ISSN: 2710-0723

Clinical Evaluation of an Acer truncatum Seed Oil–Phosphatidylserine Complex for Neurodevelopment Support

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Abstract

Background: Acer truncatum seed oil is a nervonic-acid–rich botanical lipid source with theoretical relevance to myelin-oriented neurodevelopment, while phosphatidylserine (PS) is a membrane phospholipid with a substantially larger human evidence base in cognition and attention. We evaluated whether the formulation logic of an Acer truncatum seed oil–PS complex is supported by published clinical data that are relevant to neurodevelopment, attention, memory, and phospholipid-mediated brain support. Methods: We summarized the formulation architecture described in the source patent dossier and searched primary biomedical literature through March 2026 for randomized or controlled human studies involving PS, PS-omega-3 preparations, sphingomyelin-fortified milk, and related phospholipid-based neurocognitive interventions. We prioritized pediatric and neurodevelopment-relevant studies and used adult trials only when they clarified efficacy direction, dose range, or safety. Results: The clinical evidence most directly supporting the formulation is concentrated in the PS and phospholipid axis. In very-low-birth-weight infants, sphingomyelin-fortified milk improved neurobehavioural outcomes at follow-up. In children with ADHD, soy-derived PS improved ADHD symptoms and short-term auditory memory, and PS-omega-3 reduced hyperactive/impulsive symptoms while remaining well tolerated over 30 weeks. In healthy children aged 8–12 years, sunflower-derived PS was neutral in the total cohort but improved visuospatial memory in a predefined lower-performing subgroup. In older adults with memory complaints or mild cognitive impairment, PS-containing interventions improved selected memory and cognitive measures in several randomized trials. By contrast, we did not identify a published randomized human trial of the exact Acer truncatum seed oil–PS finished formulation. Conclusions: The current clinical basis for this composition rests on convergent evidence for phosphatidylserine-centered brain support rather than on a completed trial of the exact commercial-ready formulation. The combination of nervonic-acid–rich Acer truncatum seed oil with PS and phospholipid cofactors remains biologically plausible and clinically testable, particularly in populations with high myelination demand or suboptimal neurocognitive performance.

Keywords

Acer Truncatum Seed Oil; Phosphatidylserine; Nervonic Acid; Neurodevelopment; Cognition; Randomized Controlled Trial; Translational Nutrition.

1. Introduction

We view this formulation as a neurodevelopment-support platform built around two linked ideas: a nervonic-acid–rich lipid substrate and a phosphatidylserine-centered membrane-support system. The source patent describes Acer truncatum seed oil, phosphatidylserine, cephalin, DHA-containing phospholipids, arachidonic-acid phosphatidylinositol, sialic acid,

ISSN: 2710-0723

phytosterols, and d-alpha-tocopherol within a lipid–phospholipid carrier intended for brain-directed delivery. In practical terms, that design attempts to unite structural lipid supply, membrane signaling support, oxidative protection, and delivery efficiency in a single oral matrix [1].

We regard this architecture as scientifically interesting for a specific reason: the human intervention literature is not evenly distributed across its components. Phosphatidylserine has a long randomized-trial history in attention and memory, while human intervention evidence specific to Acer truncatum seed oil or isolated nervonic-acid supplementation remains sparse. A publishable paper therefore has to distinguish clearly between direct clinical evidence for the exact finished composition and the broader human evidence supporting its component logic.

Our objective in the present article is not to claim that the exact formulation has already completed a confirmatory human trial. Instead, we evaluate whether the formulation's proposed clinical positioning is supported by published human evidence that is mechanistically and translationally relevant to neurodevelopment. To do that, we summarize the source composition, organize the strongest available clinical trials, and interpret how those results bear on the formulation's plausibility, likely target populations, and remaining evidence gaps.

2. Methods

2.1. Formulation Concept and Evidence Strategy

Table 1. Source formulation logic and translational evidence status.

Component / design domain	Role in the formulation	Human clinical evidence status	Interpretation
Acer truncatum seed oil	Nervonic-acid–rich lipid matrix intended to support membrane and myelin-oriented neurodevelopment	Direct randomized human trial of the exact Acer truncatum oil formulation was not identified	Promising differentiator, but presently supported mainly by formulation logic rather than direct human efficacy
Phosphatidylserine (PS)	Neuronal membrane phospholipid; cognition, attention, and signaling support	Multiple randomized or controlled human trials in children and older adults are available [3-9]	Primary clinical anchor of the composition
PS plus omega-3 phospholipids	Comparator-relevant membrane and delivery support	Randomized PS-omega-3 trials show efficacy and safety signals in ADHD and memory-complaint populations [4,5,7]	Supports the use of phospholipid pairing rather than PS alone
Myelin-oriented phospholipid support	Early-life structural support for neurodevelopment	Infant sphingomyelin-fortified milk trial showed favorable neurobehavioural outcomes [2]	Indirect but relevant support for the formulation's myelination narrative
Auxiliary phospholipid/lipid cofactors	Cephalin, DHA-phospholipid, arachidonic phosphatidylinositol, sialic acid, phytosterols, vitamin E	Human evidence for the exact combination is not yet available	Mechanistically coherent, but requires product-specific validation

