samples, inadequate comparators, and incomplete safety reporting limit inference.

Conclusion

Integrative care should preserve diagnostic vigilance, assess pain and functioning separately, and distinguish traditional rationale from pharmacological plausibility and demonstrated benefit. Progress requires reproducible formulations, credible comparisons, cycle-specific outcomes, and explicit tests of proposed mechanisms rather than claims of equivalence based on symptom improvement alone.

Keywords: Primary Dysmenorrhea; Uterine Hypercontractility; Prostaglandins; Neuroimmune Signalling; Pain Sensitization; Ayurveda; Udavartini Yonivyapad; Integrative Medicine

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INTRODUCTION

Primary dysmenorrhea denotes painful menstruation without an identified underlying pelvic disorder. It can disrupt education, employment, sleep, and social participation. Its designation as primary describes the clinical classification; it does not imply that the pain lacks a biological basis or that all affected individuals share the same mechanism. Contemporary clinical reviews emphasize diagnosis from the pattern of symptoms, appropriate assessment, and treatment tailored to the individual [1].

Recurrent menstrual pain also has consequences beyond a short episode of cramping. The broader literature documents changes in pain sensitivity, mood, sleep, and quality of life, although these findings differ across populations and methods [2]. Persistent symptoms must not simply be normalized. In adolescents and young adults, lack of improvement after appropriate treatment warrants reassessment for secondary causes, particularly endometriosis [3].

The conventional mechanistic account links endometrial prostanoid production to dysrhythmic uterine contractions, reduced perfusion, and nociceptor activation. It is supported by physiological observations and the effectiveness of treatments that reduce prostaglandin production or endometrial activity [4]. However, clinical nonresponse does not by itself identify a different mechanism. It can also reflect timing, adherence, an unsuitable treatment, adverse effects, or an unrecognized secondary disorder.

Ayurvedic practice approaches painful menstruation through a different conceptual vocabulary and may combine medicines, local therapies, and behavioral advice. The scientific task is to preserve the meaning of those descriptions while specifying which proposed outcomes and mechanisms can be tested. This review therefore examines three levels of explanation uterine activity, inflammatory signalling, and altered pain processing and evaluates the strength of the bridge between Ayurvedic rationale and contemporary clinical evidence. The aim is an accountable framework for interpretation, not a claim that historical constructs and molecular mechanisms are interchangeable.

MATERIALS AND METHODS

Review design

This is a critical integrative narrative review. Integration refers to comparison across explanatory traditions and evidence types rather than statistical combination. Human studies of primary dysmenorrhea were prioritized. Studies of endometriosis, animal models, or isolated compounds were not treated as direct proof of a treatment effect in primary dysmenorrhea. The review collected no original patient information and did not analyze the authors' dissertations or institutional clinical records.

Sources and selection

Targeted online searches on 4 November 2026 used combinations of primary dysmenorrhea, prostaglandins, uterine hypercontractility, menstrual effluent, cytokines, neuroimmune signalling, sensitization, experimental pain, Ayurveda, Udavartini Yonivyapad, Chaturbeeja, ginger, fenugreek, heat, exercise, and treatment trials. PubMed-indexed records, journal and publisher pages, Cochrane summaries, and relevant reference lists were consulted. Exact-title searches were used to verify bibliographic details. Classical discussion was anchored to the accessible Charaka Samhita chapter on genital-tract disorders, with chapter and verse locations reported rather than invented quotations or unspecified edition page numbers.

The selection emphasized clinically relevant reviews, primary mechanistic investigations, controlled treatment studies, directly relevant Ayurvedic reports, and measurement or reporting guidance. Full texts were used where accessible; statements based on abstract-only records were restricted to explicitly reported information. This was not an exhaustive database search, independently screened systematic review, or formal assessment of certainty using GRADE. No flow diagram, pooled estimate, or complete inventory of eligible studies is claimed.