ISSN: 2710-0723

We extracted the formulation concept from the source patent dossier and reorganized it into a publication-oriented evidence framework. The source material identifies Acer truncatum seed oil as the core lipid matrix and combines it with phosphatidylserine and additional phospholipid cofactors intended to support neurodevelopment-oriented delivery [1].

We then searched primary biomedical literature for human intervention studies published through March 2026 that were relevant to at least one of four translational pillars: (1) phosphatidylserine and pediatric attention or cognitive outcomes, (2) phosphatidylserine combined with omega-3 fatty acids, (3) phospholipid- or sphingomyelin-related early-life neurodevelopment, and (4) comparator phospholipid interventions that help interpret dose, tolerability, or expected effect direction. We prioritized randomized or controlled studies and extracted population, dose, duration, endpoints, and major findings.

We intentionally used a graded evidence approach. Pediatric and infant trials received the greatest interpretive weight because the formulation is positioned for neurodevelopment support. Adult PS trials were retained when they added important information on memory outcomes, biological activity, or safety. Studies without human outcomes were not used as clinical anchors, although they informed mechanistic interpretation.

3. Results

3.1. Human Clinical Evidence Relevant to the Formulation

The reviewed evidence indicates that the formulation's most defensible clinical anchor is phosphatidylserine. Across the pediatric, infant, and adult literature, PS repeatedly shows biologic activity in domains that matter to neurodevelopment-support positioning, including attention, short-term memory, verbal recall, and tolerability [2-9].

The human evidence is not uniformly positive, which is important for interpretation. Some trials show clear between-group advantages, whereas others show benefit only in predefined subgroups or lower-performing participants. We view that pattern as a signal that population selection matters as much as ingredient identity. It suggests that the exact Acer truncatum seed oil–PS complex would be better evaluated in at-risk or nutritionally suboptimal populations than in an unrestricted healthy cohort.

Table 2. Published human clinical trials relevant to the neurodevelopment-support positioning of the formulation.

Study	Population / design	Intervention	Duration	Main outcome	Relevance to the formulation
Tanaka et al., 2013 [2]	24 very-low-birth-weight infants; randomized controlled trial	Sphingomyelin-fortified milk vs control milk	Follow-up to 18 months	Better BSID-II behavior rating, Fagan scores, VEP latency, and sustained attention in the fortified group	Supports phospholipid-driven early neurodevelopment and myelination-oriented positioning
Hirayama et al., 2014 [3]	36 children (4–14 y) with untreated ADHD; randomized, double-blind,	Soy-derived PS 200 mg/day vs placebo	2 months	Improved ADHD symptoms, short-term auditory memory, and inattention/impulsivity measures	Direct pediatric support for the PS pillar

ISSN: 2710-0723

	placebo-controlled				
Manor et al., 2012 [4]	Children with ADHD; double-blind, placebo-controlled trial with open-label extension	PS-Omega3 300 mg/day vs placebo, followed by open-label PS-Omega3	15 weeks double-blind plus 15 weeks extension	Reduced restless/impulsive symptoms; subgroup benefit in hyperactive/impulsive and dysregulated children	Shows that PS combined with omega-3 lipids can be clinically active in pediatric attention disorders
Manor et al., 2013 [5]	200 children with ADHD; randomized safety study	PS-Omega3 300 mg/day, then 150 mg/day in extension	30 weeks total	No adverse effect on growth, vitals, or routine safety markers	Supports feasibility and tolerability of long-term PS-omega-3 use
Friling et al., 2025 [6]	Healthy neurotypical children aged 8–12 y; randomized, placebo-controlled	Sunflower-derived PS 100 mg/day	12 weeks	No overall cohort effect; predefined lower-performing subgroup improved on visuospatial memory; safe	Suggests target-population selection is important
Vakhapova et al., 2010 [7]	157 non-demented older adults with memory complaints; double-blind placebo-controlled	PS-DHA vs placebo	15 weeks	Improved verbal immediate recall and responder profile in PS-DHA group	Supports biological activity of PS–omega-3 combinations
Kato-Kataoka et al., 2010 [8]	78 older adults with memory complaints; randomized, double-blind, placebo-controlled	Soy-PS 100 or 300 mg/day vs placebo	6 months	Delayed verbal recall improved, especially in lower-baseline subgroup; good safety	Shows sustained PS activity over a longer supplementation period
Duan et al., 2025 [9]	190 Chinese older adults with mild cognitive impairment; randomized, double-blind, placebo-controlled	PS-containing multinutrient supplement vs placebo	12 months	Improved arithmetic, similarities, and short-term memory; higher serum ALA, DHA, EPA, acetylcholine, GABA, and 5-HT	Supports clinically measurable PS-centered cognitive effects in a vulnerable population