Interpretive framework

Each source was considered in relation to population, diagnostic definition, intervention or biological specimen, comparator, outcome, and permissible inference. Classical rationale, preclinical plausibility, observational association, and comparative clinical benefit were evaluated separately. Published syntheses were interpreted with attention to heterogeneity, imprecision, and the suitability of comparisons. The tables summarize selected evidence and interpretive safeguards; they are not formal treatment algorithms or comprehensive evidence maps.

RESULTS OF THE NARRATIVE SYNTHESIS

Uterine activity provides a strong peripheral account

Progesterone withdrawal and endometrial breakdown create conditions for increased production of prostaglandins and related lipid mediators. Greater myometrial activity and altered uterine perfusion can generate ischemic stress and activate pain pathways. The resulting model connects the menstrual cycle to the timing and cramping character of symptoms [4]. Its clinical strength is that it identifies a plausible target and predicts benefit from reducing prostanoid production. It does not establish that pain intensity is a direct linear readout of prostaglandin concentration.

A 2025 systematic review of menstrual-effluent biomarkers identified recurring evidence of elevated prostaglandins, while also emphasizing small samples and methodological limitations. Other candidate mediators, including 12-HETE and platelet-activating factor, were represented in a much smaller evidence base [5]. This distinction matters: a reproducible prostaglandin

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association and an exploratory biomarker finding should not receive identical interpretive weight.

The same review also found that studies of NSAID effects on prostaglandins commonly used multiple-day protocols, limiting conclusions about acute effects after a single dose [6]. Sampling time, menstrual volume, assay characteristics, and medication exposure can all affect comparisons. A treatment-associated reduction in pain is therefore not sufficient evidence that a specific patient had excessive prostaglandin production or that an intervention acted through that pathway.

Neuroimmune signalling remains a developing explanatory layer

Menstrual inflammation is a physiological process; the relevant question is when its timing, magnitude, or interaction with sensory pathways contributes to disproportionate pain. Ma and colleagues investigated cytokine-related gene expression in peripheral blood mononuclear cells across menstrual phases. Their case-control study included six participants with primary dysmenorrhea and three controls, making it exploratory rather than a basis for population-wide immune classification [6].

Several inferential boundaries follow. Peripheral-cell gene expression is not identical to cytokine concentration in the uterus, and neither directly measures neural sensitization. A cross-sectional difference can reflect cycle timing, medication, comorbidity, or responses to pain. Describing a finding as neuroimmune requires particular care when only one component of that proposed interaction has actually been measured.

More direct local sampling offers complementary information. In a 2025 adolescent menstrual-effluent study, higher prostaglandin F2α and additional oxylipin findings were associated with dysmenorrhea, while correlations between prostaglandin concentrations and pain were modest [7]. This supports investigation of a broader inflammatory context without establishing that a multi-marker panel can diagnose primary dysmenorrhea or distinguish it reliably from endometriosis.

The appropriate synthesis is that inflammatory processes can contribute to peripheral nociceptive input, while the clinical significance of most proposed markers remains unresolved. A credible mechanistic study should establish where a marker is measured, when it changes, how it relates to symptoms, and whether manipulating the proposed pathway changes a patient-important outcome. Without those links, a molecular association remains a candidate explanation rather than an actionable biomarker.

Recurrent pain can coexist with altered pain processing

Pain associated with primary dysmenorrhea is not necessarily confined to the uterus. A systematic review of experimental and procedural pain responses reported evidence of altered sensitivity across studies, but outcomes varied by stimulus, anatomical site, and menstrual phase [8]. Such observations justify examining peripheral and central pain processing together. They do not establish that every individual has central sensitization or that recurrent menstrual pain inevitably becomes a chronic generalized pain disorder.

Menstrual phase is a methodological variable rather than a minor reporting detail. Hormonal state, the presence of active menstrual pain, and the location of the experimental stimulus can influence interpretation. A review of pain across the menstrual cycle highlighted the complexity and inconsistency of this literature [9]. Studies conducted only during menstruation cannot readily distinguish an enduring characteristic from a temporary response to ongoing pain.

Evidence of heightened sensitivity at sites remote from the pelvis may be compatible with generalized pain amplification, but alternative explanations include expectation, sleep disruption, distress, medication exposure, and differences in participant selection. These factors should be assessed rather than used to dismiss the pain as psychological. Similarly, brain-imaging differences should not be labeled irreversible injury without longitudinal evidence.

Table 1 separates the principal mechanistic levels. The levels may interact, but coexistence does not establish a single causal sequence from uterine contraction to immune change to central sensitization. Directionality may differ between individuals, and the same pain severity can arise from different combinations of peripheral input and pain processing.

Domain	Evidence or observation	Plausible interpretation	What remains unproven
Uterine activity	Prostanoid-associated contractility and impaired perfusion	A peripheral source of nociceptive input	A direct linear relationship between one mediator and individual pain [4,5].
Local inflammatory signalling	Menstrual-effluent prostaglandins and other lipid mediators	Several pathways may contribute to local nociception	A validated diagnostic panel or a confirmed treatment-selection rule [5-7].
Peripheral immune expression	Cycle-related cytokine gene-expression differences	A potential systemic correlate of menstrual pain	Equivalence to uterine inflammation or neural immune activation [6].

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Pain processing	Altered responses to experimental stimuli in some studies	Peripheral or central amplification may be relevant	Universal central sensitization or inevitable progression to chronic pain [8,9].

Table 1. Mechanistic domains and limits of inference

Ayurvedic rationale should retain its conceptual boundaries

Charaka Samhita describes painful and difficult menstrual discharge under Udavartini Yonivyapad in Chikitsa Sthana 30, verses 25–26. Its explanatory account uses vayu and altered movement associated with suppression of natural urges. Management discussed in verses 110–111 includes unction, fomentation, and basti-based approaches [10]. These are historical therapeutic and explanatory descriptions; their presence in a classical source is not equivalent to contemporary trial evidence.

The most defensible correspondence is clinical: both accounts engage with painful menstruation and disturbed function. Equating Apana Vata directly with prostaglandins, a named autonomic pathway, or uterine smooth-muscle physiology would exceed the evidence. The classical description of altered movement should also not be presented as proof of anatomical retrograde menstruation or endometriosis. The comparison can instead generate questions about pain, contractility, tolerability, and functional recovery.

A small clinical report by Jahan and colleagues compared an Uttara Vasti intervention using Trivrit and Lasuna oil with an NSAID in 36 allocated participants [11]. It demonstrates that an Ayurvedic procedural rationale can be operationalized for clinical investigation. It does not, by itself, establish broad comparative effectiveness, long-term safety, or suitability for routine invasive treatment. Procedural risk assessment, infection prevention, reproductive considerations, and adequate adverse-event ascertainment remain essential.

The translational boundary is productive rather than restrictive. An investigator can define a reproducible intervention grounded in traditional practice, specify its intended outcomes, and compare those outcomes with appropriate care. The intervention does not need a forced molecular translation to be testable. Conversely, a plausible molecular narrative cannot compensate for an inadequately controlled clinical comparison.

Established treatments provide the comparison standard

The Cochrane review of NSAIDs supports their effectiveness for dysmenorrhea while recognizing adverse effects and limitations in comparative evidence between individual agents [12]. Its relevance to integrative research is methodological as well as clinical. An active comparator must be delivered appropriately; an inadequately timed or poorly adhered-to comparator can create an apparent advantage for another intervention without establishing genuine superiority.

The 2023 Cochrane review of combined oral contraceptive pills also supports pain reduction compared with placebo, with treatment-specific harms and uncertainty for some comparisons.[13] Choice of hormonal therapy depends on individual suitability, preferences, and broader reproductive-health considerations. Neither class should be portrayed as universally appropriate or universally inadequate to create a rhetorical opening for traditional treatment.