ISSN: 2710-0723

3.2. Integrated Clinical Interpretation

The strongest pediatric efficacy signal comes from phosphatidylserine itself. In untreated children with ADHD, soy-derived PS at 200 mg/day for two months improved ADHD symptoms, short-term auditory memory, and aspects of inattention/impulsivity relative to placebo [3]. In a larger 15-week trial, PS-omega-3 reduced the restless/impulsive domain and improved parent-rated emotional impact, with sustained benefit in the open-label extension; a paired safety analysis found no concerning effects on growth or routine safety parameters over 30 weeks [4,5].

Healthy-child data are more selective rather than uniformly positive. In the 2025 randomized trial of sunflower-derived PS in neurotypical children aged 8–12 years, the total cohort did not improve on the primary or secondary cognitive outcomes, but the prespecified subgroup with consistently below-median baseline performance showed benefit on visuospatial memory [6]. We consider this a clinically useful result because it tempers overstatement and suggests that baseline vulnerability may determine who benefits most from phosphatidylserine-based nutrition support.

The myelination-oriented rationale for Acer truncatum seed oil is supported more indirectly. We did not identify a randomized human trial of Acer truncatum seed oil itself, but we did identify a neonatal randomized controlled trial in which sphingomyelin-fortified milk improved behavior rating scale performance on BSID-II, Fagan test scores, VEP latency, and sustained attention in very-low-birth-weight infants [2]. Although this is not a nervonic-acid trial, it is highly relevant to the formulation's myelin-support narrative because it shows that phospholipid manipulation in early life can translate into measurable neurobehavioural outcomes.

Adult PS trials provide additional evidence that the phosphatidylserine domain is clinically active rather than hypothetical. PS-DHA improved verbal immediate recall in non-demented elderly with memory complaints over 15 weeks [7]. Soy-derived PS administered for six months improved delayed verbal recall in Japanese older adults with memory complaints, particularly among those with lower baseline performance [8]. A newer 12-month randomized trial in Chinese older adults with mild cognitive impairment also reported improvements in arithmetic, similarities, and short-term memory, together with higher serum n-3 PUFAs and neurotransmitter-related markers [9]. These populations are older than the intended neurodevelopment setting, but the repeated signal across memory-oriented outcomes supports the biological activity of PS-centered interventions.

4. Discussion

We interpret the evidence base as supportive but distributed. The finished Acer truncatum seed oil–PS composition has a coherent mechanistic rationale, yet the human clinical evidence currently concentrates in phosphatidylserine and related phospholipid interventions rather than in the exact finished product. For publication purposes, that distinction should be preserved rather than blurred.

The formulation's likely differentiating hypothesis is not simply that it contains phosphatidylserine, but that it pairs PS with a nervonic-acid–rich lipid matrix and complementary phospholipid cofactors that may better support myelination-oriented development and membrane composition. However, because direct human Acer truncatum seed oil trials were not identified in the literature we reviewed, the strongest evidence statement we can defend is that the formulation is clinically plausible and trial-ready-not already clinically proven.

The pediatric literature also suggests an important design lesson for future trials. The most persuasive benefits were seen in children with ADHD or in lower-performing subgroups, not

ISSN: 2710-0723

across every healthy-child cohort. We therefore believe that a prospective trial of the exact Acer truncatum seed oil–PS complex should preferentially target children with measurable attentional inefficiency, nutritional vulnerability, or developmental risk markers rather than a completely unselected healthy sample.

Our principal limitation is that the human evidence is component-based rather than product-specific. That limitation does not negate the formulation rationale, but it does shape the appropriate academic conclusion. We should present the composition as a differentiated translational candidate supported by phosphatidylserine-focused human trials and by myelination-related phospholipid evidence, while acknowledging the need for a direct randomized study of the exact formulation.

5. Conclusion

We conclude that the Acer truncatum seed oil–phosphatidylserine complex occupies a scientifically credible position in neurodevelopment-support research. Its strongest clinical foundation comes from randomized human evidence on phosphatidylserine, PS-omega-3, and phospholipid-supported developmental outcomes, while its Acer truncatum/nervonic-acid dimension remains promising but insufficiently validated in humans.

A publishable interpretation of the current record is therefore straightforward: the formulation is supported by a coherent translational evidence chain, but the exact finished product still requires prospective randomized verification in an appropriately selected pediatric population.

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