In practice, persistent pain after an adequate treatment trial should trigger a review of diagnosis, use of treatment, and functional impact. It should not automatically trigger escalation of an unverified integrative explanation. Clinical guidance concerning secondary causes, particularly endometriosis, remains applicable throughout integrative care [3].

Botanical evidence is product-specific

The 2016 Cochrane review of dietary supplements found limited, generally low- or very-low-quality evidence and inadequate adverse-event reporting across many comparisons [14]. A subsequent medicinal-plant review identified favorable findings in several trials but similarly emphasized low or unclear study quality [15]. These reviews support investigating particular preparations rather than treating herbal medicine as one intervention with one efficacy estimate.

A 2026 synthesis included 47 studies, with 22 contributing to meta-analysis. It reported a signal of pain reduction against placebo but substantial heterogeneity and low to very low certainty [16]. Its pooled comparison with conventional drugs did not show a statistically significant difference. That result should not be interpreted as demonstrated equivalence: failure to detect a difference in heterogeneous, imprecise studies is distinct from meeting a prespecified noninferiority or equivalence criterion.

Ginger illustrates why product and study details matter. Ozgoli and colleagues compared ginger with mefenamic acid and ibuprofen in a clinical study [17]. An observed lack of difference in such a comparison does not automatically establish interchangeability, especially when sample size, blinding, preparation, and clinically acceptable margins have not been designed to answer that question. Evidence for one dose and preparation cannot be transferred uncritically to another extract or combination.

Fenugreek seed has also been evaluated in a placebo-controlled study reporting reductions in dysmenorrhea severity and systemic symptoms [18]. This is a product-specific clinical signal, not proof that every fenugreek-containing formula has the same effect. Combination products can differ in component ratios, extraction, chemical composition, exposure, and interactions. Any proposed synergy requires a design capable of separating combination effects from those of individual ingredients.

Dhiman and Chaudhary reported Chaturbeeja powder use in 25 participants in an observational investigation [19]. Its direct relevance to Ayurvedic practice makes it worth examining, but

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an uncontrolled before-and-after change is vulnerable to expectation, natural cycle variation, regression to the mean, and co-interventions. Powder-based findings do not establish effectiveness of a concentrated Ghanvati formulation. Extraction and processing can alter the product, so pharmaceutical comparability and clinical performance must be evaluated separately.

Table 2 identifies the appropriate level of interpretation for these selected sources. It deliberately distinguishes evidence of a preliminary signal from evidence sufficient to support routine comparative claims.

Intervention or evidence source	Evidence type	Main contribution	Principal qualification
NSAIDs	Cochrane review	Supports pain relief against placebo	Adverse effects and individual suitability matter; one agent is not proven best [12].
Combined oral contraceptives	Updated Cochrane review	Supports pain reduction against placebo	Reproductive preferences and contraindications affect selection [13].
Plant-derived therapies	2026 systematic review and meta-analysis	Suggests potential analgesic benefit	High heterogeneity and low certainty; no established class-wide equivalence [16].
Ginger and fenugreek	Individual clinical trials	Supports preparation-specific investigation	Results cannot be generalized across extracts, doses, or combinations [17,18].
Chaturbeeja powder	Small observational report	Directly relevant preliminary Ayurvedic signal	Uncontrolled improvement does not establish causality; Ghanvati is a different product [19].
Uttara Vasti preparation	Small comparative report	Demonstrates feasibility of a procedural comparison	Limited scale and reporting restrict safety and effectiveness conclusions[11].

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Table 2. Selected therapeutic evidence and implications for integrative practice

Supportive interventions require their own evidence

Exercise and local heat may fit within a broader management plan, but their evidential status should be evaluated independently of the tradition in which they are recommended. A Cochrane review of exercise found potential benefit while identifying limitations in study quality, comparators, and outcome measurement [20]. A systematic review of heat therapy likewise reported pain-relief findings with limitations in the underlying evidence [21].

These findings cannot validate an entire bundled regimen. If medicine, massage, heat, exercise, and counseling are introduced together, improvement cannot identify which component contributed or whether the components interacted. A pragmatic study can evaluate the package as delivered, but its conclusion must concern that package. An explanatory study of a component or mechanism requires a more focused comparison.

DISCUSSION

Integration requires a sequence of testable claims

An integrative explanation should distinguish four propositions: the intervention is recognizable and reproducible; it changes a biological or behavioral process; it improves an outcome important to patients; and the process change accounts for that improvement. Evidence supporting one proposition does not establish the others. A chemically characterized preparation can be ineffective, while an effective intervention can have a mechanism that remains incompletely understood.

This distinction is particularly important for neuroimmune claims. Pain reduction after a botanical treatment does not prove cytokine modulation. A change in circulating inflammatory markers does not prove reduced uterine nociception. Correlated changes in a marker and pain remain vulnerable to common causes. Demonstrating mechanism requires an appropriately timed assessment and, where feasible, evidence from experimental manipulation or a sufficiently supported mediation analysis.

The same reasoning applies to Ayurvedic interpretation. A clinical benefit does not scientifically prove that a molecular pathway is identical to a traditional construct. It can support the clinical utility of a defined intervention under specified conditions. This is a stronger foundation for interdisciplinary work because it makes the contribution assessable without requiring either explanatory tradition to be reduced to the vocabulary of the other.

Assessment must capture function and the menstrual cycle

Pain intensity is indispensable but incomplete. Two individuals with similar peak ratings may differ in pain duration, rescue-medication use, school attendance, sleep, and ability to complete usual activities. WaLIDD was developed to capture dimensions including working ability, location, intensity, and duration in university students [22]. Its original study supports a specific measurement application; it does not establish universal diagnostic accuracy or responsiveness in every clinical population.

Prospective symptom diaries can clarify timing and reduce dependence on a single retrospective summary. Baseline and follow-up assessments should use comparable menstrual windows and document co-interventions. Recording both peak pain and cumulative interference can distinguish shorter severe episodes from prolonged lower-intensity discomfort. A cycle without reported pain must also be interpreted with hormonal use, missed menstruation, and medication exposure in view.

Apparent benefit is difficult to interpret when analgesic use is unrecorded. A lower pain score accompanied by increased rescue medication is different from pain relief with less medication and improved functioning. Similarly, the absence of reported adverse events in a small study is not affirmative evidence of safety. Table 3 outlines an outcome framework intended to improve the interpretability of future studies rather than prescribe a validated protocol.

Domain	Information to capture	Reason for inclusion	Interpretation safeguard
Diagnostic classification	Symptom history, examination or investigations when indicated, secondary-cause assessment	Defines the population being treated	A normal scan alone does not exclude every cause of secondary pain [3].
Menstrual timing	Cycle phase, bleeding onset, intervention timing, comparable follow-up windows	Separates phase effects from treatment effects	Avoid comparing non-equivalent periods [9].
Pain and function	Intensity, duration, interference, attendance, sleep, patient priorities	Captures patient-important benefit	Interpret scale performance in the intended population [22].
Treatment exposure	Product identity, dose, adherence, comparator delivery, rescue medication	Makes a comparison reproducible	Do not infer benefit from an inadequately delivered control [12-23].
Harms and durability	Prespecified adverse events, discontinuation, recurrence after treatment	Tests acceptability and persistence	Missing reports are not evidence that no harms occurred [14-23].

Table 3. Proposed minimum outcome and reporting domains

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Botanical identity is part of the clinical intervention

The elaborated CONSORT statement for herbal interventions emphasizes reporting that allows the actual product to be identified and evaluated [23]. For an Ayurvedic combination this includes botanical identities, plant parts, ingredient ratios, preparation method, dose, manufacturer or source, and quality-control information appropriate to the product. Standardization to one marker cannot be assumed to establish equivalence of all pharmacologically relevant constituents.

The issue is especially important when translating a traditional powder to an extract, tablet, or Ghanvati. Equal tablet mass does not demonstrate equal exposure to the original constituents. A new formulation may improve adherence or convenience, but those potential advantages are separate from proof of preserved efficacy and safety. Product characterization should therefore accompany, rather than substitute for, clinical evaluation.

Safety assessment should consider the actual formulation, concomitant medication, reproductive context, and observed adverse effects. It is inappropriate to infer safety from the words natural, traditional, or polyherbal. Likewise, a plausible interaction in a laboratory experiment should not be presented as a demonstrated clinical interaction without supporting human evidence. Transparent uncertainty permits useful decisions without overstating either benefit or risk.

Research priorities

The most immediate priority is reproducible clinical research rather than progressively more elaborate mechanistic terminology. Adequate baseline characterization, justified sample size, allocation concealment, credible blinding where feasible, prespecified outcomes, and transparent handling of missing observations would substantially strengthen interpretation. An active-comparator trial should distinguish superiority from noninferiority and justify any margin before data are analyzed.

A second priority is to connect proposed mechanisms with the same intervention studied clinically. Results from an isolated constituent at an experimental concentration cannot establish the effect of a multi-ingredient oral product at a clinical dose. Mechanistic substudies should specify a small number of plausible targets, align specimen collection to the menstrual

cycle, and measure exposure where feasible. A large exploratory marker panel increases the need for prespecification and independent confirmation.

A third priority is durability. Most short trials cannot establish whether benefits persist after treatment, recur on discontinuation, or change later susceptibility to persistent pelvic pain. Longitudinal investigation is required before describing an intervention as disease-modifying or preventive of central sensitization. Response over several cycles remains clinically relevant even when the mechanism or long-term trajectory is unresolved.

A fourth priority is diagnostic safety. A study must state how primary dysmenorrhea was distinguished from secondary conditions and how new symptoms or inadequate response were handled. Integrative management should support appropriate reassessment rather than create a pathway in which persistent pain is repeatedly attributed to an unverified constitutional or molecular explanation.

Limitations

This review is selective and interpretive. Targeted searches can miss relevant studies, full-text access was incomplete, and no duplicate screening, formal risk-of-bias assessment, or new quantitative synthesis was undertaken. The chosen studies differ in age, diagnosis, cycle phase, intervention identity, and outcome. The tables should therefore not be read as a ranked treatment guideline.

The classical discussion uses an accessible scholarly digital edition and selected, directly relevant passages. It is not an exhaustive philological comparison of all manuscripts, commentaries, or Ayurvedic traditions. Historical rationale is kept distinct from contemporary trial evidence. The authors' own clinical or dissertation experience was not treated as published evidence, and no unpublished outcome or safety claim is introduced.

The proposed integrative framework also remains unvalidated. It cannot classify patients into established mechanistic subtypes or determine which patient should receive a particular Ayurvedic product. Its contribution is to specify the evidence needed to make such claims defensible and to identify where current evidence already supports useful, narrower conclusions.

CONCLUSION

Primary dysmenorrhea is best understood through a layered account in which uterine nociceptive input interacts with inflammatory processes, pain processing, and functional context. Prostaglandin-related mechanisms remain the strongest established peripheral explanation, while additional neuroimmune and sensitization findings are promising but incompletely translated into clinical decisions.

Ayurvedic descriptions and interventions can contribute to this research agenda when their rationale, product identity, outcomes, and limitations are made explicit. Conceptual correspondence does not establish biological equivalence, and preliminary symptom improvement does not prove a mechanism or comparative superiority. Integrative care should retain appropriate evaluation for secondary causes, measure pain and functioning, and communicate the level of evidence supporting each intervention. The next scientific advance will come from more discriminating questions and more reproducible comparisons, rather than stronger claims attached to uncertain findings.

DECLARATIONS

CONFLICT OF INTEREST

The authors declare no conflicts of interest relevant to this manuscript.

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